Will the Real LHC II Kinase Please Step Forward?

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A protein kinase catalyzes phosphorylation of the chlorophyll-protein complex, light-harvesting complex II (LHC II), one of the most abundant membrane proteins in the world. Apart from an ingenious redox regulatory function that makes photosynthesis more efficient, what do we know about this elusive enzyme?

Plants and algae have evolved a range of mechanisms to deal with the variations in the quality and intensity of light that they encounter over the course of each day. The time scales of these processes vary from a few milliseconds to hours, depending on the type and level of the response. Within chloroplasts, environmental light conditions affect the photochemical reactions of photosynthesis, posttranslational protein modifications, and gene expression. Light conditions also affect nuclear transcription, cytosolic translation, and import of precursor proteins into chloroplasts. In order to maximize the efficiency of photosynthesis and to minimize damage to the delicate infrastructure that absorbs and converts light energy within the chloroplast thylakoid membrane, photosynthetic organisms are able preferentially to direct photons to either chloroplast photosystem I (PS I) or chloroplast photosystem II (PS II). The posttranslational response is one of the more rapid adaptive mechanisms. This mechanism underlies adaptations to wavelength of light known as "state transitions."

The thylakoids of chloroplasts of all plants contain LHC II, an intrinsic membrane protein that binds chlorophylls a and b and functions as the major light-harvesting antenna of photosynthesis (*1*). This protein can be phosphorylated (*2*), and phosphorylation modulates its light-harvesting function in a precise and indispensable way. Many laboratories have contributed insights into such factors as the site(s) of phosphorylation, its functional effects, and its cofactor requirements, and the conclusions seem generally to hold good for an enormous range of species, from the alga *Chlamydomonas* to plants, such as spinach and *Arabidopsis* (*3*). In all cases, the key enzyme that catalyzes LHC II phosphorylation is membrane-bound and redox-regulated. This protein kinase provides a link in a vital posttranslational feedback control mechanism and participates in a redox signaling pathway of developmental and evolutionary significance.

The best model underlying state transitions is based on an observed correlation between preferential excitation of PS II, phosphorylation of LHC II, and subsequent dissociation of phospho-LHC II from PS II. Phosphorylated LHC II then forms a functional complex with PS I, diverting absorbed light energy to PS I at the expense of PS II. If the balance of energy distribution is tipped in the opposite direction by preferential excitation of PS I, then LHC II becomes dephosphorylated and reverts to harvesting light energy for PS II (*4*) (Fig. 1). State transitions are easily visualized as changes in chlorophyll fluo-

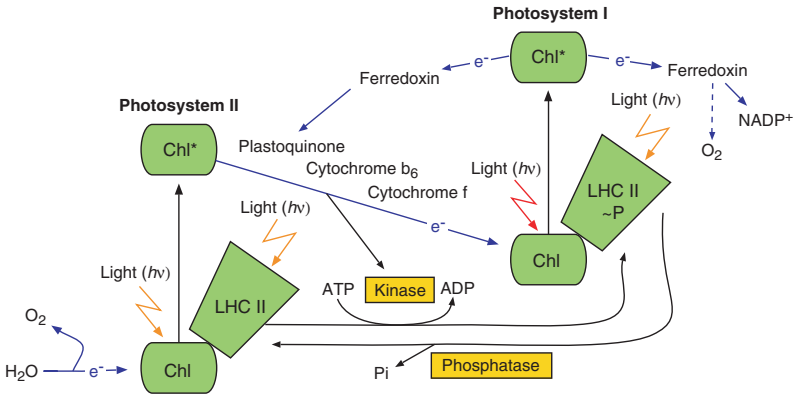

Fig. 1. State transitions and protein phosphorylation. The function of the LHC II kinase-phosphatase system in regulating distribution of light energy between photosystem I (PS I) and photosystem II (PS II) in chloroplasts. The Z scheme for transport of electrons (blue arrows) from water to nicotinamide adenine dinucleotide diphosphate ($NADP^+$) includes two photosystems. The photosystems are points in the electron transport chain where light energy ($h\nu$) generates a chlorophyll (green) excited state (Chl*), thus supplying an electron to, that is reducing, an electron acceptor. Each photosystem has its own array of light-absorbing chlorophyll molecules, together with one reaction center chlorophyll molecule that initiates and powers electron transport. The absorption and action spectra of the two photosystems are different. PS I works with red and near-infrared light (red, jagged arrow), but PS II has a "red-drop" in yield and ceases to work if the wavelength of light (orange arrow) exceeds about 670 nm. LHC II (green) is a pigment-protein complex that constitutes a mobile array of light-harvesting chlorophyll molecules. Phosphorylated LHC II (LHC II~P) supplies absorbed light energy to PS I, and unphosphorylated LHC II supplies light energy to PS II. When electrons leave PS II faster than they are used by PS I, plastoquinone and other electron carriers connecting the two photosystems become reduced. This reduction activates the LHC II kinase (yellow), and phosphorylation of LHC II takes light energy away from PS II and supplies it, instead, to PS I, thus tending to equalize the rates of electron transport through the two photosystems. If the light available then favors PS I, plastoquinone becomes oxidized, the LHC II kinase is switched off, and the LHC II phosphatase reaction (yellow) predominates, returning LHC II to PS II. The redox control of the LHC II kinase maintains maximal efficiency of photosynthesis by distributing chlorophyll molecules between the two photosystems in proportion to their capacity to use light energy to drive electron transport (*4*). This redistribution may also serve to adjust the light-harvesting capacity of the photosystems when PS I is called upon to drive ferredoxin-mediated cyclic electron transport (blue arrows on each side of ferredoxin) for ATP synthesis that proceeds independently of reduction of $NADP^+$ (*43-45*). All these events are intrinsic to chloroplast thylakoid membranes. Phosphorylation-induced changes in molecular recognition (*27*) of membrane-intrinsic protein-protein interactions are likely to decide whether LHC II serves PS I or PS II. ATP, adenosine triphosphate; ADP, adenosine diphosphate.

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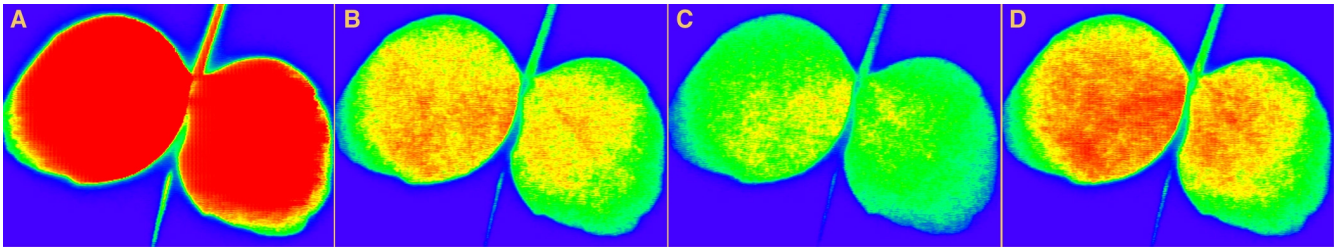

Fig. 2. Imaging state 1-state 2 transitions in pea leaves. Absorbed light energy is redistributed between the two energy-converting systems of photosynthesis. Chlorophyll is the green pigment that harvests and converts light in photosynthesis. Chlorophyll also emits light, as fluorescence, and variations in fluorescence emission report on changes in the efficiency of photosynthesis. At room temperature, changes in the yield of chlorophyll fluorescence come from photosystem II (PS II). In Movie 1, leaves of a young pea seedling were imaged by a camera screened with a 660-nm optical interference filter, detecting chlorophyll fluorescence. (**A**) Fluorescence is initially high (seen as the red color), and then (**B**) falls as the light-harvesting protein LHC II becomes phosphorylated—this redistributes energy to photosystem I (PS I) at the expense of the fluorescent photosystem, PS II. This is the state 2 transition. PS I and PS II are connected in series and must, therefore, function at the same rate. (**C**) A far-red light is then switched on, and fluorescence falls further. This is because PS I absorbs the far-red light: the fluorescent PS II can then use more energy, and less of its energy is wasted as fluorescence. Fluorescence then rises slightly, as the phosphate group is removed from phospho-LHC II, and absorbed energy is redistributed back to PS II. This is the state 1 transition. (**D**) When PS I light is switched off, PS I becomes rate-limiting, and PS II has nowhere to pass electrons, so fluorescence from PS II quickly rises. A second, slow, falling phase is a second transition to state 2: The cycle of state transitions has begun again [see Movie 1 (http://stke.sciencemag.org/cgi/content/full/sigtrans;2002/155/pe43/DC1)].

rescence emission [(Fig. 2) and Movie 1 (http://stke.sciencemag.org/cgi/content/full/sigtrans;2002/155/pe43/DC1)]. Since Bennett first reported LHC II phosphorylation in 1977 (*2*), many laboratories have searched for the protein kinase responsible for phosphorylation of LHC II. Various candidates for the LHC II kinase, some stronger than others, have come and gone. Early contenders were soluble, serine kinases identified in the laboratory of Racker (*5*, *6*). However, the true LHC II kinase activity is clearly membrane-bound (*7*). One notable, later nominee was a 64-kD protein, which is itself phosphorylated and enriched during purification of the cytochrome b_6-f complex (cyt b_6-f) (*8*, *9*), but sequencing studies revealed it to be a polyphenol oxidase (*10*, *11*). A 58-kD protein that copurifies with a complex of PS II proteins was also reported to catalyze phosphorylation of LHC II, as well as other substrates within PS II (*12*, *13*). More recently, a family of three thylakoid-associated kinases (TAKs) has been identified and purified; they, too, catalyze in vitro phosphorylation of LHC II (*14*).

The most recent study of the TAK proteins (*15*) is a systematic investigation of their binding to thylakoid protein complexes, their biochemical involvement in LHC II phosphorylation, and the effect of TAK antisense RNA in partially suppressing state transitions. All three purified TAKs promote phosphorylation of LHC II in vitro (*14*). Redox dependency is indicated by a requirement for dithiothreitol (*14*), both for LHC II phosphorylation and for autophosphorylation of the TAKs themselves. The amino acid sequence of TAKs contains a repeated glycine-serine motif, a conserved motif also found in the animal transforming growth factor-β receptor (*16*). Of the three TAKs, recombinant TAK1 was most clearly involved in binding to both LHC II and cytochrome f, a subunit of the cyt b_6-f , another multimeric component of the thylakoid membrane that is implicated in protein kinase activation (*15*). The current model describing this process is no less complicated than the question of identifying the enzyme(s) involved. Electron transfer through PS II results in the chemical reduction of a pool of plastoquinone within the thylakoid membrane. Binding of plastoquinol to the quinol oxidation site (Q_o) of the cyt b_6-f complex correlates with protein kinase activation (*17*, *18*). The details underlying this mechanism remain to be elucidated.

The conclusion that native TAK1 binds to LHC II and cytochrome f comes from an interaction of these membrane proteins with anti-TAK1 antibodies observed in Western blots (*15*). *Arabidopsis thaliana* transformants that express antisense TAK1 RNA appear normal when grown at low light intensity, but are chlorotic (pale green or yellow), and quickly set seed, or bolt, when grown at higher light intensities (*15*). TAK1 antisense transformants have decreased LHC II phosphorylation, as judged by reaction with antiphosphothreonine antibodies, and this is correlated in vivo with a decreased response of chlorophyll fluorescence emission to changes between light of spectral composition favorable either to PS I or to PS II. State transitions can easily be monitored continuously using the noninvasive technique of pulse-amplitude modulated fluorometry (*19*). However, the data comparing TAK1 antisense transformants with *Arabidopsis* wild-type plants (*15*) consist of a "PS II ratio" of chlorophyll fluorescence excited by a 2-s pulse of white light, measured after exposure of plants to 15 min of red (normally selecting for PS II) or far-red (selecting for PS I) light. The far-red intensity is given as 30 $\mu E\ m^{-2}\ s^{-1}$. Although the ratio of the two fluorescence emissions (after red and far-red illumination) is significantly different between the wild-type and TAK1 antisense plants, the full time course of fluorescence during the transition from one light to another is not recorded. This, together with the unspecified intensity of the red light, presumed to be selective for PS II, leaves room for doubt about the origin of the differences in the fluorescence ratio. Many factors, including nonphotochemical quenching (*20*) and changes in the proportion of PS I cyclic electron flow (*21*), can affect fluorescence emission, and the experimental controls that would eliminate these as factors in the TAK1 antisense phenotype do not seem to be available. Although the case for TAKs being involved in some way in threonine phosphorylation of LHC II is clear, LHC II may be phosphorylated at multiple sites (*22*), and even on different amino acid side chains (*23*). Tyrosine phosphorylation, as well as threonine phosphorylation, is implicated in controlling state transitions (*24*). Although it is attractive to consider that TAK1 is the long-sought LHC II kinase, the case that this single enzyme is the key to state transitions, at least as they are currently understood, is not yet unequivocal.

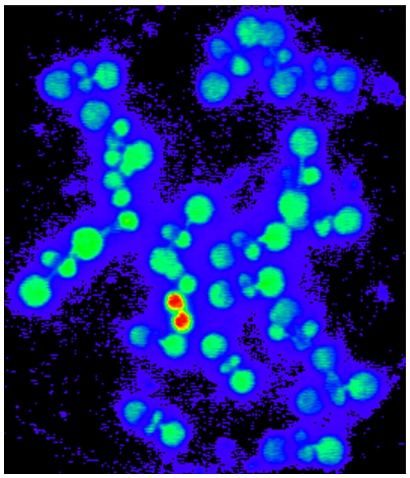

Fig. 3. Screening for state transition mutants. Leaves of the thale cress (*Arabidopsis thaliana*) were imaged by a camera that detects chlorophyll fluorescence. Fluorescence is initially high (seen as the red color), and falls after 6 min in the light (the Movie is 10 times normal speed), as LHC II becomes phosphorylated and the chloroplasts undergo the transition to state 2. A far-red is then switched on, and fluorescence falls further, to rise slightly after a further 2 min, as the phosphate group is removed from phospho-LHC II, resulting in the transition to state 1. One plant, whose two leaves are seen near the center, behaves differently, and its fluorescence stays high [see Movie 2 (http://stke.sciencemag.org/cgi/content/full/sigtrans;2002/155/pe43/DC2)].

Physical movement of LHC II within the chloroplast thylakoid membrane may contribute to its ability to redirect light between PS I and PS II. Several theories have been proposed to describe how LHC II moves within the membrane, including electrostatic repulsion and limited unstacking of the thylakoid membranes to ease diffusion within the membrane plane (*25*, *26*). The concept of molecular recognition whereby the extent to which proteins are able to interact depends on structural compatibility (*3*, *27*) might also explain why phosphorylated LHC II is disconnected from PS II and associated with PS I (*28*). This change in complementarity of binding of LHC II may take place thorugh interactions with the H subunit of PS I (*29*). The recent results from antisense mutants of the TAK family constructed in *A. thaliana* (*15*) are interpreted as arising from a different sequence of events. Rather than the only modified substrate (LHC II) somehow moving within the membrane, it is suggested that the activated protein kinase and LHC II may both diffuse away from PS II and toward PS I (*14*).

The discovery of simultaneous activation of LHC II phosphorylation and redistribution of absorbed excitation from PS II to PS I revealed the key role of plastoquinone in state transitions (*30*). A correlation between these events was established by a number of independent techniques, including chlorophyll fluorescence induction, liquid-nitrogen temperature fluorescence emission spectroscopy, and electron transport measurement (*4*). Identifying state transition-negative phenotypes by chlorophyll fluorescence imaging is a complementary and potentially very powerful approach to resolving components of the redox signal transduction pathway of state transitions (*31*). Chlorophyll fluorescence imaging has been used in screening for tagged-state transition mutants of *A. thaliana* [Fig. 3, Movie 2 (http://stke.sciencemag.org/cgi/content/full/sigtrans;2002/155/pe43/DC2)] (*31*). In LHC II-containing organisms, this approach has been applied most effectively to *Chlamydomonas* (*32*) and points to not one, but several, genes, including those for proteins of cyt b_6-f. But the qualification "in LHC-containing organisms" is important and points to the existence of essentially the same phenomenon of state transitions—a plastoquinone-mediated redox control of light energy distribution between PS II and PS I—in cyanobacteria and red algae (*3*), which have no LHC II. Instead of LHC II, cyanobacteria and red algae have a functionally analogous (but not homologous), phycobilin-based, membrane-extrinsic, mobile, light-harvesting antenna system. State-transition mutants of cyanobacteria have also been identified by fluorescence screening (*33*), but little is known from sequence homology about the key components (*34*). TAK1 is the clearest example to date of typically eukaryotic protein kinase implicated in state transitions. We think it possible that analysis of the behavior of eukaryotically derived protein kinases toward LHC II may never reveal the fundamental mechanism and origin of state transitions, but simply their adaptations to the distinctive, evolutionarily specialized, structure and function of chloroplast thylakoids.

Intriguingly, good candidates for redox sensors are the typically prokaryotic histidine sensor kinases (*35*). In plants and algae, some histidine sensor kinases contain both redox-sensor domains and photoreceptor-binding domains (*36*) and may be targeted to the chloroplast (*37*). Although phosphorylation of LHC II and state transitions can be completely explained as redox responses, without the need for photoreceptors other than chlorophyll, it is important to remember that the wavelength of light exerts profound effects on plant, algal, and bacterial development through dedicated photoreceptors, such as phytochromes (*38*). The LHC II kinase is likely to be part of an integrated network of signal transduction to which input is provided by a number of environmental signals.

What the prokaryotic cyanobacteria and their descendents, the chloroplasts, have in common is surely the essential machinery of redox sensing, redox response, and its coordination. It should be no surprise if a signal transduction system conserved from bacteria activates the LHC II kinase. A quinone-level redox signaling pathway controls transcription in *Escherichia coli* (*39*). There is detailed information on redox control of transcription in photosynthetic bacteria (*40*), including cyanobacteria (*41*), and control of reaction center gene transcription still takes place in chloroplasts (*42*). The implications of understanding these processes may stretch far beyond the fascinating domain of how plants adapt their photosynthetic machinery to changing wavelengths of light.

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