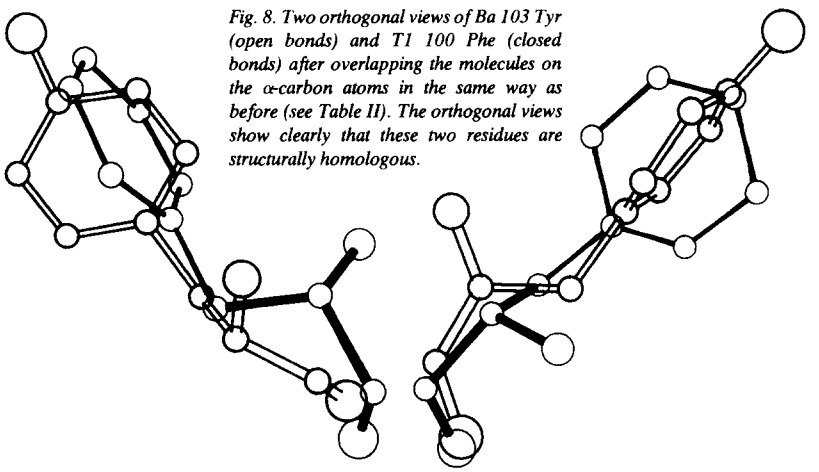

Fig. 8. Two orthogonal views of Ba 103 Tyr (open bonds) and T1 100 Phe (closed bonds) after overlapping the molecules on the α-carbon atoms in the same way as before (see Table II). The orthogonal views show clearly that these two residues are structurally homologous.

way as for the optimum Ba/T1 α-carbon superposition, we find that residues Ba 103 Tyr and T1 100 Phe are structurally equivalent (see Fig. 8).

Our structural comparisons therefore generally support the previous conclusion on ribonuclease homology. There is an homologous family of ribonucleases, separate from the pancreatic ribonucleases that spans the prokaryotes and eukaryotes. This family can be divided into two major classes that reflect the long separation of prokaryotes and eukaryotes. There exists within these main groupings, subsets of more closely related enzymes, Ba and Bi from the Barnase group, T1, Ms and C2 from the T1 group. St is very different to both Ba and T1, but we think that it is closer to Ba, because the St β-sheet is generally more like that of Ba than T1.

Work is underway at present in our laboratories to refine the native ribonucleases and substrate analogue complexes. This will enable us to understand the ribonuclease mechanisms in more detail and to compare the mechanisms of the microbial and pancreatic ribonucleases as well as differences that may occur within the microbial group. Perhaps these studies may resolve the questions of how the binding sites and the catalytic sites have evolved in these enzymes.

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References

1 Richards, F. M. and Wyckoff, H. W. (1971) *The Enzymes*, 3rd edn 4, 647–806
2 Fersht, A. (1977) *Enzyme Structure and Mechanism*, W. H. Freeman & Co
3 Blackburn, P. and Moore, S. (1982) The Enzymes, 3rd edn 15, 317–433
4 Hartley, R. W. (1980) *J. Mol. Evol.* 15, 355–358
5 Hartley, R. W. and Barker, E. A. (1972) *Nature New Biol.* 235, 15–16
6 Aphanasenko, G. A., Dudkin, S. M., Kaminir, L. B., Leshchinskaya, I. B. and Severin, E. S. (1979) *FEBS Lett.* 97, 77–80
7 Yoshida, N., Sasaki, A., Rashid, M. A. and Otsuka, H. (1976) *FEBS Lett.* 64, 122–125
8 Takahashi, K. (1965) *J. Biol. Chem.* 240, 4117–4119
9 Watanabe, H., Ohgi, K. and Irie, M. (1982) *J. Biochem.* 91, 1495–1509
10 Hashimoto, J. and Takahashi, K. (1974) *J. Biochem.* 76, 1359–1361
11 Sato, S. and Uchida, T. (1975) *Biochem. J.* 145, 353–360
12 Ohgi, K., Watanabe, H., Emman, K., Yoshida, N. and Irie, M. (1981) *J. Biochem.* 90, 113–123
13 Miyamoto, K., Arata, Y., Matsuo, H. and Narita, K. (1981) *J. Biochem.* 89, 49–59
14 Egami, F., Oshima, T. and Uchida, T. (1980) *Mol. Biol. Biochem. Biophys.* 32, 250–277
15 Takahashi, K. and Moore, S. (1982) *The Enzymes* 15, 435–467
16 Arata, Y., Kimura, S., Matsuo, H. and Narita, K. (1979) *Biochemistry* 18, 18–24
17 Inagahi, F., Kawasno, Y., Shimada, I., Takahashi, K. and Miyazawa, T. (1981) *J. Biochem.* 89, 1185–1195
18 Heinemann, U. and Saenger, W. (1982) *Nature* 299, 27–31
19 Stroud, R. M. (1974) *Scientific American* 231, 74–78
20 Mauguen, Y., Hartley, R. W., Dodson, G. G., Dodson, E. J., Bricogne, G., Chothia, C. and Jack, A. (1982) *Nature* 297, 162–164
21 Yamamoto, Y., Iwahashi, K., Nakamura, K., Iitaka, Y. and Mitsui, Y. (1981) *Nucl. Acids Res. Symp. Ser.* 10, 227–231
22 Nakamura, K., Iwahashi, K., Yamamoto, Y., Iitaka, Y., Yoshida, N. and Mitsui, Y. (1982) *Nature* 299, 564–566
23 Sevcik, J., Gàsperik, J. and Zelinka, J. (1982) *Gen. Physiol. Biophys.* 1, 255–259
24 Gasperik, J., Muranyova, M., Suranova, B., Zelinka, J. and Kostka, V. (1982) *Biologia* (Bratislava) 37, (8), 769–775
25 Levitt, M. and Chothia, C. (1976) *Nature* 261, 552–558
26 Kabsch, W. (1976) *Acta. Cryst.* A36, 922–923
27 Richards, F. M., Wyckoff, H. W., Carlson, W. D., Allewell, N. M. and Mitsui, Y. (1971) *Cold Spring Harbour Symp. on Quant. Biol.* 36, 35–43
28 James, M. N. G., Delbaere, L. T. J. and Brayer, G. D. (1978) *Can. J. Biochem.* 56, 396–402
29 Wlodawer, A., Bott, R. and Siölin, L. (1982) *J. Biol. Chem.* 257, 1325–1332

Protein phosphorylation – carburettor of photosynthesis?

John F. Allen

Even under changing light regimes, plants are able to partition absorbed excitation energy equitably between the two photochemical reactions of photosynthesis. How this 'carburettor' works was a mystery until phosphorylation of a light-harvesting, chlorophyll-protein was found to be controlled by the redox state of plastoquinone. This redox control can explain the operation of the 'carburettor', and the hypothesis is in agreement with some pertinent experimental results.

The operation, in series, of two photochemical reactions is now generally accepted as the means by which light energy drives non-cyclic electron transfer in photosynthesis in plants. This 'Z-scheme' model of photosynthetic electron transport (Fig. 1) accounts for the behaviour of electron carriers situated between the photochemical reactions, or 'photosystems'; these carriers are oxidized by photosystem 1 and reduced by photosystem 2.

The Z-scheme also accounts for the curious response of photosynthesis to lights of differing wavelength: light in the far-red region of the spectrum ($\lambda > 680$ nm) is photosynthetically rather ineffective on its own, though its effectiveness is greatly increased if a second beam of shorter-wave light is also employed. This enhancement of the photosynthetic yield of one beam by another (termed 'Emerson enhancement' after its discoverer) depends upon far-red light being absorbed more by photosystem

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1 than by photosystem 2. Thus photosystem 1 is limited in far-red light by electron flow from photosystem 2, while photosystem 2 is limited in turn by its poor light-harvesting ability. A supplementary beam of shorter-wave light provides the excitation energy necessary for photosystem 2, and photosystem 2 can then provide electrons for turnover of photosystem 1. The effectiveness of the original far-red light is thereby enhanced, and the rate of photosynthesis in both beams of light is greater than the sum of the rates in each beam alone.

Balancing the photosystems

The phenomenon of enhancement served as a major piece of evidence for Z-scheme electron transport and is still unexplained by any alternative hypothesis. The Z-scheme explanation of enhancement leads, however, to a further problem, described by Myers as the 'carburettor' problem[1]. Since the two photosystems have distinct absorption and action spectra, almost any randomly-chosen wavelength should favour one photosystem or the other, and photosynthetic quantum yield should be greatly dependent on wavelength over its entire spectral range. In practice this is not the case: quantum yield is high and relatively independent of wavelength. Thus, in order to retain our Z-scheme explanation of enhancement we must suppose that a carburettor-like mechanism exists for equitable partitioning of excitation energy between the photosystems. Enhancement itself is an indication of imbalance of excitation distribution and is found only at wavelengths (above 680 nm) where the carburettor is no longer effective.

Readjustment of the relative excitation of the two photosystems was first described by Bonaventura and Myers[2] and by Murata[3]. One ingenious experiment of Bonaventura and Myers serves here to illustrate the carburettor's operation. The results of this experiment[2] are depicted in Fig. 2. Cells of the green alga *Chlorella pyrenoidosa* were illuminated by 645 nm light that was modulated ('chopped') at a frequency of 13 Hz. During the first few minutes of illumination, the photosynthetic rate increased while chlorophyll fluorescence decreased. This was taken as an indication of slow adaptation to 645 nm light: photosystem 2 was initially light-saturated and therefore highly fluorescent, while slow redistribution of excitation energy to photosystem 1 decreased photosystem 2 fluorescence and increased photosynthetic yield of oxygen. This adaptation to photosystem 2-light (or 'light 2') was termed a transition to a 'light 2-state' or, more simply, to 'state 2'.

In the same experiment (Fig. 2), superimposition of an unmodulated, background beam of 710 nm light (absorbed primarily by photosystem 1, hence termed 'light 1') produced a transient increase in oxygen yield and a decrease in fluorescence: these effects are easily explained as an increase in turnover and hence of electron-accepting capacity of photosystem 1. Additional, slower effects of light 1 were also seen: both oxygen yield and photosystem 2 fluorescence increased. This was taken as an indication of an adaptation to light 1, that is, as a transition to a 'light 1-state', or 'state 1': excitation energy was apparently being redistributed from the light-saturated photosystem 1 to the light-limited photosystem 2. With the cells in state 1, removal of the background light 1 caused a rapid decrease in oxygen yield and an increase in fluorescence, but oxygen yield then increased slowly as fluorescence decreased. Excitation energy was being redirected once more to photosystem 1: a second transition to state 2 was taking place.

The phenomenon of state 1–state 2 transitions has been encountered in red algae[3] and cyanobacteria[4], as well as in green algae[2] and higher plants[5]. Clearly a 'carburettor' for controlled excitation distribution is a requirement of all photosynthetic organisms that possess two photosystems. No plausible mechanism for this phenomenon was available until recently, although Duysens suggested more than ten years ago[6] that such a mechanism might involve changes of redox state of an electron carrier situated between the two photosystems. In the last three years it has become clear that Duysens's suggestion is likely to be correct, but the identity of the electron carrier and the means of control have come from what would previously have seemed a wholly unexpected direction.

Phosphorylation of LHCP

In 1977, Bennett reported the phosphorylation of certain chloroplast membrane proteins and identified the most prominent phosphoprotein of pea chloroplasts as a 25 000-molecular weight component of light-harvesting, chlorophyll *a*/*b*-binding protein, or 'LHCP'[7]. The phosphorylation site is a threonyl residue in a segment of LHCP exposed to the surface, and the phosphorylation reaction is catalysed by a membrane-bound protein kinase[8]. The dephosphorylation (phosphatase) reaction is also catalysed by a membrane-bound enzyme[9]. The kinase, unlike the phosphatase, was found to have a light-dependent activity that was inhibited by the electron transport inhibitor, DCMU[10]. If ATP is added as substrate for the reaction, protein phosphorylation is unaffected by uncouplers of photophosphorylation[7,8].

The function of this protein phosphorylation remained an enigma for several years after its discovery, and the first indications that the function might be regulation of excitation energy transfer were published by Bennett *et al.*[11] and by Horton and Black[12]. Both groups showed that chlorophyll fluorescence yield at room temperature decreases in pea chloroplast preparations when ATP is added. The conditions were those required for protein phosphorylation: light, magnesium ions, and a functional photosystem 2. Moreover, the effect of ATP was insensitive to uncouplers. Similar effects of ATP could be seen on chloroplast flourescence emission spectra, obtained after cooling samples to 77 K[11,12]. The spectra suggested that LHCP

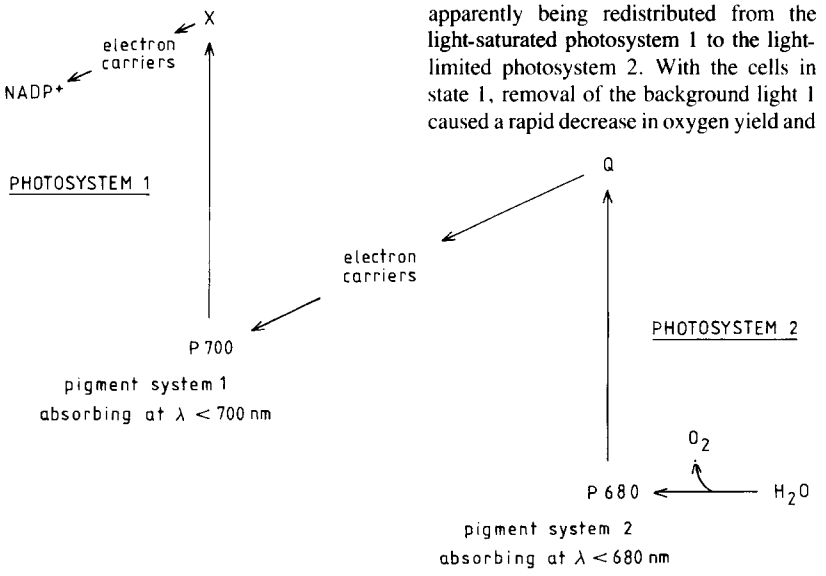

Fig. 1. The 'Z-scheme' model of photosynthetic electron transport (first proposed by Hill and Bendall – see Ref. 1) showing the sequence of electron carriers plotted on a scale of redox potential. Transfer of electrons from water to NADP+ requires two photochemical reactions which operate in series. Each photochemical reaction has its own light-absorbing pigment system, and the two pigment systems have different absorption spectra.

phosphorylation brought about a redistribution of absorbed excitation energy in favour of photosystem 1 and at the expense of photosystem 2.

In retrospect, phosphorylation-dependent redistribution of excitation energy can be seen to resemble closely the adaptive transition to state 2. However, a connection between LHCP phosphorylation and state 1–state 2 transitions was not established until it had become clear that the phosphorylation reaction was under redox control: the identity of the controlling electron carrier then made the connection virtually inescapable.

Regulation by redox state of plastoquinone

The light-dependence and DCMU-sensitivity of LHCP phosphorylation[10] suggested that the protein kinase was activated in some way by non-cyclic electron transport. The possibility that activation depended on a component of the proton motive force could immediately be discounted on the grounds of insensitivity to an uncoupler. An alternative possibility, that phosphorylation proceeded only when some electron carrier had been reduced, prompted Allen *et al.*[13] to investigate the effects of oxidizing and reducing conditions on LHCP phosphorylation and on the fluorescence changes with which it had become associated. It was found that the reductant dithionite activates the protein kinase in darkness and so causes excitation redistribution to photosystem 1[13]. Similarly, the oxidant ferricyanide inhibits light-driven protein phosphorylation and the attendant changes in low-temperature fluorescence[13].

The failure of $DCPIPH_2$ to overcome inhibition by DCMU suggested that the regulatory electron carrier lay between the photosystems[12,13]: $DCPIPH_2$ donates electrons to photosystem 1 and would be expected to activate phosphorylation if a component of photosystem 1 were involved. While DCMU inhibits protein phosphorylation, the electron transport inhibitor DBMIB at low concentrations (sufficient to inhibit oxidation of plastoquinone) does not[13]. This suggested that plastoquinone itself is the key component of the chain, a conclusion reinforced by activation of the kinase by a number of brief flashes of light just sufficient to cause reduction of the plastoquinone pool[13]. Duroquinol, a specific donor to plastoquinone, activated LHCP phosphorylation and permitted the ATP-dependent fluorescence decrease that indicates excitation transfer to photosystem 1[14].

Perhaps the most compelling evidence for control of LHCP phosphorylation by

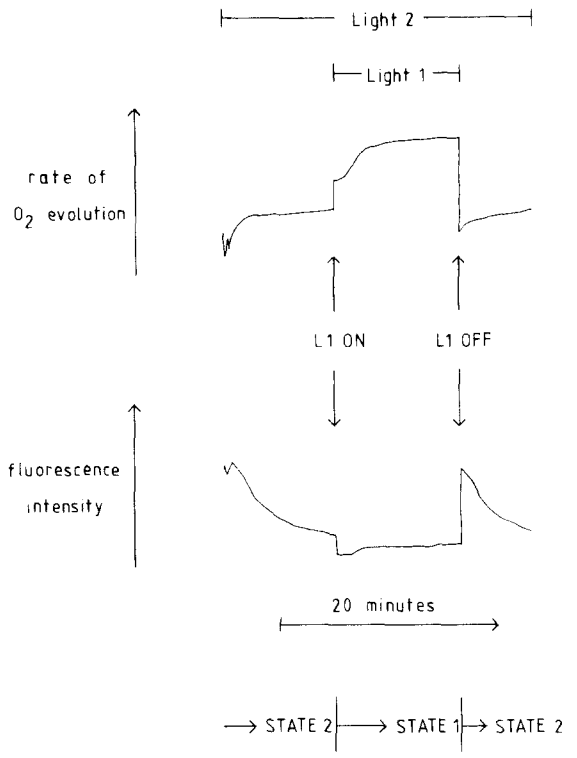

Fig. 2 State 1-state 2 transitions in Chlorella pyrenoidosa, *as described by Bonaventura and Myers*[2]. *Oxygen evolution and fluorescence (at 686 nm) were recorded at the frequency (13 Hz) of modulated 'light 2' (645 nm). Addition of continuous 'light 1' (710 nm) causes slow, reversible changes in oxygen evolution and fluorescence: these indicate redistribution of excitation energy between the two photosystems, as described in the text.*

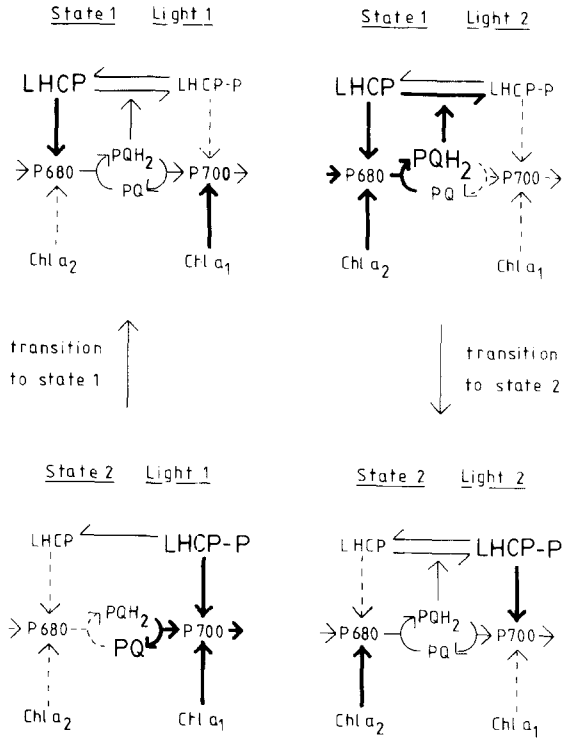

Fig. 3 Phosphorylation and dephosphorylation of LHCP as the basis of state 1–state 2 transitions, as described in the text. Chl a_1 = antenna chlorophyll of photosystem 1; Chl a_2 = antenna chlorophyll of photosystem 2; P680 = reaction centre chlorophyll of photosystem 2; P700 = reaction centre chlorophyll of photosystem 1; PQ = plastoquinone; PQH_2 = plastoquinol (plastohydroquinone); LHCP = light-harvesting chlorophyll a/b-binding protein; LHCP-P = phosphorylated LHCP.

plastoquinone redox state has been obtained by potentiometric redox titration of both LHCP phosphorylation and the fluorescence decrease in pea chloroplast membranes[15]. The midpoint potential for both dependent variables was found to be about 0 mV, while the best titration curve for both sets of data corresponds to that of a two-equivalent carrier. The controlling carrier is indistinguishable from plastoquinone by these means.

LHCP phosphorylation and state 1–state 2 transitions

If protein phosphorylation couples excitation energy distribution to the redox state of plastoquinone, then a possible mechanism for state 1–state 2 transitions becomes apparent. Plastoquinone will be reduced if excitation distribution happens to favour photosystem 2. If plastoquinone reduction activates LHCP phosphorylation, and if LHCP phosphorylation in turn causes increased excitation transfer to photosystem 1, then the initial imbalance in excitation distribution will tend to be self-correcting. Conversely, if photosystem 1 is favoured (by the introduction of light 1, for example) then plastoquinone will become oxidized, LHCP phosphorylation will cease, and net dephosphorylation of LHCP will cause excitation energy to be transferred back to the otherwise rate-limiting photosystem 2.

This hypothesis was offered independently by Allen *et al.*[13] and by Horton and Black[16], and is depicted here in Fig. 3. The upper, left-hand diagram of Fig. 3 shows an equal input of excitation energy to P680 and P700, the reaction centre chlorophylls of photosystems 2 and 1 respectively. LHCP is predominantly in its dephosphorylated state. This corresponds to state 1, and to the dark state in the experiment of Bonaventura and Myers (Fig. 2). Illumination with light 2 then causes faster turnover of P680 than of P700. This imbalance is shown in the upper, right-hand diagram of Fig. 3, which also depicts reduction of plastoquinone and activation of LHCP phosphorylation. The ensuing phosphorylation of LHCP is held to be the molecular basis of the adaptive transition to state 2. In the new steady-state (lower, right-hand diagram of Fig. 3) LHCP exists predominantly in its phosphorylated form, and its excitation energy is transferred primarily to photosystem 1. The transition itself will be accompanied by an increased yield of oxygen (as quantum efficiency increases) and by a decreased photosystem 2 fluorescence yield, exactly as observed (Fig. 2). Introduction of light 1 will then upset the balance by increasing turnover of P700. Plastoquinone will be oxidized, and LHCP phosphorylation switched off (Fig. 3: lower, left-hand diagram). Net dephosphorylation will cause a return of excitation distribution to photosystem 2, thereby resulting in the observed increase in yield of both oxygen and fluorescence. Thus net dephosphorylation of LHCP is the molecular basis of the transition to state 1, and state 1 (in light 1, at least) is an equilibrium state characterized by a predominance of dephosphorylated LHCP (Fig. 3: upper, left-hand diagram).

Results obtained by Telfer *et al.*[17] are in remarkably good agreement with this hypothesis. Using pea chloroplasts and a modulated fluorescence signal as a guide to changes in excitation distribution, they found that radiolabelling of LHCP with ^{32}P (from [$\gamma^{32}P$]ATP) did indeed accompany the fluorescence decrease that indicated transition to state 2. Subsequent addition of light 1 brought about a decrease in the level of phosphorylation of LHCP: this accompanied the fluorescence rise that indicated transition to state 1. Fig. 4 shows the time-courses of LHCP phosphorylation (transition to state 2) and LHCP dephosphorylation (transition to state 1) obtained by Telfer *et al.*[17]. In the pea chloroplast system used, and after a 20-min equilibration period, changes of the light regime were sufficient to bring about changes in LHCP phosphorylation. The half-time of LHCP phosphorylation in light 2 was 4 min, and the half-time of dephosphorylation in light 1 was 6 min[17]. Further corroboration of the hypothesis is provided by the observation[17] of inhibition of the transition to state 1 by sodium fluoride, an inhibitor of the dephosphorylation reaction[9].

The transition to state 2 in pea chloroplasts has been shown to be dependent on ATP[5,18], and ATP-dependent fluorescence changes in *Chlorella* thylakoids have been shown to resemble the state 2 transition in intact *Chlorella* cells[19].

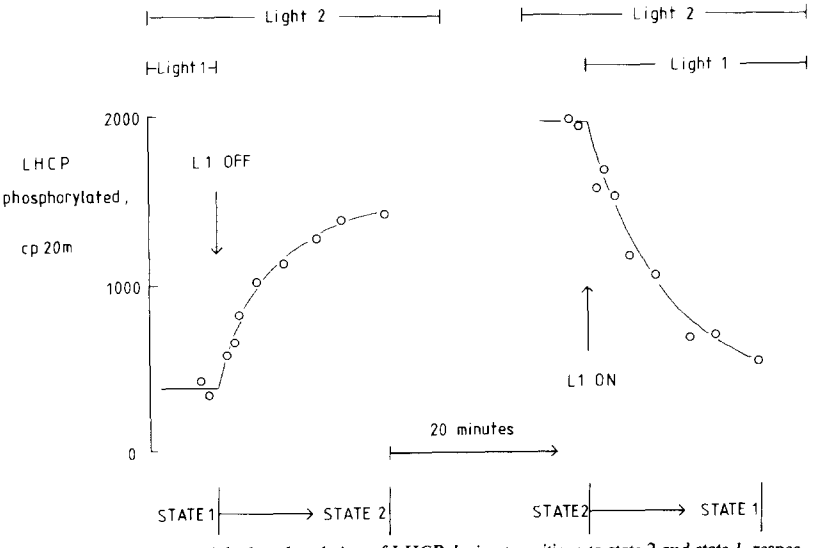

Fig. 4 Phosphorylation and dephosphorylation of LHCP during transitions to state 2 and state 1, respectively. Results obtained by Telfer et al.[17] *with osmotically-shocked pea chloroplasts. 'Light 2' is modulated (90 Hz) blue light; 'light 1' is continuous far-red light (710 nm). The initial light regime was maintained for twenty minutes before light 1 was switched on or off; [γ-^{32}P]ATP was present throughout this initial period of equilibration.*

How does phosphorylation cause changes in excitation distribution?

There is now good evidence that the components of photosynthetic electron transport are far from randomly dispersed throughout the thylakoid membrane[20]. Components of photosystem 2 appear to be concentrated in regions of the membrane that are closely appressed to similar regions of an adjacent membrane, that is, photosystem 2 is found where thylakoids are stacked to form the 'grana' of conventional chloroplast morphology. The unappressed regions of thylakoid membrane ('stroma lamellae') contain components of photosystem 1.

Experimentally-induced transfer of excitation energy from photosystem 2 to photosystem 1 ('spillover') has been associated for some time with unstacking of thylakoids, and is presumed to result from the close physical proximity of the two complexes in completely unstacked membranes with their less ordered distribution of photosystems. In the more physiological, stacked condition, the photosystems are separated and their direct interaction is likely to be small. Barber and co-workers[21] have shown that stacking is likely to result from a balance between short-range forces of attraction and electrostatic forces of repulsion between membrane surfaces. The effect of cations in maintaining membrane stacking can then be explained by their screening of negative charges fixed on the

membrane surface.

It is clearly quite reasonable to suppose that membrane proteins become more negatively charged upon phosphorylation and hence more likely to partition themselves into unstacked regions of the membrane. In this way, migration of phosphorylated LHCP from appressed regions (rich in photosystem 2) to unappressed regions (rich in photosystem 1) might be able to bring about redistribution of excitation energy in favour of photosystem 1. Such models of phosphorylation-controlled excitation distribution in relation to spatial organization of chlorophyll–protein complexes have now been published by a number of authors[21–24].

In support of the view that the charge of LHCP is important in determining excitation distribution, Horton and Black[25] have shown that the effect of phosphorylation on fluorescence is strongly dependent on magnesium ion concentration. Evidence that partial unstacking of thylakoid membranes is a result of LHCP phosphorylation has also been obtained[24,26]. Migration of phosphorylated LHCP from appressed to unappressed regions of membrane is supported by the results of Andersson *et al.*[27], who found the ratio of phosphorylated to non-phosphorylated LHCP to be ten times higher in unappressed than in appressed thylakoids. Phosphorylation-induced migration of a particle 8 nm in diameter has been inferred from freeze–fracture electron micrographs by Kyle *et al.*[24]. Migration of the particle from appressed to unappressed regions of the thylakoid was correlated with fluorescence changes indicative of a state 2 transition. In addition, particle migration was accompanied by the appearance in an unappressed membrane fraction of chlorophyll *b* and LHCP-like polypeptides of 25 000 molecular weight.

In summary, it seems that phosphorylation of LHCP causes it to migrate from grana (stacked) to stroma (unstacked) lamellae, and hence to transfer excitation energy more to photosystem 1 than to photosystem 2. It may even be that the 'function' of thylakoid stacking is primarily to allow a reversible redistribution of light-harvesting complexes between the photosystems[20], and hence to permit balanced excitation distribution under varying conditions of illumination.

How widespread is protein phosphorylation in photosynthesis?

Up to twenty thylakoid polypeptides are phosphorylated under appropriate conditions. Of these, none but LHCP has been unambiguously identified. A 9000 molecular weight polypeptide is reversibly phosphorylated under the same conditions as LHCP[15,17], but its dephosphorylation is slower than that of LHCP, and too slow for it to be implicated in the transition to state I[28]. No other thylakoid phosphoprotein has yet been found to be under redox control[15] or to be influenced by the conditions causing state 1–state 2 transitions[17]. LHCP is known to be phosphorylated in the green algae *Euglena gracilis*[29] and *Chlorella vulgaris*[19] as well in chloroplasts of higher plants.

It is possible that reversible phosphorylation of LHCP has physiological functions other than in adaptations to changing light quality. Since cyclic photophosphorylation directly involves only photosystem 1, redistribution of excitation energy may be necessary in order to maximize the quantum efficiency of ATP synthesis during physiological changes in the relative rate of cyclic and non-cyclic photophosphorylation[30]. The required changes in LHCP phosphorylation under light of constant wavelength can, in principle, be caused by changes of plastoquinone redox state, since this will depend on the availability of an electron acceptor ($NADP^+$) and hence, indirectly, on the demand for ATP by coupled assimilatory reactions such as CO_2-fixation.

Two promising anomalies remain: these lie in the area of prokaryote photosynthesis. Cyanobacteria are devoid of LHCP, and carry out light-harvesting by phycobilins instead of with chlorophyll *b*. Nevertheless, they exhibit state 1–state 2 transitions[4]. The same is true of red algae[3]. The mechanism of adaptation in these organisms must therefore differ substantially from that described here for green plants, though it is clearly still possible that a redox-sensitive phosphorylation reaction is involved. Preliminary studies (Allen, J. F. and Cogdell, R. J. unpublished observations) indicate that a number of photosynthetic purple bacteria reversibly phosphorylate polypeptides of light-harvesting bacteriochlorophyll-protein complexes. Photosynthetic bacteria have a single photosystem and obviously cannot carry out state 1–state 2 transitions: at present the role of this phosphorylation reaction in bacterial photosynthesis is purely a matter for conjecture.

Phosphorylation of light-harvesting pigment-proteins in plants and bacteria is too great a coincidence for one to rule out a common evolutionary origin and homology of function. Perhaps regulation of excitation transfer has further, unsuspected similarities that underly such apparently diverse photosynthetic machinery?

References

1 Myers, J. (1971) *Annu. Rev. Plant Physiol.* 22, 289–312
2 Bonaventura, C. and Myers, J. (1969) *Biochim. Biophys. Acta* 189, 366–389
3 Murata, N. (1969) *Biochim. Biophys. Acta* 172, 242–251
4 Murata, N., Troughton, J. H. and Fork, D. C. (1975) *Plant Physiol.* 56, 508–517
5 Chow, W. S., Telfer, A., Chapman, D. J. and Barber, J. (1981) *Biochim. Biophys. Acta* 638, 60–68
6 Duysens, L. N. M. (1972) *Biophys. J.* 12, 858–863
7 Bennett, J. (1977) *Nature (London)* 269, 344–346
8 Bennett, J. (1979) *Eur. J. Biochem.* 99, 133–137
9 Bennett, J. (1980) *Eur. J. Biochem.* 104, 85–89
10 Bennett, J. (1979) *FEBS Lett.* 103, 342–344
11 Bennett, J., Steinback, K. E. and Arntzen, C. J. (1980) *Proc. Natl Acad. Sci. USA* 77, 5253–5257
12 Horton, P. and Black, M. T. (1981) *Biochim. Biophys. Acta* 635, 53–62
13 Allen, J. F., Bennett, J., Steinback, K. E. and Arntzen, C. J. (1981) *Nature (London)* 291, 25–29
14 Allen, J. F. and Horton, P. (1981) *Biochim. Biophys. Acta* 638, 290–295
15 Horton, P., Allen, J. F., Black, M. T. and Bennett, J. (1981) *FEBS Lett.* 125, 193–196
16 Horton, P. and Black, M. T. (1980) *FEBS Lett.* 119, 141–144
17 Telfer, A., Allen, J. F., Barber, J. and Bennett, J. (1983) *Biochim. Biophys. Acta* 722, 176–181
18 Sinclair, J. and Cousineau, C. (1981) *FEBS Lett.* 136, 213–215
19 Saito, K., Williams, W. P., Allen, J. F. and Bennett, J. (1983) *Biochim. Biophys. Acta* 724, 94–103
20 Anderson, J. M. and Andersson, B. (1982) *Trends Biochem. Sci.* 7, 288–292
21 Barber, J. (1982) *Annu. Rev. Plant Physiol.* 32, 261–295
22 Haworth, P., Kyle, D. J., Horton, P. and Arntzen, C. J. (1982) *Photochem. Photobiol.* 36, 743–748
23 Horton, P. (1983) *FEBS Lett.* 152, 47–52
24 Kyle, D. J., Staehelin, L. A. and Arntzen, C. J. (1983) *Arch. Biochem. Biophys.* 222, 527–541
25 Horton, P. and Black, M. T. (1983) *Biochim. Biophys. Acta* 722, 214–218
26 Biggins, J. (1981) *Biochim. Biophys. Acta* 635, 259–266
27 Andersson, B., Akerlund, H.-E., Jergil, B. and Larsson, C. (1982) *FEBS Lett.* 149, 181–185
28 Steinback, K. E., Bose, S. and Kyle, D. J. (1982) *Archs. Biochem. Biophys.* 216, 356–361
29 Beliveau, R. and Bellemare, G. (1979) *Biochem. Biophys. Res. Commun.* 88, 797–803
30 Allen, J. F. (1983) *CRC Crit. Revs. Plant Sci.* 1, 1–22