Biochimica et Biophysica Acta 848 (1986) 239–246
Elsevier

BBA 41922

Analysis of chlorophyll fluorescence induction kinetics exhibited by DCMU-inhibited thylakoids and the origin of α and β centres

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(Received June 10th, 1985)
(Revised manuscript received September 11th, 1985)

Key words: Chlorophyll fluorescence; Photosystem II; Photosynthesis; Fluorescence induction; (Pea thylakoid)

Analyses of room-temperature chlorophyll fluorescence curves from DCMU-inhibited thylakoids were used to investigate the proposed PS II structural heterogeneity of α and β centres. The kinetics of the area growth curves, representative of Q_A photoreduction, could be modified in the presence of DCMU by exogenous electron acceptors and by added reductants of the PQ pool. The effect of altered DCMU levels (range 0.2–100 μM) on the induction curve kinetics was to modify preferentially the slow-β component, while having only a very small effect on the total variable fluorescence yield. Over the DCMU concentration range used, the unnormalized area of the induction curve (A_{max}) decreased with increasing herbicide concentration by approx. 45%, indicating that less quanta were required to reduce Q_A. It was found that the dark reoxidation of Q_A in the presence of DCMU and Ant 2p after a light pretreatment regenerated the slow kinetic component. When chlorophyll fluorescence emission at 685 and 731 nm was measured, no difference was observed in the kinetics of the induction curve. The analysis of PS II-enriched, oxygen-evolving membranes indicated the presence of both the fast and slow kinetic components, although this type of preparation showed a modified fast phase. The above observations led to the conclusion that several of the previously proposed characteristics of PS II_α and PS II_β centres do not hold and that a type of PS II heterogeneity involving different degrees of DCMU inhibition is sufficient to explain many of the observations made.

Introduction

Room temperature chlorophyll fluorescence induction measured using thylakoids in which electron transfer from Q_A to Q_B has been inhibited by DCMU produces a curve which is not indicative of a single first-order photochemical event. The apparent biphasic nature can be readily detected, when thylakoids are suspended in 'high' salt to produce appressed and non-appressed membrane regions, by analysing the growth of the normalised area over the induction curve [1]. The semilogarithmic plot of such an analysis distinguishes two kinetically different phases, one being fast and nonexponential, while the other is slow and ex-

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Abbreviations: Chl, chlorophyll; PS II, Photosystem II; PS I, Photosystem I; LHC, light harvesting Chl *a*/*b*-protein complex; Q_A, primary stable electron acceptor of PS II; Q_B, secondary stable electron acceptor of PS II; PQ, plastoquinone; DCMU, 3-(3,4-dichlorophenyl)-1,1-dimethylurea; Ant 2p, 2-(3-chloro-4-trifluoromethyl)anilino-3,5-dinitrothiophene; P-680, primary electron donor to PS II; Tricine, *N*-[2-hydroxy-1,1-bis(hydroxymethyl)ethyl]glycine; F_m, maximum chlorophyll fluorescence; F_0, minimum chlorophyll fluorescence; $F_v = F_m - F_0$; A_{max}, unnormalised area over fluorescence induction curve; β_{max}, intercept at time zero of linearly extrapolated exponential phase of the semilog plot of area-growth curve; k_α, initial first-order rate constant for PS II_α area-growth curve; k_β, first-order rate constant for PS II_β area-growth curve; BBY, PS II-enriched preparation made according to Ref. 4.

ponential. These two phases have been attributed to two distinct forms of Photosystem II (PS II), termed α and β centres, respectively [2]. It has been proposed that PS II_α is located in a statistical pigment bed with excitation-energy transfer between PS II reaction centres, while PS II_β units are separate entities unable to transfer energy between each other. Over the past several years other differences between the two types of PS II centre have been investigated and (a)–(g) has been claimed.

(a) PS II_α is only found in granal membrane fragments [3] and 'BBY'-type [4] preparations [5], while PS II_β is detected in stromal membrane fragments [3].

(b) PS II_β has associated with it less LHC than PS II_α [6] and contains antenna chlorophylls having an absorption maximum which is far-red shifted compared with the chlorophylls of PS II_α [7].

(c) Unlike PS II_α, PS II_β is not associated with the two-electron gate Q_B [8].

(d) PS II_α and PS II_β have different primary stable electron acceptors, termed Q_α and Q_β, respectively, having different midpoint potentials [9].

(e) PS II_β can be preferentially altered by high herbicide concentrations [10].

(f) Only PS II_α fluorescence emission is affected by Mg^{2+} levels [11].

(g) The two types of centre have different fluorescence emission characteristics [12].

In contrast to the above interpretation it has been suggested that the biphasic nature of the fluorescence rise simply reflects different degrees of PS II 'connectivity' and does not require either membrane differentiation or differences in antenna size [13]. Other explanations are that the biphasicity could be a result of the degree of chloroplast integrity linked to alterations in DCMU affinity [14,15] or that it is a consequence of different degrees of PS II/LHC interaction [16].

The work presented in this paper investigates some of the above-mentioned explanations for the origin of PS II_α and PS II_β by measuring and analysing room-temperature chlorophyll fluorescence induction curves, using isolated pea thylakoids subjected to various modifying pretreatments. This approach was complemented by studies of fluorescence inductions from PS II-enriched membranes obtained by subfractionation of isolated thylakoids.

Materials and Methods

Chloroplasts were isolated as in Ref. 17 from *Pisum sativum* and oxygen-evolving PS II-enriched membranes ('BBY') were prepared from the isolated chloroplasts as in Ref. 4.

All fluorescence induction curves were measured at room temperature using the apparatus and procedures previously described in Ref. 18 and analysed using the methods of Melis and Homann [1,2].

Intact chloroplasts were shocked, before use, at twice the final cation concentration and finally resuspended to give 5 μg Chl per ml, 10 mM Tricine (pH 8.2, KOH), 0.33 M sorbitol, 10 mM KCl and 5 mM $MgCl_2$, for subsequent investigation.

To investigate the effect of exogenous electron acceptors on the fluorescence curves, the thylakoids were incubated in the dark for 4 min before the introduction of 20 μM DCMU followed by either 100 μM methyl viologen or 0.5 mM potassium ferricyanide before or after DCMU addition. Fluorescence was measured after a further 2 min dark incubation. The effect of 4.2 μM *Spirulina maxima* ferredoxin and 0.5 mM NADPH on the fluorescence rise kinetics were investigated using the same procedures.

In certain experiments various concentrations of DCMU (0.2–100 μM) were added to the thylakoids after a 4.5 min dark-time with fluorescence being measured at 685 nm after a total dark adaptation of 5 min (in some cases fluorescence was measured at 731 nm). To monitor the dark reoxidation of Q_A^-, thylakoids were suspended as above but in the presence of 4.5 μM Ant 2p and 20 μM DCMU. Control membranes were dark incubated for 5 min before measurement, while other thylakoid samples were illuminated for 5 s with white light (intensity, 50 $W \cdot m^{-2}$) and allowed to dark-adapt for 15 s before measurement.

Fluorescence measurements on 'BBY'-type membranes were carried out at either pH 6.0 or 8.0 in the presence of 20 μM DCMU after a 5 min dark preincubation using the same basic medium described above for intact membranes. The enrich-

ment of PS II in these membrane fragments was established by several measurements based on knowledge presented in the literature including Chl *a*/*b* ratio, low-temperature fluorescence emission properties and SDS-polyacrylamide gel electrophoresis polypeptide patterns.

Results

It has previously been shown that altering the redox conditions under which thylakoids are suspended leads to changes in the kinetics of the fluorescence curves so that at lower redox potentials the slow, β-phase is selectively removed [15,19]. To examine if the slow phase could result from an inefficient blockage of electron flow by DCMU the effect of various electron acceptors and of *Spirulina maxima* ferredoxin/NADPH on the fluorescence kinetics was investigated. Fig. 1 compares the semilog plots and area growth curves of thylakoids suspended in the presence of potassium ferricyanide (Fig. 1a), *Spirulina maxima* ferredoxin/NADPH (Fig. 1b) or methyl viologen (Fig. 1c). It can be seen that the addition of potassium ferricyanide after DCMU addition led to a faster rate of Q_A photoreduction, due to an increase in the first-order rate constant k_β for PS II_β area growth, while the addition of potassium ferricyanide before DCMU addition produced a slower rate of Q_A accumulation by decreasing the first-order rate constant k_α for PS II_α area growth. The addition of *Spirulina maxima* ferredoxin/NADPH (like dithionite) and, somewhat surprisingly, the addition of methyl viologen both led to a faster rate of Q_A reduction by suppressing the slow, kinetic phase. Similar trends to those shown in Fig. 1 have been observed by Joliot and Joliot [20].

Schreiber and Pfister [14] first reported that by increasing the level of DCMU the PS II_β contribution to the fluorescence rise could be reduced. However, Horvath et al. [10] have since suggested that high concentrations of herbicides which act on PS II, preferentially damage PS II_β centres so that they become unavailable for photoreduction. This effect was detected as a loss of variable chlorophyll fluorescence. Fig. 2 shows DCMU titration curves of various fluorescence parameters. It can be seen from Fig. 2a that increasing the DCMU concentration from 0.2–100 μM brought about very little change in the level of maximum chlorophyll fluorescence (F_m), minimum chloro-

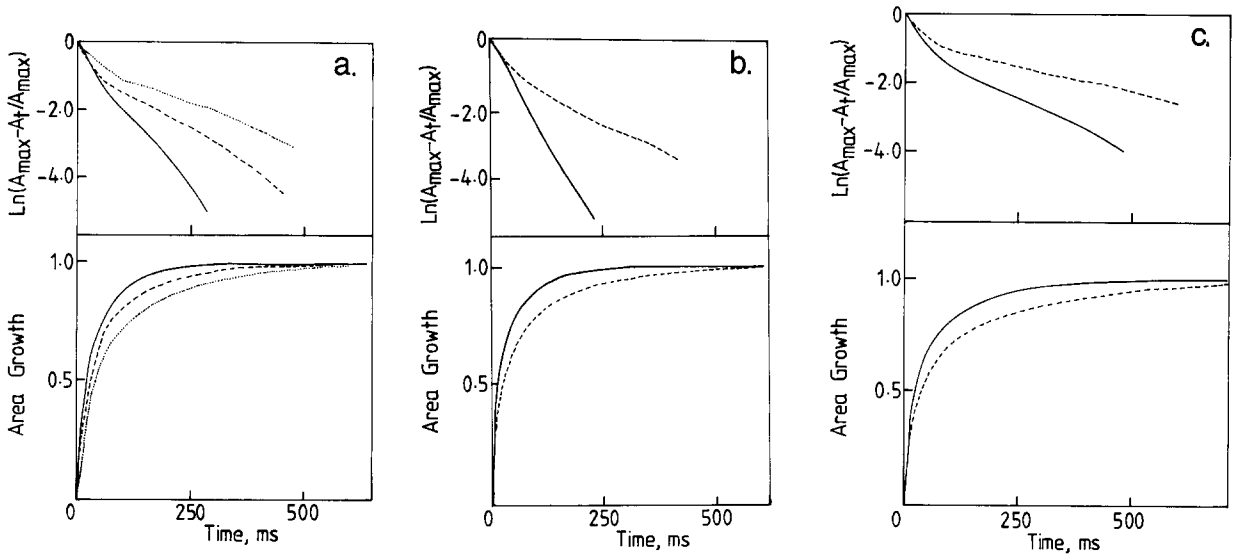

Fig. 1. The semilog plots, $\ln(A_{max} - A_{(t)}/A_{max})$, and the growth of the normalised area over room-temperature fluorescence induction curves (area growth) for thylakoids suspended in the presence of 5 mM Mg^{2+} in: (a) the presence of 0.5 mM potassium ferricyanide before (·····) or after (————) DCMU addition and in the absence of potassium ferricyanide (- - - - -); (b) the presence (————) and absence (— — —) of 4.2 μM *Spirulina maxima* ferredoxin and 0.5 mM NADPH; (c) the presence (————) and absence (- - - - -) of 100 μM methyl viologen. All samples contained 20 μM DCMU.

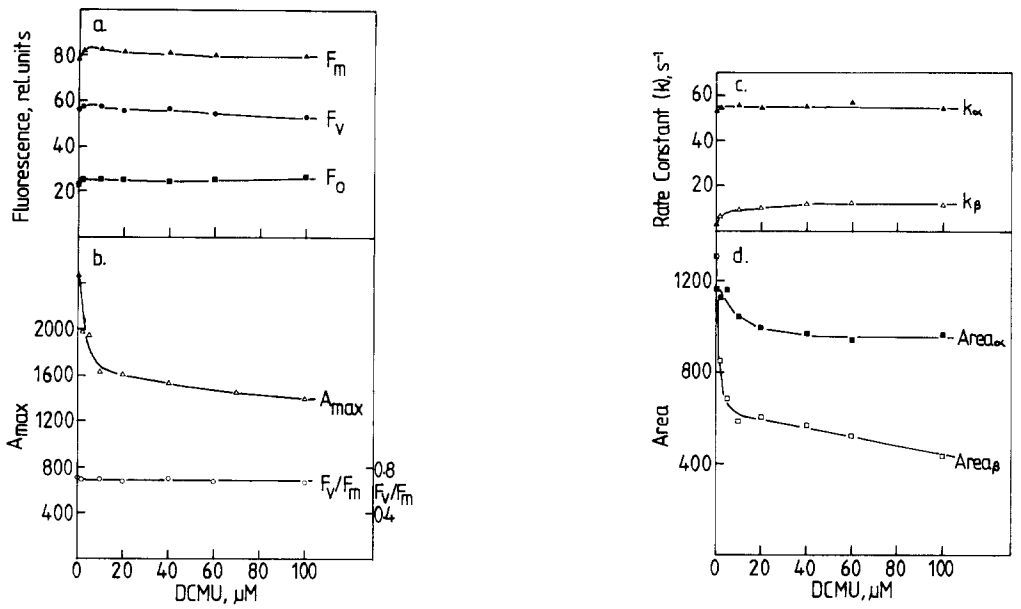

Fig. 2. The effect of DCMU concentration on various room-temperature fluorescence induction curve parameters; (a) F_m (▲), F_v (●) and F_0 (■); (b) A_{max} (■) and F_v/F_m (○); (c) k_α (▲) and k_β (△); (d) area$_\alpha$ (■) and area$_\beta$ (□). All samples were suspended in the presence of 5 mM Mg^{2+}.

phyll fluorescence (F_0) and variable fluorescence ($F_v = F_m - F_0$), while A_{max} decreased by 43% with no significant alteration in the F_v/F_m ratio (Fig. 2b). It can be seen, however, that increasing the DCMU level preferentially affected the slow β-associated parameters so that k_β increased by 58% (with only a 7% change in k_α) (Fig. 2c). From the various β_{max} values generated, the area due to PS II_α and PS II_β was calculated as a function of DCMU concentration (Fig. 2d). This area is representative of the number of quanta required to reduce Q_A, and hence is related to the number of PS II centres. This again indicates a preferential effect of increasing DCMU concentration on PS II_β centres, leading to a 68% decrease in Area$_\beta$ over the concentration range investigated (while Area$_\alpha$ decreased only by 17%).

After a saturating light treatment to reduce Q_A, even in the presence of DCMU and an inhibitor of PS II back reactions, Q_A still becomes slowly and partially reoxidized in the dark. Fig. 3 shows semilog plots and area-growth curves for control thylakoids (no light pretreatment) and thylakoids given 15 s dark reoxidation after a light pretreatment. It can be seen from Fig. 3 and Table I that the reoxidised Q_A was subsequently rereduced with mainly slow, exponential kinetics attributable to PS II_β (as seen in Table I by the similar k_β values).

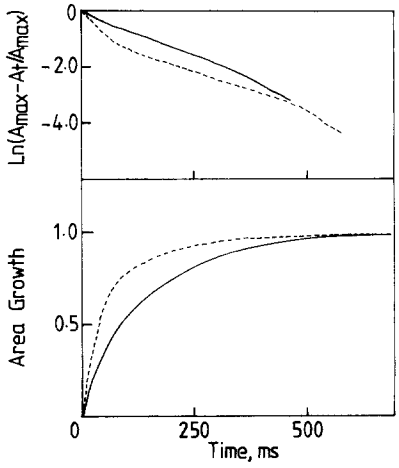

Fig. 3. The semilog plots $\ln(A_{max} - A_{(t)}/A_{max})$, and the area-growth curves exhibited by control thylakoids, given no preillumination (------) and light-pretreated membranes given a 15 s dark reoxidation time after preillumination (————). All samples were suspended in the presence of 20 μM DCMU, 4.5 μM Ant 2p and 5 mM Mg^{2+}.

TABLE I

THE EFFECT OF A PREILLUMINATION LIGHT TREATMENT ON VARIOUS FLUORESCENCE INDUCTION CURVE PARAMETERS

Condition	F_0	F_m	A_{max}	k_α (s^{-1})	k_β (s^{-1})	β_{max}	Area$_\alpha$	Area$_\beta$
Control (no light treatment)	25	75.4	1470	30	5.4	0.35	956	515
15-s reoxidation (after light treatment)	61	72.7	398	64	5.9	0.81	77	322

TABLE II

VARIOUS FLUORESCENCE INDUCTION CURVE PARAMETERS MEASURED AT 685 nm AND 731 nm

Condition	F_0	F_m	A_{max}	k_α (s^{-1})	k_β (s^{-1})	β_{max}	F_v/F_m	Area$_\alpha$	Area$_\beta$
F_{685}	25	90.5	1340	41.9	4.6	0.37	0.72	840	500
F_{731}	20.2	58.5	777	44.5	4.9	0.40	0.65	467	310

It has recently been suggested that PS II$_\alpha$ fluorescence has a maximum at 685 nm while PS II$_\beta$ has its fluorescence maximum at 715–730 nm [12]. Table II gives various fluorescence parameters from fluorescence induction curves measured at either 685 nm or 731 nm. Although the F_0, F_m and A_{max} values cannot be compared due to different filter transmissions and altered fluorescence yields, the normalised kinetic data at the two wavelengths are, however, comparable. Table II shows that there are no observable differences between k_α, k_β and β_{max} when fluorescence is measured at 685 nm or 731 nm.

The original proposal of Melis and Homann [1,2] that PS II$_\alpha$ is located in the grana while PS II$_\beta$ is situated in the stromal lamellae has since been apparently substantiated by various kinetic analyses of thylakoid membrane fragments [3,5]. Table III compares fluorescence parameters obtained from induction curves generated by oxygen evolving PS II-enriched preparations ('BBY') at both pH 6.0 and pH 8.0. A slow component, perhaps corresponding to PS II$_\beta$, can be seen in this type of preparation. A comparable slow kinetic

TABLE III

VARIOUS FLUORESCENCE INDUCTION CURVE PARAMETERS MEASURED FROM INTACT PEA CHLOROPLASTS AND A PS II PREPARATION ('BBY')

Condition	F_0	F_m	A_{max}	k_α (s^{-1})	k_β (s^{-1})	β_{max}	F_v/F_m
pH 8.0							
BBY	40	68.5	1170	27.0	4.0	0.57	0.42
Intact	34	104.5	1600	38.4	4.1	0.34	0.68
pH 6.0							
BBY	40	88.5	3800	20.0	2.1	0.70	0.55
Intact	30	93.0	2400	31.0	2.7	0.52	0.68

phase was also detected from PS II-enriched membranes isolated using a non-detergent method (see Ref. 27) (data not shown).

Discussion

The data presented above suggest that the observed biphasic nature of room-temperature chlorophyll fluorescence of induction curves from DCMU-inhibited thylakoids may not simply be described as two distinct forms of PS II located in different membrane regions as previously proposed by Melis and co-workers [1–3]. In a previous report [15], which showed the removal of the slow phase by the addition of dithionite, it was suggested that the β-component could represent certain PS II centres not easily blocked by DCMU [14]. Brearley and Horton [12] have also suggested, from DCMU titrations of F_m, that α and β centres show different DCMU affinities (I_{50} of 0.05 and 365 μM, respectively). Moreover, Joliot and Joliot [20] have also noted that high DCMU levels preferentially affect PS II_β so that the reduction of Q_A associated with the slow phase was inversely proportional to DCMU concentration (see also Fig. 2c). Such reports are all consistent with the idea that an altered DCMU affinity may partly explain the slow rise in fluorescence. However, Horvath et al. [10] have suggested that high herbicide levels preferentially damage PS II_β via an undefined conformational change, so as to inhibit their photoreduction leading to a decrease in F_v with little change in F_0. This trend is not seen in Fig. 2a, however the β phase was still drastically altered over the DCMU concentration range investigated so that k_β increased and Area_β diminished. This latter finding does infer a conversion of PS II_β into PS II_α (Fig. 2c and d), although even at 100 μM DCMU a slow component still persisted perhaps reflecting those centres exhibiting an I_{50} value of 365 μM.

Other published work also appears to agree with the notion that a slow fluorescence rise could be manifested by a leak of electrons through the DCMU block. Diner and Delsome [21] have reported, contrary to Melis and Schreiber [22], that Q_β reduction does not give rise to a C550 absorption change nor to a transmembrane potential (this latter point being confirmed by Meiburg and Van Gorkom in Ref. 23). It was suggested that if Q_β had an E_m value of +120 mV [9], then it was not related to a PS II primary-electron acceptor, but could reflect a secondary acceptor or be related to PS II with modified herbicide binding [21]. This latter conclusion is consistent with the observation that β centres are not associated with the two-electron gate mechanism [8]. This work also showed that Tris-washed thylakoids exhibited no slow rise. Tris treatment inhibits electron donation to P-680$^+$ and might imply the requirement for more than one PS II turnover to generate PS II_β.

The ability of exogenous electron acceptors to modify the slow rise kinetics in the presence of DCMU is also consistent with a leakage mechanism (see Fig. 1a and c). Similar observations using methyl viologen and potassium ferricyanide have been reported by Joliot and Joliot in Ref. 20 who explained their results in terms of the removal of the $Q_AQ_B^-$ state which is present in the dark, and is known to reduce the affinity of DCMU by 20-fold [24]. They concluded that the slow rise was due to both the $Q_AQ_B^-$ state and PS II_β centres. However, another explanation could be that k_β increases and β_{max} decreases because of a poor evaluation of A_{max}. Bell and Hipkins [25] have shown that only a 1% underestimation of F_m can lead to a 17% decrease in A_{max} which produces an apparent faster reduction of Q_A. If a population of PS II centres is not inhibited by DCMU then methyl viologen and potassium ferricyanide might accelerate the reoxidation of PS II_β via PS I electron flow leading to an extended, very slow rise which could lead to an underestimation of A_{max}. The effect of *Spirulina maxima* ferredoxin/NADPH (Fig. 1b) is similar to that previously observed for dithionite [15] and again suggests the requirement for oxidised PQ in the manifestation of the slow phase.

The data of Fig. 3 and Table I can also be explained by a leak of electrons through the DCMU block. The major component which becomes reoxidised in the dark in the presence of DCMU and Ant 2p (which inhibits PS II reoxidation via back reactions, unpublished observation) can be attributed to PS II_β centres. This cannot be explained by the $Q_AQ_B^-$ state as the 5 s preillumination would have generated several turnovers and subsequently removed this redox state. Such

observations are in contrast to those of Thielen and Van Gorkom [8] who apparently found that PS II_α was the major component reoxidised.

It has already been mentioned that PS II_β centres have been reported to contain a smaller antenna consisting of red-shifted chlorophyll *a* molecules ($\lambda_{max} \approx 683$ nm) compared with PS II_α [7]. This observation led Brearley and Horton [12] to investigate the fluorescence emission characteristics of the two types of centre. In the presence of methyl viologen and at 80 μg Chl/ml they showed that PS II_β had a maximum at 725–735 nm. However, Table II shows that in the absence of methyl viologen and using a more dilute suspension of thylakoids no such difference in the β_{max} level was found when fluorescence was measured at 685 nm and 731 nm. Such an observation seems to indicate that the chlorophylls associated with the two types of centre are also similar.

It has also been reported, using membrane fragments, that only PS II_α centres can be detected in the appressed, granal lamellae [3,5]. Table III, which gives various fluorescence parameters for 'BBY', does not appear to confirm these previous observations, as the 'BBY' preparations exhibited similar slow kinetics when compared with the unfractionated thylakoids. In fact such PS II-enriched membranes exhibited a higher β_{max} level due to a lower k_α and perhaps a lower F_v/F_m ratio as did PS II-enriched membranes prepared by non-detergent methods (not shown). This trend was seen at both pH 6.0 and pH 8.0. Furthermore, if PS II_β centres were located in the stromal lamellae then they would be expected to spillover excitation energy to PS I (see Ref. 26), thereby lowering their fluorescence emission. Such an effect would lead to an apparent underestimation of the number of PS II_β centres from the subsequent fluorescence analysis. However, this type of kinetic analysis produces similar α/β ratios to those obtained from the analysis of ΔA_{320} absorption changes which monitor Q_A reduction and would not be diminished by spillover [11]. This would not be expected unless either PS II_β centres do not spillover excitation energy to PS I or they are not located in the stromal membrane regions.

It is interesting to note that much of the original work on α/β heterogeneity was carried out on tobacco mutants lacking LHC (see Refs. 6 and 7). Such photosynthetic material shows a correlation between the number of β centres (as determined by β_{max}) and the degree of membrane appression. This trait is similar to that observed when thylakoids are suspended in the presence of different cation levels [15,18] in which case the actual number of α and β centres does not change [11]. Therefore the mutants may not have contained more PS II_β centres, the apparent increase simply reflecting a decrease in PS II_α connectivity due to the absence of thylakoid stacking with preferentially modifies k_α leading to an increase in β_{max}.

The literature concerning this form of PS II heterogeneity is very confused. This is partly because of the utilisation of different types of photosynthetic material, while always determining the α/β ratio by the fluorescence analysis which is sensitive to membrane conformation [15] and to the accuracy of the A_{max} determination [25]. The fluorescence rise is probably far more complex than just the two phases it is usually reported to represent. The results presented above do not seem to be consistent with the concept of two spectrally and functionally distinct PS II complexes initially put forward by Melis and Homann [1,2], but suggests that a different from of PS II heterogeneity could be present which is related to the ability of DCMU to inhibit linear electron flow away from PS II.

Acknowledgements

We wish to thank the SERC and AFRC for financial support. M.H. held a SERC-CASE studentship with GCRI, Littlehampton. We are also indebted to Drs. K.K. Rao and G. Renger for their generous gifts of *Spirulina maxima* ferredoxin and Ant 2p, respectively, and Dr. P.A. Millner for the preparation of the 'BBY' membranes.

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