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Minireview

State 1/State 2 changes in higher plants and algae*

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Abstract. Current ideas regarding the molecular basis of State 1/State 2 transitions in higher plants and green algae are mainly centered around the view that excitation energy distribution is controlled by phosphorylation of the light-harvesting complex of photosystem II (LHC-II). The evidence supporting this view is examined and the relationship of the transitions occurring in these systems to the corresponding transitions seen in red and blue-green algae is explored.

Abbreviations: CCCP – carbonylcyanide-m-chlorophenylhydrazone; Chl *a* – chlorophyll *a*; Chl *b* – chlorophyll *b*; DAD – diaminodurene; DBMIB – 2,5-dibromo-3-methyl-6-isopropyl-p-benzoquinone; DCCD – N,N′-dicyclohexyl carbodiimide; DCMU – 3-(3,4-dichlorophenyl)-1,1′-dimethylurea (also called diuron); FCCP – carbonylcyanide-p-trifluoromethoxyphenylhydrazone; FSBA – 5′-fluorosulphonylbenzoyl adenosine; kDa – kilodalton; LHC-II – light-harvesting Chl *a*/Chl *b* protein; PMS – phenazine methosulfate; PS I – photosystem I; PS II – photosystem II; SDS – sodium dodecyl sulfate; TPTC – triphenyl tin chloride.

Introduction

The ability of higher plants and algae to vary the distribution of excitation energy between their two photosystems so as to maximize the overall efficiency of photosynthesis under any given light conditions was first clearly recognized by Bonaventura and Myers (1969) and Murata (1969). They showed that exposure to light predominantly absorbed by PSII (Light 2) resulted in a redistribution of excess excitation energy from PS II to PS I while exposure to light preferentially absorbed by PS I (Light 1), prevented such transfer. The states arising from exposure to Light 1 and Light 2 are referred to as State 1 and State 2 respectively and the transitions between such states as State 1/State 2 transitions.

 The molecular basis of State 1/State 2 transitions has been widely reviewed over the years (Williams, 1977; Haworth et al., 1982a; Allen, 1983; Barber, 1983; Bennett, 1983; Horton, 1983; Staehelin and Arntzen, 1983; Barber, 1986; Fork and Satoh, 1986). This review will, therefore, concentrate on recent progress in this field. In particular, it will address the question as to whether State 1/State 2 transitions in higher plants and green algae can adequately be explained in

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* This paper follows our new instructions for citation of references — authors are requested to follow Photosynth Res 10: 519–526 (1986) — editors.

terms of the changes in the relative absorption cross-sections of PS I and PS II associated with the phosphorylation of chlorophyll a/b light-harvesting protein (LHC-II) and the possible alternative mechanisms that might be operating in photosynthetic systems lacking this protein.

LHC-II and LHC-II phosphorylation

Sodium dodecyl sulfate-polyacrylamide gel electrophoresis (SDS-PAGE) of chloroplast thylakoids under mildly denaturing conditions normally yields two or three different LHC-II bands. It is still not clear whether these represent different multimeric forms of a single LHC-II complex or differing forms of LHC-II. The different bands are, however, made up of the same two apo-polypeptides combined in approximately similar ratios (Bennett et al., 1981). The molecular weights of the apo-polypeptides vary somewhat with the source material but generally lie in the range 23–29 kDa. They are synthesized from different mRNAs (Schmidt et al., 1981) but have very similar amino acid compositions and immunological properties (Apel, 1977; Chua and Blomberg, 1979; Hoober et al., 1980).

Bennett (1977) showed that incubation of intact chloroplasts in light with [^{32}P]-orthophosphate results in the phosphorylation of a number of thylakoid proteins. The most conspicuous of these are the 24 kDa and 26 kDa polypeptides of LHC-II but 10 kDa, 33 kDa, 35 kDa and 45 kDa polypeptides, all associated with PS II, are also phosphorylated (Owens and Ohad, 1982; Steinback et al., 1982; Delepelaire, 1984). Bennett and his collaborators have shown that phosphorylation of LHC-II is brought about by a membrane-bound kinase that is normally inactive in the dark but that can be activated by light (Bennett, 1979; Allen et al., 1981). A light-independent phosphatase system dephosphorylates LHC-II under conditions in which the kinase is inactive (Bennett, 1980).

LHC-II phosphorylation and excitation energy distribution

The idea that phosphorylation of LHC-II leads to a change in the relative absorption cross-sections of PS I and PS II, and hence that it might be involved in the control of State 1/State 2 transitions, can be traced back to observations that phosphorylation of LHC-II leads to a decrease in the room temperature fluorescence emission of Chl a associated with PS II (Bennett et al., 1980; Horton and Black, 1980, 1981a; Allen et al., 1981). Measurements of low-temperature (77 K) emission suggested that this decrease in PS II emission was accompanied by a corresponding increase of fluorescence emission from PS I. This has since been confirmed by experiments in which fluorescent internal standards were included so that absolute rather than relative changes in PS I and PS II emission could be measured (Krause and Behrend, 1983; Saito et al., 1983). For a complete review on Chl a fluorescence emission see Govindjee et al (1986).
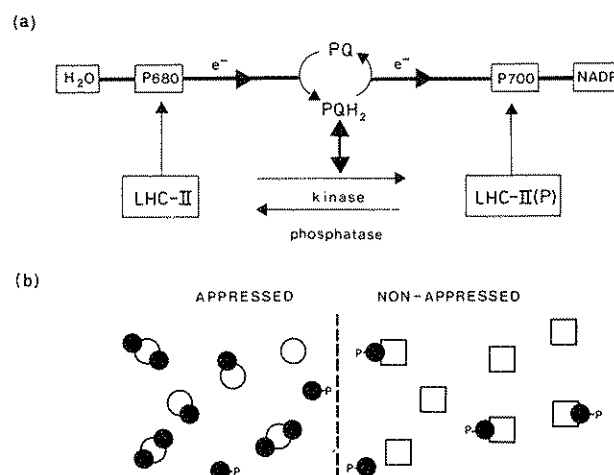
(a)

(b)

APPRESSED   NON-APPRESSED

Fig. 1. Diagrammatic representation of the LHC-II phosphorylation model. (a) shows the kinase control system and its relationship to the redox state of the PQ pool and (b) the effect of LHC-II phosphorylation on the distribution of LHC-II (solid circles) between the appressed and non-appressed regions of the thylakoid membrane and its interactions with the main light-harvesting units of PS I (open squares) and PS II (open circles).

The currently accepted model for control of excitation energy distribution by LHC-II phosphorylation is summarized in Fig. 1. The activity of the LHC-II kinase is believed to be determined by the redox state of the plastoquinone (PQ) pool located between the two photosystems. Any imbalance in the distribution of excitation energy between the two photosystems in favour of PS II would, it is argued, be expected to lead to a reduction of the PQ pool, the activation of the kinase system and hence to the phosphorylation of LHC-II. This, in turn, is thought to lead to a dissociation of LHC-II from the light-harvesting units of PS II that are mainly located in the appressed membranes of the grana stacks. The free phosphorylated LHC-II complexes can then migrate to the non-appressed stromal membranes where they are able to interact with the light-harvesting apparatus of PS I. Over-stimulation of PS I is thought to result in a net oxidation of the PQ pool and hence the deactivation of the kinase. Under these conditions, phosphorylated LHC-II would be expected to become dephosphorylated by the phosphatase system and to return to the appressed membrane regions and to reassociate with PS II.

LHC-II kinase system

A number of lines of evidence favour the idea that the activity of the kinase system is determined by the redox state of the PQ pool. The kinase is normally activated by illumination with red light (Light 2) and deactivated by illumination with far-red light (Light 1). This activation is abolished by DCMU, which inhibits the reduction of PQ by PS II, but not by DBMIB which inhibits PQ

oxidation by PS I (Allen et al., 1981; Horton and Black, 1981a). It is also activated in the dark by reducing agents such as reduced ferredoxin or dithionite that can reduce the PQ pool (Bennett, 1979; Allen and Horton, 1981; Allen et al., 1981). Redox titrations in the dark indicate that the activation of the kinase system involves a two-electron carrier with a redox mid-point of about $0\,mV$ (Horton et al., 1981) supporting the idea that PQ is involved in the control system. The relative insensitivity of the redox state of the PQ pool of green algae to Light 1 and Light 2 illumination has led Dominy and Williams (1985) to suggest that it might be the redox state of specialized quinone species associated with the reducing side of PS II, rather than that of the main PQ pool, that is of importance in the control of the kinase. However, whatever the precise point of redox control, it is clear from studies on isolated thylakoids that LHC-II phosphorylation occurs during the transition to State 2 and dephosphorylation during the transition to State 1 (Telfer et al., 1983).

Evidence is emerging for the existence of a number of different protein kinases in chloroplasts. Lin et al. (1982) isolated two chloroplast protein kinases, which they refer to as $ChlPK_1$ and $ChlPK_2$, but neither were capable of phosphorylating chlorophyll-protein complexes (Lucero et al. 1982). Lucero et al. (1982) were able to demonstrate ^{32}P incorporation into purified LHC-II by an ammonium sulfate precipitate prepared from thylakoids but this activity was lost during subsequent steps in their purification procedure. Farchaus et al. (1985) identified a 50 kDa polypeptide, by selective labeling with the adenosine affinity inhibitor 5'-flurosulphonylbenzoyl adenosine (FSBA), that appears to phosphorylate LHC-II. Addition of FSBA resulted in a loss of 95% of LHC-II phosphorylation and a 35% decrease in phosphorylation of the 10 kDa polypeptide of PS II. Treatment with $[^{14}C]$-FSBA labeled several polypeptides but only the 50 kDa species was protected by the addition of ADP or adenosine suggesting that it was a kinase. The differential inhibition of phosphorylation of the 10 kDa and LHC-II polypeptides by FSBA led Farchaus and his co-workers to suggest the possible existence of different kinases for the two species. Clark et al. (1985) reported the partial purification of a thylakoid protein kinase that can phosphorylate LHC II which, on further purification, has been shown to have a molecular weight of about 64 kDa (Coughlan and Hind 1986a, b). None of these species have been shown to be redox-sensitive and the basis of redox control of LHC-II kinase activity remains to be elucidated.

The existence of two, or more, light-dependent kinases capable of phosphorylating different PS II polypeptides has also been suggested by Millner et al. (1982) on the basis of differential effects of N-ethylmaleimide treatment on the activity of the kinase(s) responsible for the phosphorylation of LHC-II and the 10 kDa polypeptide. Further support for this idea has come from reports by Markwell et al. (1983) on the effects of Zn^{2+} on the phosphorylation of these two species. Canaani (1985) reported the existence of two maxima, in the regions pH 6.0–6.5 and pH 8.0–8.5, in the pH dependencies of the light-driven transition to State 2 in intact leaves and ATP-induced fluorescence quenching of isolated spinach chloroplasts which she also interpreted as evidence for two light-depen-

dent kinases. Black et al. (1984) observed differences in the phosphorylation patterns of the LHC-II and 10 kDa polypeptides in peas grown under intermittent light but they were of the opinion that such changes could be explained in terms of substrate competition for a single species of kinase.

The fact that the kinase(s) phosphorylate a number of PS II polypeptides raises the question as to which particular species are associated with the ATP-induced fluorescence changes believed to be linked with State 1/State 2 transitions. The two most obvious candidates are the LHC-II polypeptides and the 10 kDa polypeptide. Under most conditions, the extents of phosphorylation of these species vary in parallel. Steinback et al. (1982), however, reported appreciable differences in their dephosphorylation kinetics. They showed that the restoration of PS II fluorescence followed the dephosphorylation of LHC-II rather than that of the 10 kDa polypeptide. This link between LHC-II phosphorylation and excitation energy distribution has been confirmed by measurements comparing the degree of phosphorylation of the two species and extent of fluorescence quenching under ATP-limiting conditions (Black et al., 1984). Owens and Ohad (1982, 1983) have suggested that the phosphorylation of PS II polypeptides other than those of LHC-II might be associated with the maintenance of the overall stability of the PS II light-harvesting apparatus.

Allen and Findlay (1986) have recently shown that the amino acid composition of the 10 kDa polypeptide resembles that of LHC-II. This has led them to propose that it is a chlorophyll-binding protein that acts as a link in excitation energy transfer between LHC-II and P680, the reaction center chlorophyll of PS II. They further suggest that it is the mutual repulsion of these two species on phosphorylation that causes the migration of LHC-II to the non-appressed regions of the membrane (see Allen and Holmes, 1986). Farchaus and Dilley (1986) have reported that the N-terminus of the 10 kDa phosphoprotein of spinach PS II particles is characterized by an unusual nine amino acid sequence. This sequence shows homology with an open reading frame in tobacco chloroplast DNA which codes for a membrane protein of 73 amino acids (Shinozaki et al., 1986). However, this protein, unlike the 10 kDa polypeptide studied by Allen and Findlay, lacks histidine (M. Sugiura — personal communication) and as such does not appear to correspond to their proposed chlorophyll-binding protein. Clearly further work, including complete protein sequencing, is desirable if the question of the identity and function of these proteins is to be resolved.

Lateral migration of phosphorylated LHC-II

There are two potential mechanisms for the redistribution of excitation energy in photosynthesis. These are to change the extent of spillover of excess energy from PS II to PS I or to change the fraction of incident light initially absorbed by PS I (usually referred to as α change) by direct transfer of light-harvesting pigments from one photosystem to the other. It is now generally accepted that

the light-harvesting units of PS II are largely confined to the appressed membranes of the grana stacks while the corresponding units of PS I are located in the non-appressed granal end-membranes and the stoma lamellae along the lines proposed by Andersson and Anderson (1980). This lateral separation of the photosystems is believed to be determined by the surface charge characteristics of the different light-harvesting units (see Barber (1982, 1983)).

Phosphorylation is thought to result in the introduction of additional negative charge into LHC-II which results in a mutual repulsion of phosphorylated membrane surfaces across the partition gap that leads to a partial unstacking of the grana and the subsequent migration of LHC-II from the appressed to the non-appressed regions of the thylakoid membrane (Barber, 1982; Haworth et al., 1982c; Kyle et al., 1983). If this migration involved whole PS II light-harvesting units, any excitation energy distribution changes would be expected to be dominated by changes in the extent of spillover while if only the antennae complexes undergo migration, these changes should primarily lead to changes in the value of α. Analyses of the low temperature fluorescence induction curves of phosphorylated and non-phosphorylated thylakoid membranes performed by Haworth et al. (1982b, c) indicated that the changes in both spillover and the value of α were involved but that there were distinct differences in the changes induced by phosphorylation and those induced by changes in Mg^{2+} levels where spillover changes predominated suggesting that only the antennae particles migrate on LHC-II phosphorylation (see also analyses of phosphorylation and cation-induced fluorescence changes of Wong et al. (1981), Argyroudi-Akoyunoglou (1984), Lombard and Strasser (1984) and Tsala and Strasser (1984); see also Strasser (1986)).

Berens et al. (1985) have obtained evidence from fluorescence lifetime studies for changes in the connectivity of PS II light-harvesting units on LHC-II phosphorylation. Their model is based on the idea introduced by Melis and Homann (1975) that PS II light-harvesting units can exist in two forms, referred to as α- and β-centers. Berens and her coworkers suggest that phosphorylation of LHC-II converts α-centers to β-centers. Holzwarth (1986) has proposed that phosphorylated LHC-II migrates from α-centres, located in the appressed granal membranes, to β-centers, located in the non-appressed stromal membranes, and that the resulting increase in the absorption cross-section of the β-centers may then facilitate increased spillover from PS II β-centers to PS I. For a more detailed discussion, see the review of Holzwarth (1986).

The detailed analysis of the fluorescence changes accompanying LHC-II phosphorylation has proved to be a very difficult task. The problem is exacerbated by the fact that the effects of LHC-II phosphorylation on the fluorescence emission of thylakoids appears to vary quite markedly with the level of Mg^{2+} present in the preparations and the developmental state of the chloroplasts (Horton and Black, 1983; Black and Horton, 1984). Horton and Black attribute these variations to differences in the relative effects of LHC-II phosphorylation on the surface charge of the thylakoids. At low Mg^{2+} concentrations, or early stages of development, the charge-screening effects of the divalent cations are
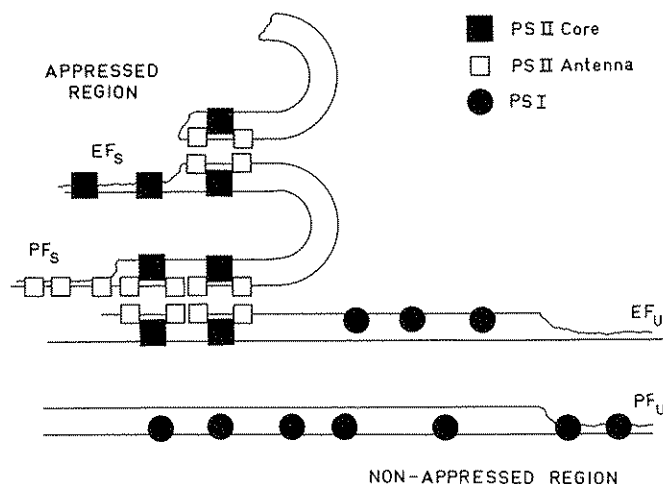
Fig. 2. Diagrammatic representation illustrating the different fracture-faces of higher plant thylakoid membranes revealed by freeze-fracture.

relatively low and protein phosphorylation tends to lead to relatively greater changes in membrane surface charge. This, in turn leads to a greater tendency for membrane destacking and an increase in the contribution of spillover to the excitation energy changes triggered by phosphorylation (see also Telfer et al., 1984a and Hodges and Barber (1984)).

Freeze-fracture studies indicate that LHC-II phosphorylation normally leads to relatively small changes in grana stacking. Kyle et al. (1983) found a decrease of 23%, and Simpson (1983) a decrease of 5%, in the stacking of phosphory-lated as opposed to non-phosphorylated thylakoids. Measurements of linear dichroism (Biggins 1982), light scattering changes (Haworth et al., 1982a, c), thin-section electron microscopy (Telfer et al., 1984b) and membrane fractiona-tion studies (Chow et al., 1981; Larsson et al., 1983) all indicate limited destack-ing on membrane phosphorylation.

Analyses of changes in freeze-fracture particle distributions performed by Kyle et al. (1983) suggest that phosphorylation leads to a migration of some of the 8 nm diameter particles fracturing with the protoplasmic fracture face of the stacked membranes (PF_s), believed to correspond to the light-harvesting atten-nae of PS II, to the protoplasmic faces of the unstacked membranes (PF_u) — see Fig. 2 for summary of fracture-face nomenclature. They found no significant changes in the distribution of the larger particles in the corresponding exoplas-mic faces (EF_s and EF_u) that are believed to correspond to the core complexes of PS II. Simpson (1983), in an independent study, came to rather different conclusions. In his study, there appeared to be a migration of both types of particles from the grana to the stroma lamellae. At the same time, a new population of larger particles appeared in the PF_s face. Simpson was uncertain of the origin of these latter particles but suggested that they might reflect either

conformational changes in the light-harvesting complexes of PS II or movement of PS I complexes from the stromal lamellae into the granal membranes.

The view that phosphorylation leads to a preferential migration of LHC-II from the granal to the stromal membranes is strongly supported by membrane fractionation studies (Kyle et al., 1983, 1984; Larsson et al., 1983; Larsson and Andersson, 1985). SDS-polyacrylamide gel electrophoresis of granal and stromal fractions indicates that phosphorylation results in increases in LHC-II, but not of the PS II core polypeptides, in the non-appressed membranes. Larsson and Andersson (1985) have compared the phosphorylation patterns of the 25 and 27 kDa polypeptides of LHC-II in the two regions. Their results indicate the existence of two pools of LHC-II one of which is strongly bound to PS II and contains the 27 kDa and 25 kDa polypeptides combined in the ratio 4:1 and a more mobile pool, which is more easily phosphorylated, in which they are combined in the ratio 2:1. The existence of such pools suggest the possibility that LHC-II might migrate as relatively small oligomeric light-harvesting complexes rather than as the relatively larger sub-units visualised by freeze-fracture electron microscopy. If so, it may well be that small changes in the size of the freeze fracture particles could be as significant as changes in their absolute numbers.

Is there any evidence against the LHC-II phosphorylation model?

The role of LHC-II phosphorylation in the control of excitation energy distribution has been questioned at various times by different research groups on the basis of difficulties encountered in demonstrating that LHC-II phosphorylation results in a transfer of excitation energy from PS II to PS I as opposed to a simple inhibition of PS II activity. Haworth and Melis (1983), for example, observed a 20–30% quenching in the variable component of PS II fluorescence on phosphorylation of LHC-II but found little or no change in the rates of photo-oxidation of P700, the reaction center chlorophyll of PS I. Horton and Black (1981b) saw no changes in cytochrome f oxidation on phosphorylation of LHC-II. Telfer et al. (1984c), in contrast, reported an increase in the extent of P700 oxidation following LHC-II phosphorylation. Measurements of the stimulatory effect of LHC-II phosphorylation on PS I-mediated electron transport have also proved difficult. Horton and Black (1982) observed such stimulation only at relatively low light intensities while Farchaus et al. (1982) were forced to take very stringent precautions to eliminate possible artifacts associated with photoinhibitory effects on both PS I and PS II electron transport before they could demonstrate PS I stimulation.

Markwell et al. (1983, 1984) have criticized the LHC-II phosphorylation model on rather different grounds. They found that addition of 1–2 mM Zn^{2+} stimulated the phosphorylation of LHC-II at low ATP concentrations but inhibited ATP-induced fluorescence quenching and the transfer of excitation energy from PS II to PS I. However, as pointed out by Farchaus et al. (1985),

Zn^{2+} ions bind tightly to ionizable groups on thylakoid membranes (Dilley and Rothstein 1967) and as such might block the phosphorylation-induced migration of LHC-II from appressed to non-appressed membrane regions. Under these conditions, little or no ATP-induced quenching or changes in excitation energy transfer between the photosystems would be anticipated.

Measurements of the intensity dependence of electron transport in chloroplasts showing ATP-induced fluorescence quenching indicate that phosphorylation of LHC-II leads to the stimulation of a dissipative cyclic electron transport pathway around PS II (Horton and Lee, 1983, 1984) and there is increasing evidence to suggest that LHC-II phosphorylation has a range of effects on the organization of PS II other than control of excitation energy transfer (Shocat et al., 1982; Jursinic and Kyle, 1983). It is possible that some of these other effects interfere with measurements of the rate of turnover of PS I.

The demonstration of the re-direction of excess excitation energy from PS II to PS I, as opposed to its dissipation by PS II, continues to be a matter of controversy. Nevertheless, given the success of the LHC-II phosphorylation model in accounting for the changes seen in low temperature fluorescence emission and the prediction of LHC-II migration from the appressed to the non-appressed region of the thylakoid membrane there seems little reason for doubting its validity under in vitro conditions at least.

LHC-II phosphorylation in leaf tissue

It has proved much more difficult to establish the role of LHC-II phosphorylation in State 1/State 2 transitions in leaf tissue and algae than in isolated chloroplasts. The problems associated with leaf and algal systems are rather different and it is thus convenient to discuss the systems separately.

The first evidence for State 1/State 2 transitions in leaf tissue was provided by Telfer and Barber (1981). They used modulated fluorescence techniques to show that exposure of detached pea and barley leaves to Light 2 resulted in a lowering, and exposure to Light 1 in a raising, of the fluorescence yield of PS II. In a related set of experiments, Chow et al. (1981) demonstrated that leaves of a Chl b-less mutant of barley that lacks LHC-II show no such changes (see also Markwell et al. (1985)).

The introduction of photoacoustic techniques that are able to monitor modulated O_2 evolution has opened up the possibility of measuring Emerson enhancement ratios in leaf tissue (Canaani et al., 1982). Using this technique, Canaani and Malkin (1984) have demonstrated that State 2 adaptation leads to a lowering, and State 1 adaptation to a raising, of the enhancement ratio of tobacco leaves in a manner exactly analogous to that reported by Bonaventura and Myers (1969) in their original demonstration of State 1/State 2 transitions in the green alga Chlorella. Analysis of these changes indicate that the excitation energy distribution changes associated with State 1/State 2 transitions reflect changes in the relative absorption cross-sections of PS I and PS II of the type

associated with the α-change mechanism rather than spillover changes. This conclusion has recently been confirmed by Malkin et al. (1986) on the basis of a detailed analysis of the modulated fluorescence changes seen in pea leaves.

Another useful technique for the study of excitation energy distribution in leaf tissue is the "leaf-powder" method for avoiding problems associated with fluorescence reabsorption in the measurement of low temperature emission spectra introduced by Weis (1984). Using this technique Weis (1985a, b) has been able to show that State 2 adaptation leads to an increase in the fraction of total excitation energy reaching PS I. Analysis of the induction curves of low temperature fluorescence again confirm that this increased transfer is predominantly due to a change in the relative absorption cross-sections of the two photosystems.

Bennett (1983) has provided convincing evidence that phosphorylation of LHC-II in leaf tissue under in vivo conditions shows the same sensitivity to the balance of excitation light between the two photosystems as seen under in vitro conditions i.e. Light 2 leads to the phosphorylation, and Light 1 to the dephosphorylation, of LHC-II (see also Bennett, 1984a, b). An important link between the LHC-II phosphorylation model and State 1/State 2 transitions in vivo appears to have been provided by Canaani et al. (1984). They showed that pre-treatment of leaf tissue with NaF in order to inhibit the LHC-II phosphatase system blocks State 1, but not State 2, adaptation. It should be noted that NaF is a general inhibitor of phosphatases and might be expected to inhibit the State 1 transition by virtue of its inhibition of the Calvin cycle. This result does not, therefore, provide unambiguous evidence of the involvement of LHC-II phosphorylation in State 1/State 2 transitions under in vivo conditions in leaf tissue. However, further work with more specific inhibitors might perhaps resolve this point.

LHC-II phosphorylation in green algae

The situation in the unicellular green algae is less clear. There is no reason to believe that the LHC-II complexes associated with green algae differ in any substantive way from those of higher plants. Measurements of $[\gamma\text{-}^{32}P]$ ATP-induced LHC-II phosphorylation (Owens and Ohad, 1982; Saito et al., 1983; Wollman and Delepelaire, 1984) and PS II fluorescence quenching (Saito et al., 1983) using thylakoids isolated from green algae all show similar changes to those seen for higher plant chloroplasts. The relationship between these changes and the light-induced State 1/State 2 changes of the type reported by Bonaventura and Myers (1969), however, is still an area of dispute.

The problems associated with this topic can be divided into two main areas. The first relates to a report by Owens and Ohad (1982) that indicates that similar amounts of $[^{32}P]$ orthophosphate are incorporated into the LHC-II of wild type and mutant strains of *Chlamydomonas reinhardtii* incubated in the dark, in the light in the presence, or absence, of DCMU, or in red or far-red light. Owens

and Ohad (1982, 1983) have suggested that the apparent insensitivity of LHC-II phosphorylation to the wavelength of illumination could be explained by the existence of two pools of phosphorylated LHC-II, one associated with the maintenance of PS II organization which is not influenced by the redox state of PQ and a second smaller pool that controls State 1/State 2 adaptation which is sensitive to the state of the PQ pool.

An alternative explanation, offered by Wollman and Delepelaire (1984), is that LHC-II as a whole is sensitive to the redox state of the PQ pool but that the fluctuations in the pool are, under normal conditions, quite small. They re-examined the levels of phosphorylation of LHC-II in wild-type *Chlamydomonas* and found an approximately 20% lower level of phosphorylation in State 1 as opposed to State 2 adapted cells. In order to test their hypothesis further, they examined the levels of phosphorylation in cells of the F34 mutant of *Chlamydomonas* that had either been exposed to light or dark-adapted under anaerobic conditions. The F34 mutant lacks PS II reaction centres and hence PQ becomes strongly oxidized in light. Dark-adaptation under anaerobic conditions, in contrast, is thought to lead to high levels of reduced PQ as a consequence of an interruption of the flow of electrons from NADPH to O_2 via the chlororespiration pathway identified by Bennoun (1982). The difference in levels of LHC-II labeling were, as anticipated, much greater than those seen in the wild-type cells. Direct measurements of the redox state of the PQ pool and of light-driven fluorescence increases associated with the dephosphorylation of LHC-II would appear to confirm this analysis (Wollman and Delepelaire, 1984; Delepaire and Wollman, 1985).

Godde and Trebst (1980) and Godde (1982) have reported the existence of membrane bound NADH-PQ-oxidoreductase activity in *Chlamydomonas* and such activity is unlikely to be unique to this alga. The relatively minor variations in the redox state of the PQ pool of *Chlorella* under different conditions reported by Dominy and Williams (1985) are probably reflections of such activity. Analyses of the fluorescence transients accompanying State 1/State 2 adaptation in *Chlorella* also tend to support the idea that State 1/State 2 changes involve changes in LHC-II phosphorylation (Hodges and Barber, 1983).

The second area of contention is associated with the interpretation of the mechanism underlying the slow light-induced fluorescence changes that take place in DCMU-poisoned algae. These changes, which were first studied by Papageorgiou and Govindjee and their co-workers in their early studies of excitation energy distribution (Govindjee and Papageorgiou, 1971; Papageorgiou, 1975), are widely used to study State 1/State 2 transitions in algae. Addition of DCMU to algal cells blocks electron flow from PS II, hence ensuring that any subsequent illumination preferentially stimulates PS I-mediated electron transport. It also closes all PS II traps ensuring that all fluorescence measurements are made at the maximal, F_M, level. Following the addition of DCMU, State 2 and dark-adapted algae usually show slow light-driven increases in fluorescence yield of 20–25% and 30–35%, respectively. The corresponding increase for State 1 adapted algae is normally < 5% (Williams
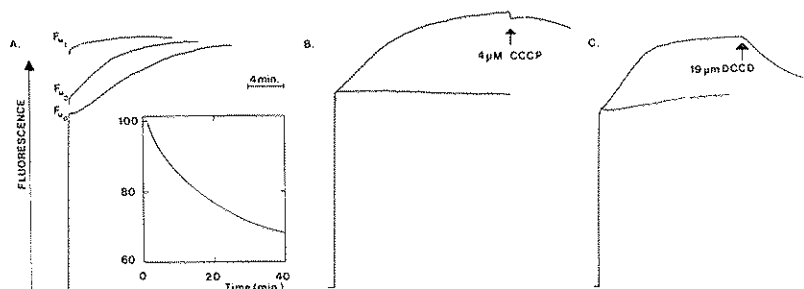
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Fig. 3. (A) Slow light-driven increases in the fluorescence yield (F$_M$) of *Chlorella* cells poisoned with DCMU (6 μM) after pre-adaptation to State 1, State 2 or the dark state. Initial fluorescence yields are those typical of the pre-adapted states. The dark reversal of these fluorescence changes, measured at very low light intensities in the presence of NH$_2$OH (250 μM), is shown in the inset. (B) and (C) show the effects of the uncoupler CCCP and ATPase inhibitor DCCD on these changes when added either with the DCMU or after the completion of the light-driven changes (after Williams (1984)).

et al., 1980). A typical set of such traces, measured using the alga *Chlorella vulgaris*, is shown in Fig. 3. Analogous changes are seen in blue-green and red algae but discussion of the significance of these changes will be deferred until the next section.

In terms of the LHC-II phosphorylation model, the fluorescence increases seen in DCMU-poisoned green algae are usually interpreted as reflecting the dephosphorylation of LHC-II under conditions in which the PQ pool is oxidized (Catt et al., 1984a; Williams, 1984). If the algae are left in the dark, in the presence or absence of DCMU, the value of F$_M$ falls back to a level close to that typical of State 2. There is abundant evidence suggesting that the distribution of excitation energy in dark-adapted green algae normally approximates closely to that of State 2 (Williams and Salamon 1976; Saito et al., 1983; Satoh and Fork 1983a; Catt et al., 1984a). If this is the case, the simplest explanation of these observations is that the reduction in PS II fluorescence in the dark reflects the reduction of PQ and the rephosphorylation of LHC-II.

Two sets of observations complicate this interpretation. The first relates to the finding that the slow light-driven fluorescence increases of the type illustrated in Fig. 2 are abolished by the addition of uncouplers and ATPase inhibitors (Papageorgiou and Govindjee, 1968a; Sane et al., 1982; Catt et al., 1984a) conditions which might be expected to prevent LHC-II phosphorylation. Following the addition of such agents, the value of F$_M$ normally falls back to that of the dark-adapted state. The second relates to a report by Satoh and Fork (1983a) that the dark-adapted cells of a *Scenedesmus* mutant lacking LHC-II show State 1/State 2 transitions similar to those seen in the wild-type algae.

Satoh and Fork (1983b) suggested that the fluorescence increases seen in DCMU-poisoned algae reflect changes in thylakoid organization brought about by proton fluxes associated with cyclic electron transport around PS I rather than changes in LHC-II phosphorylation levels. On this basis these increases
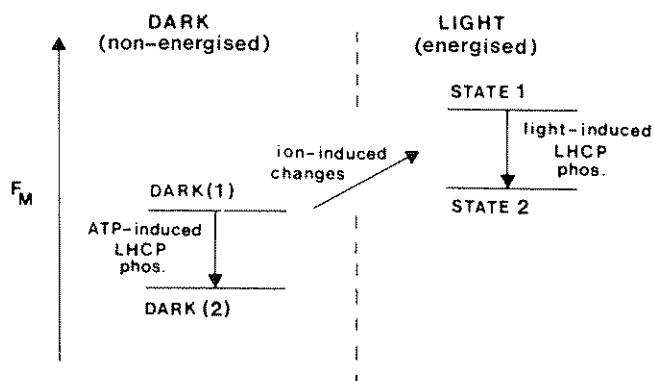
DARK
(non-energised)

LIGHT
(energised)

F_M

STATE 1

light-induced
LHCP
phos.

ion-induced
changes

STATE 2

DARK(1)

ATP-induced
LHCP
phos.

DARK (2)

Fig. 4. Model proposed by Catt et al. (1984) to account for the relationship between the dark-adapted state and State 1/State 2 changes. See text for details.

can be considered as reflecting an "energization" of the membrane and the fluorescence decrease seen in the dark, or in the presence of uncouplers, a relaxation of the membrane to a non-energized state. Williams and his co-workers attempted to combine the concepts of membrane energization and LHC-II phosphorylation in a single model (Catt et al., 1984a; Williams, 1984). They postulated the existence of two mechansims for altering excitation energy distribution, one corresponding to the wavelength-dependent State 1/State 2 transitions and the other to a wavelength-independent redistribution of ions within the chloroplast. These changes, it was suggested, act synergistically to give rise to two sets of State 1/State 2 changes characterized by different F_M levels as indicated in Fig. 4. The light-driven fluorescence increase seen in dark-adapted algae treated with DCMU would, on this basis, be interpreted as a reflection of a transition of the non-energized form of State 1 to its energized form rather than a transition from State 2 to State 1. It would be expected to be independent of the presence of LHC-II thus explaining its occurrence in the *Scenedesmus* mutant studied by Satoh and Fork (1983a).

The main problem with these types of model lies in explaining the nature and origin of the changes in excitation distribution associated with the energization process. Catt et al. (1984a) suggested that they might reflect stacking changes brought about by a redistribution of Mg^{2+} in response to light-driven proton-fluxes associated with cyclic electron transport. It is not clear, however, whether or not this explanation can be extended to cover the case of the *Scenedesmus* mutant studied by Satoh and Fork (1983a). This approach also fails to explain why ATPase inhibitors, which block the utilization rather than the formation of trans-membrane pH gradients, appear to act in the same way as uncouplers.

If the problem of the *Scenedesmus* mutant is set aside, the question of the sensitivity of the light-driven changes seen in DCMU-poisoned algae to ATPase inhibitors and uncouplers could, in principle at least, be reconciled with the conventional LHC-II phosphorylation model by postulating that inhibition of cyclic phosphorylation leads to a stimulation, or release of constraints on,

chlororespiration which then results in an increased reduction of the PQ pool that over-rides oxidation by Light 1. Given the presence of sufficiently high endogenous levels of ATP, LHC-II might then be expected to become rephosphorylated and the cells to relax from State 1 to State 2. The possibility that substrate-level phosphorylation associated with early steps in the chlororespiratory pathway might play some part in the maintenance of chloroplast ATP concentrations under these conditions should not be neglected. Chloroplast adenylate kinase activity might also play a role in this process (J. Bennett, 1986 – personal communication).

The main advantage of such an explanation is that it removes the requirement for an energized state while remaining consistent with the observation that algae appear to be in State 2 in the dark. It also offers, as will be explained in the next section, a possible link between the changes seen in the green algae and those seen in blue-green and red algae.

Excitation energy distribution in red and blue-green algae

Measurements of low temperature emission spectra (Murata, 1969; Ley and Butler, 1980; Biggins, 1983; Fork and Satoh, 1983; Satoh and Fork, 1983c), action spectra of O_2 evolution (Ried et al., 1977), kinetics of cytochrome f oxidation (Biggins 1983; Fork and Satoh, 1983) and flash-induced O_2 evolution (Ley 1984) all indicate that red and blue-green algae undergo State 1/State 2 transitions analogous to those seen in green algae. Like green algae, their excitation energy distribution in the dark appears to be similar to that of State 2-adapted algae (Ried and Reinhardt 1977; Williams et al., 1981; Fork and Satoh 1983; Satoh and Fork, 1983b).

State 1/State 2 transitions in the red and blue-green algae tend, however, to involve much larger changes in excitation energy distribution (Ried et al., 1977; Ley, 1980). They also take place much more rapidly. Ried and Reinhardt (1977, 1980), for example, reported transition times of 2–5s for State 1 adaptation and 10–20s for State 2 adaptation in several different classes of red algae. The corresponding transition rates in blue-green algae appears to be rather slower but are still much more rapid than those seen in green algae (Fork and Satoh, 1983; Catt et al., 1984b). Biggins and Bruce (1985) have reported that the state changes occurring in the red algae *Porphyridium* can be completed, under optimal conditions, in times of 1–2s. They also showed, using single-flash measurements, that the formation of State 1 and State 2 involved intermediates with lifetimes of several hundred milliseconds and less than 30 ms, respectively.

State 1/State 2 transitions in red and blue-green algae can be conveniently monitored by measurements of light-induced fluorescence increases in DCMU-poisoned cells in a similar manner to that described above for green algae. Again, these increases are blocked by the addition of uncouplers and ATPase inhibitors (Papageorgiou and Govindjee, 1967, 1968b; Mohanty and Govindjee, 1973a, b; Fork and Satoh, 1983). Some confusion has arisen regarding the
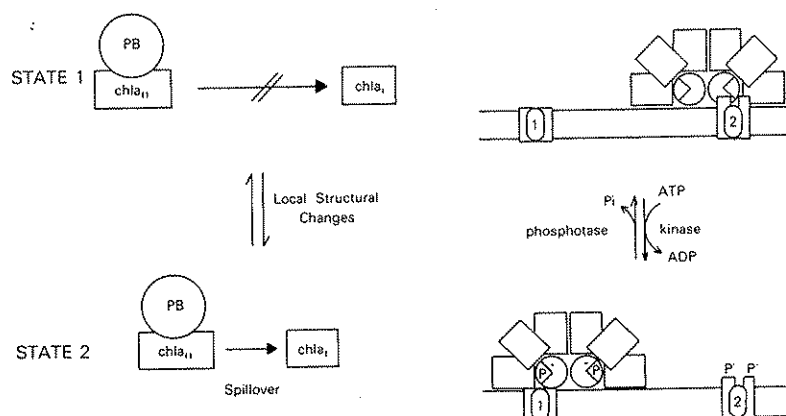
Fig. 5. (a) Diagrammatic representation of the model proposed by Biggins et al. (1984) to account for State 1/State 2 transitions in red and blue-green algae. The model is based on the idea that excess coupled cyclic electron flow around PS I leads to localized structural changes in the arrangement of the pigment bed that facilitate spillover of excess excitation energy from PS II to PS I. (b) Model proposed by Allen et al. (1986) for phycobilisome-containing organisms. State 2 adaptation is suggested to be the result of phosphorylation (i) of the 18.5 kDa polypeptide that forms one sector of the phycobilisome core and (ii) of a surface-exposed component of the intrinsic antenna of PS II. Mutual electrostatic repulsion, or conformational dissociation, between the phycobilisome and PS II is then thought to alter the pathway of energy transfer in favour of PS I at the expense of PS II.

effects of different ATPase inhibitors owing to a failure to check their ability to penetrate into algal cells. In our experience, phlorizin does not readily enter alga cells and hence cannot block State 1/State 2 transitions, while DCCD and TPTC, when used at appropriate levels, are extremely effective at blocking transitions from State 2 to State 1 (unpublished results).

Initial attempts to demonstrate the existence in red and blue-green algae of pigment-protein complexes capable of undergoing reversible light-driven phosphorylation akin to those exhibited by LHC-II were largely unsuccessful. Biggins et al. (1984) were able to identify at least twelve phosphorylated polypeptides in *Porphyridium* but their phosphorylation appeared to be independent of whether the algae were illuminated by Light 1 or Light 2. Schuster et al. (1984) reported the light-independent phosphorylation of a 34 kDa chlorophyll *b*-containing polypeptide in cells of the prokaryote *Prochloron* isolated from *Diplosma virens*. The cells did not, however, appear to undergo State 1/State 2 transitions. The same authors also demonstrated light-independent kinase activity in the blue-green algae *Fremyella diplosiphon* but its relationship to State 1/State 2 changes remains to be established.

In view of their failure to identify light-induced phosphorylation of pigment-protein complexes in red and blue-green algae, Biggins and his co-workers proposed the model for State 1/State 2 transitions in red and blue-green algae shown in Fig. 5a. The model is based on the idea that Light 1 causes an

;over-stimulation of cyclic electron transport. Proton fluxes associated with such transport are then thought to lead to localized changes in the electrical balance of the thylakoid membrane that in some way bring about conformational changes reducing spillover of excess excitation energy from PS II to PS I. This model has the advantage of providing a simple explanation of why dark-adapted algae appear to be in State 2. This has, however, to be set against the fact that there appears to be no obvious reason to suppose that cyclic electron transport leads to changes in membrane energization that differ from those brought about by non-cyclic electron transport. In this context, it is noteworthy that addition of reduced electron transport co-factors such as DAD and PMS, which are believed to stimulate cyclic electron transport, to green (Mohanty and Govindjee, 1973c) and blue-green algae (Papageorgiou, 1975) leads to a light-driven, uncoupler-sensitive lowering of PS II fluorescence emission of the type associated with high energy quenching rather than the increase in fluorescence yield predicted by the cyclic electron transport model.

Allen and his co-workers (Allen et al., 1985; Sanders et al., 1986) reported the light-dependent phosphorylation of a 15 kDa and a 18.5 kDa polypeptide in the blue-green algae *Synechococcus* 6301 (*Anacystis nidulans*). They tentatively identified these two polypeptides as an antenna chlorophyll-binding protein of PS II and the linker protein of the phycobilosome respectively. Sanders and Allen (1986) have since confirmed that the 18.5 kDa polypeptide is a phycobilosome component. Low temperature fluorescence measurements carried out on thylakoid preparations incubated in the light in the presence of ATP and FCCP prior to freezing show that polypeptide phosphorylation is accompanied by a decrease in fluorescence from PS II and an increase in fluorescence from PS I of the type consistent with the occurrence of a transition from State 1 to State 2. Further support for the phosphorylation model has come from photoacoustic measurements of modulated O_2 evolution in which addition of the phosphatase inhibitor NaF has been shown to block State 1 adaptation in the blue-green alga *Nostoc* (Canaani, 1986).

On the basis of their observations, Allen et al. (1985) proposed the model shown in Fig. 5b in which the transition from State 1 to State 2 is triggered by a phosphorylation of the phycobilosome-linker protein and a surface-exposed pigment of the PS II antenna complex. This, it is suggested, leads to a transfer of the phycobilosome from the PS II to the PS I light-harvesting apparatus. Several lines of evidence, however, appear to be incompatible with the idea of phycobilisome transfer. Williams et al. (1981) showed that the fluorescence changes associated with State 1 adaptation in *Anacystis nidulans* were essentially the same if they were monitored by excitation via chlorophyll or phycocyanin suggesting that they involved changes in the interaction between the bulk chlorophyll *a* components of PS II (Chl a_{II}) and of PS I (Chl a_I) rather than between the phycobilisomes and the chlorophyll-cores of the light-harvesting units. Bruce et al. (1985) have come to a similar conclusion on the basis of time-resolved picosecond fluorescence measurements on red and blue-green algae. (Also see Bruce et al., 1986). Bruce and Biggins (1985) reported differen-

ces in the low temperature linear dichroism spectra of *Anacystis* cells chemically fixed in State 1 or State 2 which they suggested might reflect changes in orientation of the allo-phycocyanin core of the phycobilisomes. None of these observations, however, preclude the possibility that phosphorylation triggers conformational changes that facilitate interaction between Chl a_{II} and Chl a_I.

Analyses of the low temperature fluorescence emission of *Porphyridium* performed by Ley and Butler (1980) tend to support the idea that State 1/State 2 transitions in red algae reflect spillover changes rather than exchange of pigments between the PS II and PS I. Mullineaux et al. (1986) have, however, reported changes in the fluorescence induction curves of State 1 and State 2 adapted *Synechococcus* cells which they believe to be consistent with changes in the absorption cross-sections of the two photosystems. Canaani (1986) is also of the opinion that it is changes in absorption cross-section that predominate during the State 1/State 2 transitions she observes in *Nostoc*.

Acceptance of a pigment-protein phosphorylation model for red and blue-green algae raises the same question as posed for green algae. Namely, how can such an idea be reconciled with the observation that addition of uncouplers and ATPase inhibitors to DCMU-poisoned cells appears to lead to a transition from State 1 to State 2? In the case of the green algae, it was suggested that such agents led to a stimulation of chlororespiration. Hirano et al. (1980) reported that the respiratory and photosynthetic electron transport chains of blue-green algae are connected at the level of PQ (see also Scherer and Boger (1982), Scherer et al. (1982) and Matthis et al. (1984)). If so, stimulation of the flow of reductants from the respiratory chain into the PQ pool, as a result of a removal of respiratory control following the inhibition of cyclic photophosphorylation, given adequate cytoplasmic ATP levels, could give rise to the necessary conditions for pigment-protein complex phosphorylation and the return of the cells to State 2. Some support for this idea comes from a study, performed by Imafuku and Katoh (1976), in which the addition of the uncoupler CCCP to DCMU-poisoned *Anabaena variabilis* was shown to result in a halving of cellular ATP concentrations and a concomitant relief of photo-inhibition of respiratory electron flow. Measurements of State 1/State 2 transitions in starved *Synechococcus* cells performed by Mullineaux and Allen (1986) also provide strong support for the idea that the respiratory chain can influence the redox level of the PQ pool in this way.

In addition to the phosphorylation changes seen in the blue-green algae, Allen and his co-workers (Holmes et al., 1986; Holmes and Allen, 1986) and Loach et al. (1984) have reported that phosphorylation of a bacteriochlorophyll pigment protein complex of the photosynthetic bacterium *Rhodospirillum rubrum* is associated with changes in the cooperativity of neighbouring photosynthetic units. Allen and Holmes (1986) have attempted to unify the State 1/State 2 transitions seen in higher plants and green algae, the corresponding changes occurring in red and blue-green algae, and changes in photosynthetic unit connectivity in photosynthetic bacteria by the suggestion that all these phenomena reflect related changes brought about by light-driven phosphoryla-
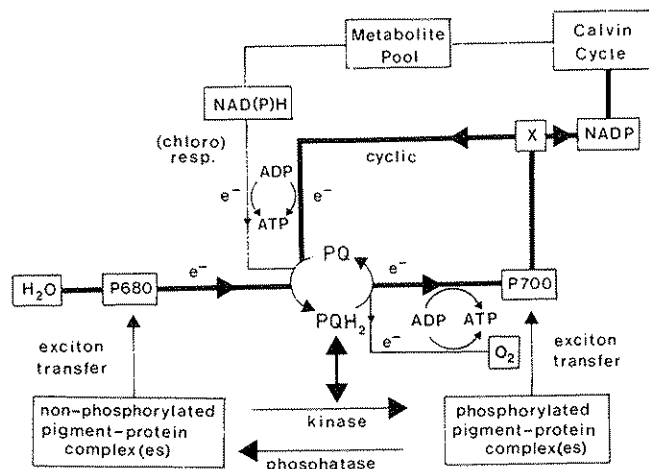
Fig. 6. General model for the control of State 1/State 2 transitions in photosynthetic algae. See text for explanatory details.

tion and the mutual electrostatic repulsion of peripheral and intermediate light-harvesting complexes. More detailed information regarding the phosphorylation changes occurring in the algal and bacterial systems is required in order for this hypothesis to be fully evaluated.

General model

There are currently two main schools of thought regarding the molecular basis of State 1/State 2 transitions. One is based on the idea that state adaptation is a result of changes in excitation energy distribution between the two photosystems associated with the phosphorylation of certain key pigment-protein complexes and the other that it is associated with structural changes in membrane organization triggered by excess cyclic electron transport. The similarity between the light-driven fluorescence yield changes seen in green, blue-green and red algae strongly argues for a common basis for State 1/State 2 adaptation in the different systems. There is, at present, insufficient evidence to allow a final decision between the two proposed mechanisms. The recent identification of pigment-protein complexes that undergo light-driven phosphorylation in phycobilisome-containing organisms tends, however, to favor the pigment-protein phosphorylation model.

 A possible scheme for the control of excitation energy distribution in photosynthetic algae is presented in Fig. 6. The state of the kinase system controlling the phosphorylation of the pigment-protein complex(es), as in the conventional LHC-II phosphorylation model, is determined by the redox state of the PQ pool. This, in turn, is determined by the rate of flow of electrons into

the PQ pool from PS II and the (chloro)respiratory pathway, on the one hand, and their rate of flow out of the pool by non-cyclic transport through PS I and by respiratory flow and/or leakage to O_2. In the case of the cyanobacteria, Matthijs et al. (1984) have provided evidence for the occurrence of coupled electron flow through the PQ pool to a cell-membrane bound cytochrome c:O_2 oxidoreductase system. Matthijs et al. suggest that reducing equivalents are transferred from PQ to the oxidase system by a soluble protein electron-shuttle. However, as they point out, the possible existence of a second cytochrome c:O_2 oxidoreductase system located on the thylakoid membrane cannot be completely excluded. Omata and Murata (1985) have presented evidence indicating that the cytochrome oxidases of *Anacystis* and *Synechocystis* PCC 6714 are located exclusively in their thylakoid membranes. Godde (1982) has demonstrated the existence of NADH:PQ oxidoreductase activity in the chloroplast of the green alga *Chlamydomonas* and Ohyama et al. (1986) and Shinozaki et al. (1986) have reported the existence of genes coding for what appear to be NADH:PQ oxidoreductase components in the chloroplast genomes of liverwort and tobacco, respectively. The question as to whether such systems also possess terminal oxidases as in the blue-greens has, however, yet to be answered.

The scheme shown in Fig. 6 contains a number of potential control points. Kinase activation would be expected to be dependent on the initial distribution of excitation energy between PS I and PS II (i.e. Light 1 and Light 2); the availability of $NADP^+$ as a terminal electron acceptor for non-cyclic electron transport; the rate of (chloro)-respiration and the rate of flow of electrons from the PQ pool to molecular O_2. In addition to the requirement for ATP for pigment-protein phosphorylation, ATP is likely to play a key role in determining the relative efficiencies of these electron transport pathways. As pointed out above, ATP concentrations probably play an important part in controlling the rate of respiratory electron flow through the PQ pool. They may also influence the availability of $NADP^+$, and hence the relative efficiencies of non-cyclic and cyclic electron transport, by control of the re-oxidation of NADPH in the Calvin cycle where the ATP-requiring phosphoglycerate kinase reaction is tightly coupled to the NADPH-oxidizing glyceraldehyde-3-phosphate dehydrogenase reaction.

The main advantage of the present model over the conventional LHC-II phosphorylation model is that it reconciles the State 1/State 2 changes seen in red and blue-green algae with those seen in the green algae. It also brings these changes into line with those seen in higher plants. Another important feature is that it emphasizes the fact that ATP concentrations in photosynthetic organelles and cells, as might be expected in such metabolically active systems, tend to be fairly high and that pigment-protein phosphorylation is normally determined by the availability of reducing equivalents to the PQ pool rather than ATP availability. At the same time, it explains how a lowering of ATP concentrations, or the creation of a demand for additional ATP synthesis, might alter the state of the pigment-protein kinase system so as to favour increased ATP synthesis even under apparently adverse illumination conditions.

Finally, it is perhaps worth noting that as the kinase system is operative both in the dark and in State 2 that instead of thinking of State 1/State 2 transitions as being under the control of a light-activated kinase, it might be more appropriate to think of them as being controlled by a kinase that is switched off by Light 1. This underlines the fact that the pigment-protein complexes are likely to be at least partially phosphorylated under in vivo conditions even in State 1. This might explain some of the difficulties that have been encountered in demonstrating their light-dependent phosphorylation in the red and blue-green aglae.

Acknowledgements

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