REVIEW / SYNTHÈSE

Energy balance, organellar redox status, and acclimation to environmental stress

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Abstract: In plants and algal cells, changes in light intensity can induce intrachloroplastic and retrograde regulation of gene expression in response to changes in the plastoquinone redox status. We review the evidence in support of the thesis that the chloroplast acts as a general sensor of cellular energy imbalance sensed through the plastoquinone pool. Alteration in cellular energy balance caused by chloroplast or mitochondrial metabolism can induce intracellular signalling to affect chloroplastic and nuclear gene expression in response, not only to light intensity, but to a myriad of abiotic stresses. In addition, this chloroplastic redox sensing also appears to have a broader impact, affecting long-distance systemic signalling related to plant growth and development. The organization of the respiratory electron transport chains of mitochondria and heterotrophic prokaryotes is comparable to that of chloroplast thylakoid membranes, and the redox state of the respiratory ubiquinone pool is a well-documented cellular energy sensor. Thus, modulation of electron transport component redox status by abiotic stress regulates organellar as well as nuclear gene expression. From the evidence presented, we suggest that the photosynthetic and respiratory machinery in prokaryotic and eukaryotic organisms have a dual function: primary cellular energy transformation, and global environmental sensing.

Key words: acclimation, chloroplasts, energy balance, redox status, sensing, signalling.

Résumé : Chez les cellules des plantes et des algues, les changements d'intensité de la lumière peuvent induire une régulation intrachloroplastique rétrograde des gènes, en réaction aux changements du statut redox des plastoquinones. Les auteurs revoient la preuve qui supporte la thèse à l'effet que le chloroplaste agisse comme senseur général du débalancement de l'énergie cellulaire, perçu par le pool de plastoquinones. Une altération de la balance de l'énergie cellulaire, causée par le métabolisme chloroplastique ou mitochondrial, peut induire des signaux intracellulaires qui affectent l'expression des gènes chloroplastiques et nucléiques, en réaction non seulement à l'intensité lumineuses, mais aussi à une myriade de stress abiotiques. De plus, ce senseur chloroplastique redox semble avoir un impact plus large, affectant les signalements systémiques à longue distance, reliés à la croissance et au développement de la plante. L'organisation des chaînes de transport des électrons respiratoires des mitochondries et des procaryotes hétérotrophes se compare à celle des membranes thylakoïdes des chloroplastes; on sait très bien que l'état redox du pool d'ubiquinones respiratoires agit comme senseur de l'énergie cellulaire. Ainsi, la modulation du statut des composantes redox du transport des électrons par des stress abiotiques règle l'expression des gènes des organelles et du noyau. Sur la base des preuves présentées, les auteurs suggèrent que la machinerie photosynthétique et respiratoire chez les organismes procaryotes et eucaryotes ont une double fonction; transformation énergétique cellulaire primaire, et senseur global de l'environnement.

Mots clés : acclimatation, chloroplastes, débalancement de l'énergie, statut redox, senseur, signalements.

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Introduction

Photosynthetic eukaryotes are distinguished by the presence of three genetic compartments in the cell. The chloroplasts and mitochondria contain the genes encoding a portion of their own proteome and translational machinery, with the remainder imported from a nuclear/cytoplasmic origin. This spatial genetic separation introduces an added level of complexity for the effective co-ordination of gene expression by requiring communication among compartments. It has been suggested that this spatial separation has been retained during endosymbiotic evolution as a way to ensure that gene expression can be tightly controlled by bioenergetic redox status (Allen 2003). The proposed theory for the co-localization of genes and their gene products to account for the redox regulation of gene expression (Allen 2003) is supported by the fact that energy-producing reactions need to be tightly coupled to the capacity or requirement for that energy. However, the redox active protein complexes required for electron transport in chloroplasts and mitochondria are composed of proteins of both nuclear and organellar origin (Table 1; Fig. 1), with a complete complement of subunits normally required for stable complex assembly and function. It appears that cellular energy balance is the driving force behind signals, originating in the energy-producing organelles, which coordinate nuclear and organellar gene expression.

Spatial complexity of photosynthesis

Plants, algae, and cyanobacteria have evolved a very specialized thylakoid membrane system that enables them to transform light energy into useable chemical energy in the form of ATP and NADPH for the assimilation of carbon and essential nutrients. Photosystem II (PSII) and photosystem I (PSI) are integral thylakoid membrane pigment-protein complexes each consisting of numerous distinct polypeptides (Table 1; Fig. 1). While the photosystems are the sites of primary photochemistry, the bulk of the chlorophyll and carotenoids present within the chloroplast thylakoid membrane are bound to the Lhca and Lhcb family of light-harvesting complex (LHC) polypeptides associated with PSI and PSII, respectively. The individual LHC components are known to form complex arrays within the thylakoid membrane (Jansson 1994). These arrays, also called light-harvesting antennae, greatly increase the capacity of the photosynthetic apparatus to absorb light energy. Thus it is the LHC antenna complexes that absorb the majority of the light energy for photosynthesis. The PSI and PSII reaction center polypeptides (Table 1; Fig. 1) accept light energy absorbed by the antenna complexes and use that energy for charge separation reactions (Green and Durnford 1996). As indicated in Table 1, the genetic information required to construct PSI and PSII in eukaryotes is shared between the chloroplast and the nucleus. For PSII, 16 out of 25 genes known to encode structural components are chloroplast encoded. In contrast, only 5 of 14 genes encoding PSI components originate in the chloroplast (Table 1). However, all of the main light-harvesting proteins are encoded by nuclear genes. This pattern of gene distribution is similar to that observed for mitochondrial electron transport complexes, with gene location split between the organelle and the nucleus. Important questions are thus raised regarding how gene expression in different compartments is co-ordinated to ensure stoichiometric amounts of the proteins and cofactors are available for complex assembly.

Beyond the structural components of the electron transport chain, there are numerous gene products required for their proper assembly and incorporation into the thylakoid membrane. One of the best-studied plant systems is PSI. Chloroplast-encoded assembly factors Ycf3 and Ycf4, found in photosynthetic organisms ranging from cyanobacteria to angiosperms (Rochaix 2002), are required for the stable assembly of PSI (Boudreau et al. 1997). Ycf3 is thought to interact with and promote the assembly of the nuclear- and chloroplast-encoded subunits (Naver et al. 2001). Another well-investigated example is the mitochondrial ATP synthase, which produces ATP from energy stored in the proton motive force (pmf). It requires at least five chaperone-type factors (Atp10p, Atp11p, Atp12p, Atp22p, and Fmc1p) for its assembly and integration into the mitochondrial inner membrane of the yeast *Saccharomyces cerevisiae* Hansen (Ackerman and Tzagoloff 1990; Lefebvre-Legendre et al. 2001). Two of these factors (Atp11p and Atp12p) are found in almost all eukaryotes tested to date (Pícková et al. 2005). The number of chaperone-type assembly factors required in the chloroplast and mitochondria for the assembly of membrane protein complexes (Ackerman and Tzagoloff 1990; Schulte 2001) suggests a general requirement for peptide assembly factors in the assembly of membrane complexes in chloroplasts and mitochondria.

There is another level of complexity in the assembly of the chloroplast and mitochondrial protein complexes due to the presence of a number of redox cofactors. Among the electron transport chain components there are Fe-S clusters, hemes, and quinones. In addition, carotenoid and chlorophyll molecules are not only required for photosynthesis, but also for protein-pigment complex stability (Hoober and Eggink 2001). These pigments and cofactors appear to be incorporated at the time of assembly, and are made in the chloroplast using proteins encoded in the nucleus. The protein components required for the incorporation of these cofactors in the chloroplast are only currently being identified (Merchant and Dreyfuss 1998; Lezhneva et al. 2004; Stockel and Oelmuller 2004; Shimada et al. 2005).

These examples highlight the interactive processes required to meld cytoplasmic and organellar subunits into functional protein complexes in organelle membranes. They also support the existence of mechanisms to overcome the cellular spatial separation to bring protein subunits and cofactors together in the proper location, stoichiometry, and geometry to allow for co-ordinated metabolism and the maintenance of cellular energy balance.

Temporal complexity of photosynthesis

The initial absorption of a photon and excitation energy transfer through the LHC and chlorophyll molecules of the reaction center proteins occur on a time scale of femtoseconds to picoseconds (10^{-15}–10^{-12} s), which makes this probably the fastest process in all of biology. Photosystem II reaction centers are said to be open or closed depending upon whether they are reduced and can accept an exciton of light energy to excite an electron (open), or oxidized and thus unable to excite a second electron (closed). When the

Table 1. Photosystem II and photosystem I protein subunit identity, their associated genes, and intracellular location of the genetic information in eukaryotic organisms.

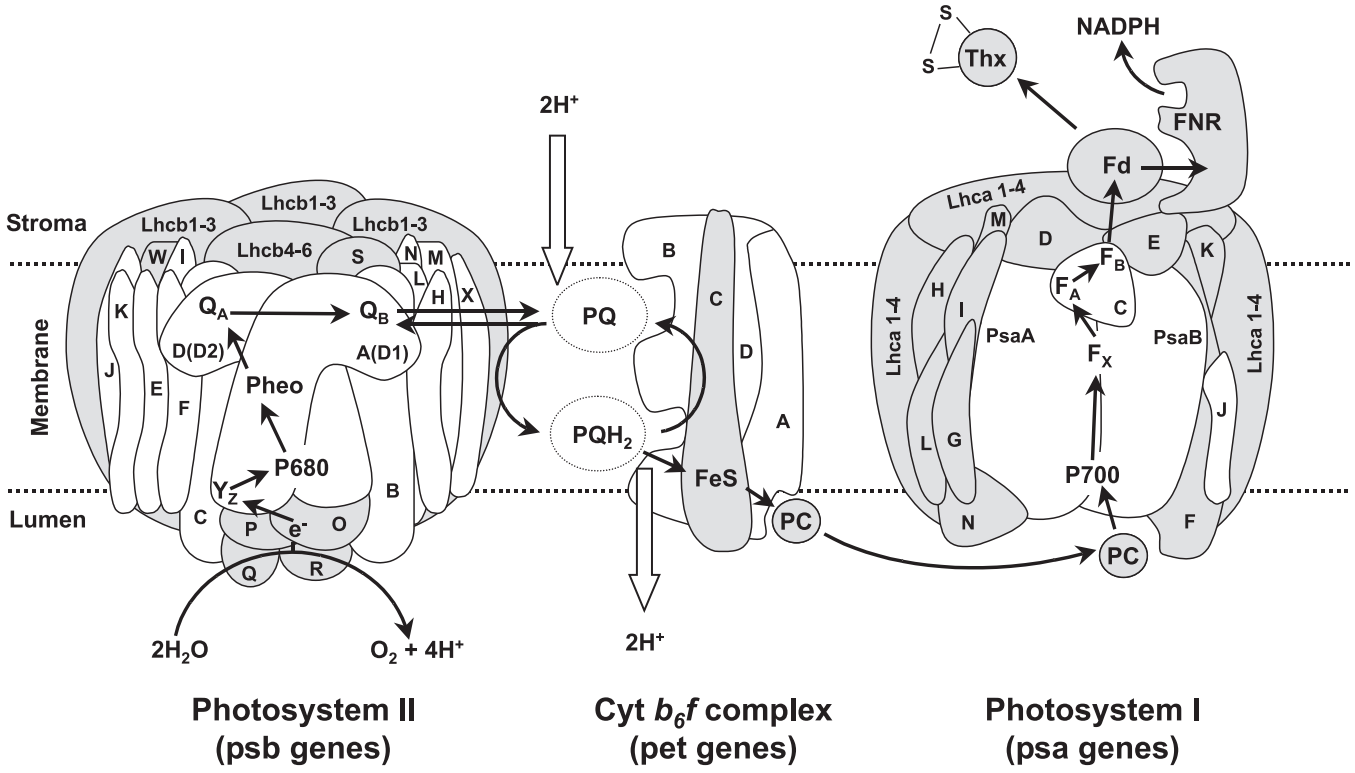
PSII			PSI		
Subunit	Gene	Gene location	Subunit	Gene	Gene location
PSII-A (D1)	*psbA*	C	PSI-A	*psaA*	C
PSII-B (D2)	*psbD*	C	PSI-B	*psaB*	C
PSII-B (CP47)	*psbB*	C	PSI-C	*psaC*	C
PSII-C (CP43)	*psbC*	C	PSI-I	*psaI*	C
PSII-E	*psbE*	C	PSI-J	*psaJ*	C
PSII-F	*psbF*	C	PSI-D	*PsaD*	N
PSII-I	*psbI*	C	PSI-E	*PsaE*	N
PSII-H	*psbH*	C	PSI-F	*PsaF*	N
PSII-J	*psbJ*	C	PSI-G	*PsaG*	N
PSII-K	*psbK*	C	PSI-H	*PsaH*	N
PSII-L	*psbL*	C	PSI-K	*PsaK*	N
PSII-M	*psbM*	C	PSI-L	*PsaL*	N
PSII-N	*psbN*	C	PSI-M	*PsaM*	N
PSII-T	*psbT*	C	PSI-N	*PsaN*	N
PSII-X	*psbX*	C			
PSII-Z	*psbZ*	C			
PSII-O	*PsbO*	N			
PSII-P	*PsbP*	N			
PSII-Q	*PsbQ*	N			
PSII-R	*PsbR*	N			
PSII-S	*PsbS*	N			
PSII-U	*PsbU*	N			
PSII-V	*PsbV*	N			
PSII-W	*PsbW*	N			
PSII-Y	*PsbY*	N			
LHCII-outer	*Lhcb1*	N	LHC-I (720)	*Lhca1*	N
LHCII-outer	*Lhcb2*	N	LHC-I (680)	*Lhca2*	N
LHCIIa-outer	*Lhcb3*	N	LHC-I (680)	*Lhca3*	N
CP29	*Lhcb4*	N	LHC-I (720)	*Lhca4*	N
CP26	*Lhcb5*	N			
CP24	*Lhcb6*	N			

Note: PSII, photosystem II; PSI, photosystem I; LHCII, chlorophyll *a* or *b* light-harvesting complex of photosystem II; LCHI, chlorophyll *a* or *b* light-harvesting complex of photosystem I; C, chloroplast; N, nucleus.

excitation energy reaches an "open" PSII reaction center [Y_Z P680 Pheo Q_A Q_B], P680 is photooxidized and an electron is transferred to the first stable quinone electron acceptor, Q_A (Fig. 1). The induction of stable charge separation [Y_Z $P680^+$ Pheo Q_A^- Q_B] occurs on a time scale of nanoseconds to microseconds (10^{-9}–10^{-6} s) and results in a closed PSII reaction center. A closed reaction center is unable to process another quantum of excitation energy. PSII reopens upon reduction of $P680^+$. To reduce $P680^+$, an electron is removed from H_2O by the Mn-oxygen evolving complex, transferred to the Y_Z tyrosine of the D1 protein, and then onto $P680^+$ to give P680 (Fig. 1). At the same time, the electron originally transferred to Pheo is transferred to Q_A and then to Q_B to yield a reopened reaction center [Y_Z P680 Pheo Q_A Q_B^-]. Thus, the pathway that an electron travels after being taken from H_2O by the oxygen evolving complex is first to the Y_Z tyrosine of the D1 protein (Fig. 1), then to the P680 special pair, followed by transfers to pheophytin (Pheo), Q_A, and Q_B (Fig. 1). Thus electrons from H_2O are used to doubly reduce Q_B producing plastoquinol (PQH_2) in a process driven by photooxidation.

To process excitation energy from the light-harvesting and core-antenna pigments on a continuous basis, photooxidation followed by the sequential reduction of the reaction centers of PSI and PSII must also occur on a continuous basis. In two successive photochemical events, electrons from Q_A^- are transferred to Q_B to give Q_B^- and then Q_B^{-2}, which attracts two H^+ ions from the stroma to form PQH_2. This mobile electron carrier diffuses away from PSII inside the thylakoid membrane and subsequently is oxidized by the cytochrome b_6f complex, regenerating PQ and releasing the two H^+ ions into the thylakoid lumen (Fig. 1). This reaction occurs on the time scale of milliseconds (10^{-3} s), and is normally the rate-limiting step in photosynthetic electron transport (Haehnel 1984). The PSI reaction center P700 also undergoes photooxidation ($P700 \rightarrow P700^+ + e^-$). $P700^+$ oxidizes the cytochrome b_6f complex via the mobile electron carrier plastocyanin (PC), converting $P700^+$ back to P700 (Fig. 1). Two successive photochemical reactions in PSI excite the electrons needed for $NADP^+$ to be reduced to NADPH via ferredoxin (Fd) and the enzyme ferredoxin NADPH reductase (Fig. 1). Thioredoxins (Thx) are key regu-

Fig. 1. A model of a eukaryotic thylakoid membrane illustrating the major polypeptides and electron transport cofactors. Subunits encoded in the nucleus and imported into the chloroplast following translation in the cytoplasm are shaded in gray. The transfer of electrons from water to $NADP^+$ in linear electron transport (Z-scheme) is illustrated with solid arrows. Open arrows illustrate the associated translocation of protons. Photosystem II: D1 (PsbA) and D2 (PsbB) are the hydrophobic subunits of the PSII reaction center binding the photochemical Chl P680, pheophytin, and the bound quinones Q_A and Q_B. The major (Lhcb1–3) and minor (Lhcb4–6) light-harvesting polypeptides are also presented attached to the reaction center periphery. The chlorophyll *a*-containing CP43 (PsbC) and CP47 (PsbD) subunits function in exciton transfer from the antenna proteins. The hydrophobic subunit S (PsbS) has a major role in photoprotection. The remaining PSII subunits (E through Z) appear to have mainly structural roles, ensuring stability and proper geometry of the reaction center, with the exception of subunits O–Q, which make up the Mn-oxygen evolving complex. Cytochrome b_6f complex: Cyt *f*, Cyt b_6, Rieske-FeS, and subunit IV (PetA, B, C, and D, respectively) are the major polypeptides of the cytochrome b_6f complex, transferring electrons from reduced plastoquinone to oxidized plastocyanin. Photosystem I: The reaction center heterodimer of PSI (PsaA/PsaB) binds the special photochemical Chl pair (P700) and 4Fe-4S cluster (F_X). The PsaC polypeptide carries two redox active 4Fe-4S clusters (F_A, F_B). The remaining subunits of PSI have a variety of functions. For example: PsaD and E are involved in ferredoxind (Fd) binding, PsaF facilitates binding of plastocyanin (PC), and subunits PsaH, L, and I are required for the proper binding of LHCI subunits (Lhca1–4) to the reaction center. Electrons end up as reducing power stored in the form of NADPH, Fd, or thioredoxin (Thx).



latory proteins in photosynthesis. They contain a pair of conserved cysteins, whose SH side chains can be oxidized to form a disulfide bond. The disulfide bond can be reduced by Fd from the S-S to the SH SH form. Reduced Thx can in turn reduce target proteins to control activation or deactivation (Jacquot et al. 2002). Thus, electron transport activity can regulate enzyme activity through the reduction of Thx and its subsequent targets. In the chloroplast, this occurs in a number of situations; however, the best-studied situation is that of Calvin cycle enzymes such as fructose-1,6-bisphosphatase and sedoheptulose-1,7-biphosphatase (Jacquot et al. 2002). Thus, photosynthetic reducing power stored as the mobile carriers NADPH, Fd, and Thx can be both consumed in a variety of biosynthetic reactions, and regulate those biosynthetic pathways. However, biochemical processes such as carbon fixation, lipid biosynthesis, and nutrient assimilation generally operate on the time scale of milliseconds to seconds. In addition to being much slower reactions, these biosynthetic pathways and the diffusion-dependent electron transfer steps that move electrons from complex to complex (Fig. 1) are temperature sensitive. Thus, potential energy imbalances can occur if the photochemical and electron transport chain reactions outpace the use of reducing power by cellular biochemical processes.

Both the oxidation of water by PSII and the oxidation or reduction of PQ release protons into the thylakoid lumen (Fig. 1). The proton gradient generated by photosynthetic electron transport is used for the chemiosmotic synthesis of ATP by the chloroplast H^+-dependent ATP synthase. This links photooxidation processes and electron transport reactions to the vectorial movement of H^+ ions and thus ATP synthesis as proposed by Peter Mitchell (1966).

In addition to properly assembling the photosynthetic machinery, the cell must be able to maintain the redox-state of the electron transport chain within a narrow range to allow the oxidation and reduction reactions to occur. This requires the co-ordination of reactions that occur on time scales spanning many orders of magnitude, while keeping the

trans-thylakoid pH gradient large enough to produce ATP, but small enough so as not to inhibit electron transport. Environmental fluctuations make this regulation more tenuous and exacerbate any imbalances.

In this review, we address how organellar redox status and energy balance is monitored by the cell to co-ordinate nuclear and organellar gene expression. Particular emphasis is given to the response of photosynthesis to environmental stress. We review data from our own laboratories, as well as those of others and provide support for the thesis that a common feature of all abiotic stresses is the establishment of an imbalance in energy budget due to the absorption of light in excess of that consumed by metabolism. We suggest that a major component of any acclimation response to abiotic stress is the re-establishment of cellular energy balance, focusing on acclimation to low-temperature and high-light stress in green algae and terrestrial plants.

Energy balance and redox poise

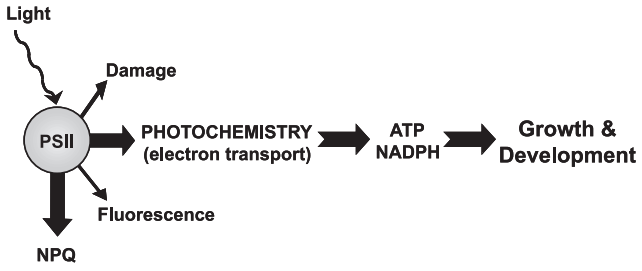
Photosynthetic organisms have a predisposition to maintain a balance between energy captured by temperature-insensitive photochemical reactions and energy used through temperature-dependent biochemical reactions (Melis 1998; Huner et al. 2002*a*). This balance between energy sources and sinks can be represented by the equation, $\sigma_{PSII} \cdot E_k = \tau^{-1}$ (Falkowski and Chen 2003), where σ_{PSII} is the effective absorption cross-section of PSII, E_k is the irradiance at which the maximum photosynthetic quantum yield balances photosynthetic capacity, and τ^{-1} is the rate at which photosynthetic electrons are consumed by terminal electron acceptors such as CO_2 or NO_3^- under light-saturated conditions. The product $\sigma_{PSII} \cdot E_k$ is, by and large, insensitive to temperature in the biologically significant range, because it reflects the photophysical processes of light absorption and energy transfer within the light-harvesting antennae, which result in charge separation. In contrast, τ^{-1} is highly temperature sensitive. The ultimate consumption of the photosynthetic electrons through metabolic sinks such as the Calvin cycle, photorespiration, and nutrient assimilation are complex enzymatic pathways that are dependent upon rates of diffusion. Thus, τ^{-1} follows the Q_{10} rule, where a 10 °C change in temperature will result in a twofold change in reaction rate. In the green alga *Chlorella vulgaris* Beij., this is reflected by changes in growth rate. Under otherwise identical conditions of light and nutrient availability, a 10 °C decrease in growth temperature suppresses the growth rate by a factor of 2.3 (Wilson and Huner 2000). Thus, an imbalance between energy absorbed versus energy used will occur whenever the rate at which the energy absorbed through PSII and the rate at which electrons are injected into photosynthetic electron transport exceeds temperature-dependent metabolic electron sink capacity, that is, whenever $\sigma_{PSII} \cdot E_k > \tau^{-1}$. Such an imbalance can be created by increasing the growth irradiance to exceed E_k at a given σ_{PSII} or by lowering the growth temperature at a constant irradiance, causing a temperature-dependent decrease in τ^{-1}. Adjustments of photosynthesis to balance the flow of energy can either occur via an increase in the rate of sink processes and (or) a decrease in the rate of energy provided through the source processes.

Figure 2 illustrates the possible fates of light energy absorbed through the photosynthetic apparatus. Under conditions of high excitation, energy balance could be reattained by either decreasing σ_{PSII} through diminished light-harvesting antenna size and (or) by dissipating energy nonphotochemically as heat (Horton et al. 1996). Alternatively, the same result could be attained by altering the turnover rate (τ^{-1}), that is, the electron sink capacity of metabolic processes that consume photosynthetic reductant. This could be achieved by changes in PSII:PSI stoichiometry to optimize the flow of electrons. Alternatively, elevating the levels or activity of Calvin cycle enzymes and the enzymes involved in cytosolic sucrose biosynthesis would increase the capacity for CO_2 assimilation relative to the capacity for photosynthetic electron transport. Photoautotrophs appear to exploit a number of mechanisms to maintain cellular energy balance in environments that exhibit daily, as well as seasonal, changes in irradiance, temperature, water availability, and nutrient status.

Chlorophyll *a* fluorescence is a property exhibited by all photosynthetic organisms because of the photochemical properties of the chlorophyll molecule. The essential role of this pigment in the structure and function of the photosynthetic apparatus allows the use of chlorophyll fluorescence as a diagnostic tool. Under normal circumstances, up to 3% of light absorbed by chlorophyll molecules is re-emitted as fluorescence. At room temperature, most of the chlorophyll *a* fluorescence emanates from PSII (Fig. 2). Both quantitative and qualitative aspects of chlorophyll *a* fluorescence induction have proven to be extremely useful in assessing the structure and function of PSII, and the overall process of photosynthesis (Krause and Weis 1991; Schreiber et al. 1994). The change in Q_A reduction as a result of $\sigma_{PSII} \cdot E_k > \tau^{-1}$ can be estimated in vivo by the pulse amplitude modulated chlorophyll *a* fluorescence quenching parameter 1-qP (Schreiber et al. 1994). An increase in 1-qP induced by various environmental conditions has been called excitation pressure (Dietz et al. 1985; Huner et al. 1998, 2002*a*). Excitation pressure thus reflects the relative reduction state of Q_A, that is $[Q_A^-] / [Q_A + Q_A^-]$ (Dietz et al. 1985; Schreiber et al. 1994), providing a nondestructive means to explore changes in energy balance as a result of changing environmental conditions. Indeed, chlorophyll *a* fluorescence has been used extensively to examine the effect of numerous environmental stresses on photosynthetic function. The effects of heavy-metal poisoning, growth temperature, drought, and nutrient limitation have all been shown experimentally to increase steady-state excitation pressure (Gray et al. 1997; Campbell et al. 1998; Wykoff et al. 1998; Golding and Johnson 2003), suggesting that all of these stressors alter the cellular energy balance through changes in τ^{-1}.

Excitation pressure may be induced by changes in several different environmental parameters. For example, increasing growth irradiance at a constant temperature would cause an overreduction of Q_A because of an increase in irradiance, and thus an increase in $\sigma_{PSII} \cdot E_k$. Assuming no changes in the capacity to utilize the absorbed energy, that is no change in τ^{-1}, energy balance would be disrupted. Theoretically, a

Fig. 2. An illustration of the possible fates of absorbed light energy through the photosynthetic apparatus. Photosystem I is not included in the diagram, because the oxidation of photosystem II (PSII) through intersystem electron transport is assumed to be the rate-limiting step in photosynthetic electron transport (Haehnel 1984). Normally, excitation energy transferred to PSII is consumed through useful photochemistry, which drives photosynthetic electron transport and the production of ATP and NADPH. When the energy absorbed by PSII exceeds that which can be used for photochemical electron transport, the excess energy can be dissipated as heat through nonphotochemical quenching (NPQ). If photochemical and nonphotochemical capacity is exceeded, PSII reaction centers are damaged, leading to photoinhibition. Typically less than 3% of the light absorbed by PSII at room temperature is lost as chlorophyll fluorescence. The amount of chlorophyll fluorescence changes in response to the redox state of Q_A, and hence reflects rates of photochemistry and nonphotochemical quenching. As a consequence, chlorophyll fluorescence is a very sensitive, noninvasive probe of PSII structure and function.



similar overreduction of Q_A could be created by maintaining the same irradiance but decreasing the growth temperature. The lower temperature would decrease the rate of the biochemical reactions that utilize the absorbed energy, decreasing τ^{-1}, with no change in $\sigma_{PSII} \cdot E_k$. Similarly, drought or the lack of specific essential nutrients would also cause a decrease in τ^{-1} because of limitations in the availability of electron acceptors such as CO_2, NO_3^-, or SO_4^{2-}. The assimilation of NO_3^- alone is estimated to account for about 25% of total energy expenditures (Bloom 1997).

Acclimation to light and low temperature

According to the discussion above, exposure of plants to either high light or low temperature may induce high excitation pressure and create an energy imbalance. However, photosynthetic organisms have evolved a number of mechanisms to ameliorate such conditions. Chronic exposure to high excitation pressure may lead to photoinhibition of photosynthesis. Photoinhibition is defined as the light-dependent decrease in photosynthetic rate that occurs when the photon flux is in excess (Melis 1999). A rapidly reversible form of photoinhibition is a consequence of an increase in thermal energy dissipation (Fig. 2; NPQ, nonphotochemical quenching), which leads to a decrease in the effective σ_{PSII} and a downregulation of PSII activity (Öquist et al. 1992). This process or any other process that protects PSII from overexcitation in the absence of protein synthesis is referred to as photoprotection (Fig. 2). If, however, the absorbed energy exceeds both the photochemical and nonphotochemical quenching capacity, the result is irreversible photoinhibition or photodamage. This type of photodamage is normally associated with the controlled degradation of the D1 and D2 proteins of PSII (Barber and Andersson 1992). While photodamage to PSII occurs even under dim light, it is greatly increased when Q_A is more reduced, i.e., when 1-qP is high. When the rate of destruction exceeds the rate of repair of the D1 protein, the result is an overall decrease in photosynthetic electron transport with a resulting decrease in σ_{PSII} (Melis 1999). Thus, photoinhibition should have very little effect on productivity, because the supply of energy is in excess of cellular demand. It follows that the acclimation response to high light and (or) low temperature should reflect the mechanisms such as photoinhibition, by which photosynthetic organisms attempt to reduce excitation pressure to reestablish a balance in energy budget.

Photoacclimation

The process whereby adjustments are made to the structure and function of the photosynthetic apparatus in response to changes in growth irradiance is called photoacclimation. The study of photoacclimation has tended to focus on light absorption ($\sigma_{PSII} \cdot E_k$). However, any process affecting cellular energy balance that can induce changes in cellular metabolism should be considered a form of photoacclimation. One mechanism of photoacclimation involves the modulation of the size and composition of the LHC of PSI and PSII (Melis 1998). Generally, there is an inverse relationship between growth irradiance and LHC size. Escoubas et al. (1995) used the green alga *Dunaliella tertiolecta* to show LHC size is modulated in response to the PQ pool redox state. When the PQ pool is reduced by exposure to light in the presence of DBMIB (Fig. 1), simulating $\sigma_{PSII} \cdot E_k > \tau^{-1}$, the transcription of the *Lhcb* genes is downregulated, decreasing the size of the LHC and producing a high-light phenotype. In contrast, the PQ pool remains oxidized in the presence of DCMU (Fig. 1), mimicking $\sigma_{PSII} \cdot E_k < \tau^{-1}$, and cells maintain a low-light phenotype (Escoubas et al. 1995). This acclimation mechanism is consistent with the notion that energy balance in response to high light may be attained through modulation of σ_{PSII}.

It is now established that the xanthophyll cycle, championed by Demmig-Adams and Adams, is an important regulator of nonphotochemical dissipation of excess light (Demmig-Adams et al. 1999; Niyogi 1999; Ort 2001). Xanthophyll-cycle-dependent antenna quenching is due to the light-dependent conversion of the light-harvesting xanthophyll violaxanthin to the energy-quenching xanthophylls antheraxanthin and zeaxanthin (Gilmore 1997; Niyogi 1999). There is now a consensus that a close relationship exists between an increase in the capacity for NPQ, the extent of the thylakoid ΔpH, and the increase in xanthophyll-cycle activity (Gilmore 1997; Demmig-Adams et al. 1999). The capacity for NPQ is also closely related to the expression of *PsbS*, a gene required for NPQ in *Arabidopsis thaliana* (Li et al. 2000, 2002; Peterson and Havir 2000). Acclimation to prolonged exposure to high light appears to result first in an increase in xanthophyll-cycle pigments and second in a persistent engagement of the xanthophyll cycle and sustained antenna quenching of excess energy through NPQ as first described by Adams et al. (1994). This aids in the maintenance of energy balance via a functional decrease in σ_{PSII}. The capacity to regulate NPQ to

maintain energy balance has a dramatic impact on the fitness of *Arabidopsis thaliana* (Arabidopsis) plants measured as net seed production under natural field conditions (Kulheim et al. 2002).

The spectral distribution of the solar radiation reaching the chloroplast can be differentially attenuated by a leaf canopy or by water in the case of algae. Because PSII and PSI have slightly different absorbance maxima, altered spectral distribution can lead to an imbalance in the absorption of light between the photosystems. The result is decreased efficiency of linear electron transport, as one population of reaction centers is excited to a greater degree than the other. In plants and algae, light absorbed preferentially by PSII relative to PSI (state 2) leads to an overreduction of the PQ pool, whereas preferential excitation of PSI relative to PSII (state 1) results in oxidation of the PQ pool. As a short-term response, the redox state of the PQ pool regulates a thylakoid protein kinase that controls the phosphorylation state of the peripheral Lhcb antenna polypeptides. In the phosphorylated state, Lhcb polypeptides associate with PSI, increasing PSI excitation and keeping the electron transport chain in a more oxidized state (Haldrup et al. 2001). Thus, the PQ pool redox state acts as a sensor of relative energy imbalances between PSI and PSII in addition to its role in regulating Lhcb abundance. While generally thought to be a short-term response, the disruption of state transitions leads to decreased growth under fluctuating photon flux densities (Bellafiore et al. 2005). Thus, rather than only maintaining electron transport rates in response to light quality, state transitions are also an important factor in maintaining productivity in response to rapidly changing light regimes.

As a poignant example of the link between chloroplast and mitochondrial metabolism, a genetic screen for state transition mutants in *C. reinhardtii* uncovered a mitochondrial protein, Moc1 (Schönfeld et al. 2004). The *Moc1* gene product is thought to regulate the transcription of mitochondrial genes, and the mutant cells do not appear to regulate levels of mitochondrial respiratory chain components in response to light, as is seen in wild-type cells (Schönfeld et al. 2004). Thus, when more light energy is available, the *moc1* mutant is unable to upregulate their respiratory capacity, causing a situation where $\sigma_{PSII} \cdot E_k > \tau^{-1}$. Thus, the Moc1 protein is required to maintain cellular energy balance. As a result, the *moc1* mutant has a more reduced PQ pool compared with wild-type cells, keeping it blocked in state 1 (Schönfeld et al. 2004). This emphasizes the role of the PQ redox state as a regulator of whole-cell energy balance. The change in τ^{-1} due to depressed mitochondrial respiration is sensed in the chloroplast and results in altered LHC state transitions.

A long-term functional equivalent to state transitions is adjustment of photosystem stoichiometry. Fujita et al. (1994) first proposed that modulation of photosystem stoichiometry is a response to changes in the redox state of the intersystem electron transport chain, which ensures equal rates of electron flow through PSI and PSII populations. Pfannschmidt et al. (1999) showed that the transcription of the chloroplast-encoded *psbA* (D1 protein) and *psaAB* (PSI reaction center core polypeptides) genes is controlled by the redox state of the PQ pool. Changes in the PQ pool redox state not only alter LHC migration as a result of LHCII phosphorylation but also affect *psaAB* and *psbA* transcript abundance (Pfannschmidt et al. 1999; Allen and Pfannschmidt 2000). Thus, the PQ pool is a redox sensor that appears to control both long- and short-term acclimation processes as an integrator of cellular metabolic status with light energy inputs.

It has been argued that the regulation of state transitions and the regulation of photosystem stoichiometry are coupled processes, because they both respond to the redox state of the PQ pool. However, Morgan-Kiss et al. (2005) showed for the first time that the capacity to regulate state transitions can be functionally uncoupled from the regulation of photosystem stoichiometry. *Chlamydomonas raudensis* UWO 241, an Antarctic psychrophile (Pocock et al. 2004), is naturally unable to undergo state transitions (Morgan-Kiss et al. 2002). However, this green alga has retained the capacity to adjust the PSII/PSI ratio through the modulation of the redox state of the PQ by light quality (Morgan-Kiss et al. 2005; Pfannschmidt 2005).

Cold acclimation

Based on the hypothesis that photosynthetic organisms respond to energy balance rather than high light or low temperature, low temperature should induce a high-light phenotype as the organism adjusts to decreased τ^{-1}. This was demonstrated to be the case during cold acclimation of the unicellular green algae *C. vulgaris* and *Dunaliella salina*, where the regulation of photosynthesis by growth at low temperature and moderate irradiance 5 °C / 150 $\mu mol \cdot m^{-2} \cdot s^{-1}$ (5/150) mimics photoacclimation at moderate temperatures and high light (27/2200) (Huner et al. 1998). Cells grown at 5/150 are indistinguishable from those grown at 27/2200 with respect to photosynthetic efficiency, photosynthetic capacity, pigmentation, Lhcb content, and sensitivity to photoinhibition. These results are explained on the basis that cultures grown at either 5/150 or 27/2200 are exposed to comparable excitation pressure measured as 1-qP (Huner et al. 1998). Similar conclusions regarding the role of excitation pressure have been reported for thermal and photoacclimation of *Laminaria saccharina* (Machalek et al. 1996) and the filamentous cyanobacterium, *Plectonema boryanum.*

(Miskiewicz et al. 2000). These results are consistent with the thesis that exposure to low temperature creates a similar imbalance in energy budget as exposure to high light, and that similar protective mechanisms are used to defend the organism.

Neither *C. vulgaris* nor *D. salina* is able to upregulate carbon metabolism and thus adjust the capacity of electron-consuming sinks during growth and development at low temperature (Savitch et al. 1996). As a consequence, these organisms exhibit a minimal capacity to adjust τ^{-1}. At the whole-cell level, this is observed for *C. vulgaris* in its inability to adjust exponential growth rates as a function of growth irradiance during growth at either 5 or 27 °C (Wilson and Huner 2000). As a result, *C. vulgaris* and *D. salina* appear to primarily adjust σ_{PSII} through a reduction in the size of the PSII light-harvesting complex coupled with an increased capacity for NPQ through the xanthophyll cycle as a mechanism for maintaining energy balance under changing growth conditions (Wilson and Huner 2000).

Cold–temperate conifers, such as lodgepole pine (*Pinus contorta* L.), and herbaceous cereals, such as winter wheat (*Triticum aestivum* L.) and winter rye (*Secale cereale* L.), are representative of some of the most cold-tolerant plants that retain their foliage during the autumn and winter (Öquist et al. 2002). The capacity to cold acclimate is an essential requirement for surviving subzero temperatures during winter. However, these two groups of plants exhibit different strategies for the utilization of light energy during growth and cold acclimation (Verhoeven et al. 1999; Savitch et al. 2002; Öquist and Huner 2003). Cold acclimation of conifers induces the cessation of primary growth in contrast with winter cereals that require continued growth and development during the cold acclimation period to attain maximum freezing tolerance (Griffith and McIntyre 1993). In the context of these different growth strategies, the requirement for photosynthetic assimilates also differs considerably. Conifers exhibit a decreased requirement for photosynthetic assimilates upon the induction of dormancy and cold acclimation, representing a decrease in τ^{-1}. In contrast, overwintering cereals maintain a high demand for photoassimilates during cold acclimation, thus keeping τ^{-1} relatively constant.

As a consequence of the decreased sink demand for photoassimilates, that is, a decrease in τ^{-1}, conifers exhibit feedback inhibition of CO_2 assimilation (Savitch et al. 2002). To maintain energy balance under these conditions, conifers decrease their capacity and efficiency to absorb light by reducing PSII and LHC protein levels. In addition, conifers increase their capacity for NPQ through the upregulation of *PsbS*, accumulation of xanthophyll-cycle pigments, and aggregation of the major light-harvesting pigment-proteins into energy-quenching complexes (Ottander et al. 1995; Savitch et al. 2002). Energetically, this allows the plant to dissipate the majority of absorbed light as heat, effectively decreasing σ_{PSII}. Conifers recover fully from this quenched state with the onset of spring (Ottander et al. 1995), suggesting the capacity to downregulate photosynthesis during cold acclimation is an important mechanism for the successful establishment of evergreen conifers in cold–temperate and subarctic climates.

In contrast, winter cereals such as wheat and rye grown and acclimated to low temperatures maintain both high efficiency and capacity for light absorption with a minimum investment in nonphotochemical quenching (Savitch et al. 2002). However, excitation pressure, measured as 1-qP, is moderate because of the fact that a high $\sigma_{PSII} \cdot E_k$ is matched by an increased capacity for CO_2 assimilation through the upregulation of transcription and translation of genes coding for Rubisco and the regulatory enzymes of cytosolic sucrose and vacuolar fructan biosynthesis (Stitt and Hurry 2002). Thus, the capacity for cold-acclimated wheat and rye to maintain energy balance upon exposure to low temperature appears to be primarily due to an enhanced capacity to utilize the absorbed light energy through an upregulation of carbon metabolism (τ^{-1}). The reprogramming of carbon metabolism to match the continued absorption of light energy at low temperature has a dual function; it not only maximizes the chemical energy and carbon pool available for the renewed growth in the spring, but the accumulation of photosynthetic end-products such as sucrose provides cryoprotectants to stabilize the cells during freezing events during the winter (Hurry et al. 1996; Stitt and Hurry 2002). Spring wheat cultivars exhibit a significantly lower capacity to maintain energy balance following cold acclimation, because they are unable to adjust carbon metabolism to as great an extent as winter cultivars (Pocock et al. 2001). For maximal low temperature survival, upregulation of photosynthesis is absolutely critical for protection from both freezing and low-temperature induced photodamage. Not surprisingly, the differential capacity to maintain cellular energy balance in winter and spring wheat cultivars is correlated with freezing tolerance measured as LT50 and field survival (Pocock et al. 2001). Gray et al. (1997) demonstrated that winter rye plants grown at cold-hardening temperatures require adequate light to reach maximal freezing resistance; growth at 5/250 decreased the LT50 by 8 °C compared with plants grown at 5/50. Thus, we would like to emphasize the critical role of photosynthesis in supplying the energy required for the acclimation processes needed to protect photoautotrophs from seemingly nonphotosynthetic stresses such as freezing.

A herbacious dicot, Arabidopsis appears to have an intermediate acclimation mechanism. In contrast with the extremes observed for conifers and winter cereals, cold-acclimated Arabidopsis exhibits an incomplete ability to adjust photosynthetic capacity relative to nonacclimated controls (Savitch et al. 2001). As a consequence, the decreased sensitivity of Arabidopsis to photoinhibition appears to be the result of an upregulation of carbon metabolism (increased τ^{-1}) combined with enhanced nonphotochemical quenching via the xanthophyll cycle to reduce σ_{PSII}. Thus, rather than only altering σ_{PSII} (green algae and conifers) or only adjusting τ^{-1} (winter cereals), Arabidopsis maintains energy balance through a combination of both processes. This probably reflects a more generalist approach that most plant species have to cold acclimation.

Alternative photoprotective quenching mechanisms

Nonphotochemical dissipation of excess absorbed light by PSII antenna quenching (NPQ) and state transitions are considered major mechanisms for protection of eukaryotic photoautrophs from photodamage. However, by comparing wild-type (WT) barley to the *chlorina F2* mutant, which lacks the major LHC polypeptides, Lhcb1 and Lhcb2, Ivanov et al. (2003) showed that significant photoprotection can occur independently of xanthophyll cycle-mediated antenna quenching and independent of state transitions. They addressed this apparent enigma by using thermoluminescence, a very sensitive spectroscopic technique to probe PSII charge recombination events in vivo in WT and the *F2* mutant of barley as well as the model green alga, *C. reinhardtii*. Ivanov et al. (2003) showed that the propensity for PSII charge recombination is reversible and under dynamic, redox regulation by the environment through modulation of excitation pressure. Thermoluminescence of the nonphotochemical quenching mutant *npq4–1* of Arabidopsis indicated that the PSII subunit PsbS, while integral to antenna quenching, is not involved in the regulation of energy quenching through PSII charge recombina-

tion. It was concluded that PSII reaction center quenching complements photoprotective NPQ in a time-nested fashion during long-term acclimation of oxygenic photoautotrophs as diverse as cyanobacteria, green algae, herbaceous plants, and conifers (Ivanov et al. 2003, 2006).

How does growth environment affect the propensity for PSII charge recombination? Steady-state growth at either high light or low temperature results in comparable excitation pressure, because both growth regimes induce a comparable condition whereby $I > E_k$, and as a consequence, $\sigma_{PSII} \cdot I > \tau^{-1}$ because of metabolic limitations. This limitation feeds back through the intersystem electron transport chain to cause an increased reduction of the PQ pool (PQH_2:PQ), which in turn, causes a decrease in the free energy gap between Q_A and Q_B, such that the equilibrium between Q_A:Q_A^- and Q_B:Q_B^- favours the accumulation of Q_A^-. This results in a 30%–40% decrease in total thermoluminescence yield, which is consistent with a dark decay pathway for charge recombination and photoprotection through nonradiative PSII charge recombination. As reported for Arabidopsi*s*, this PSII charge recombination pathway may account for up to 65% of the total capacity for NPQ (Sane et al. 2003).

This mechanism for photoprotection, which occurs independently of either antenna quenching via PsbS and the xanthophyll cycle or state transitions, is evolutionarily conserved in all oxygenic photoautotrophs from cyanobacteria to pine trees. In contrast with the rapidly reversible antenna quenching occurring on a time scale of seconds to minutes (Demmig-Adams et al. 1999; Horton et al. 1999; Dall'Osto et al. 2005; Kopecky et al. 2005; Niyogi et al. 2005), the capacity for PSII reaction center quenching is retained during steady state growth at high excitation pressure and develops more slowly on a time scale of hours in *C. reinhardtii* and *Synechococcus* sp. PCC 7942 (Sane et al. 2002) and on a time scale of weeks during cold acclimation of Arabidopsis (Sane et al. 2003) and pine under natural, overwintering field conditions (Ivanov et al. 2002). This proposed mechanism supports recent proposals for alternative quenching mechanisms that invoke a role for PSII reaction centers (Bukhov et al. 2001; Sane et al. 2002, 2003; Finazzi et al. 2004; Matsubara and Chow 2004) and support the thesis that antenna quenching and reaction center quenching are complementary energy dissipative mechanisms for photoprotection of PSII through feedback de-excitation (Krause and Weis 1991). Since PSII reaction center quenching may be induced on a time scale that is several orders of magnitude slower than antenna quenching under the environmental conditions tested, Ivanov et al. (2003, 2006) suggest that these complementary quenching processes should be viewed in the context of a time-nested response (Falkowski and Chen 2003) to an imbalance in energy budget during photoacclimation under steady-state growth conditions, much in the way state transitions and photosynthetic stoichiometry attempt to correct short-term and long-term energy imbalances, respectively.

Mechanisms of energy sensing

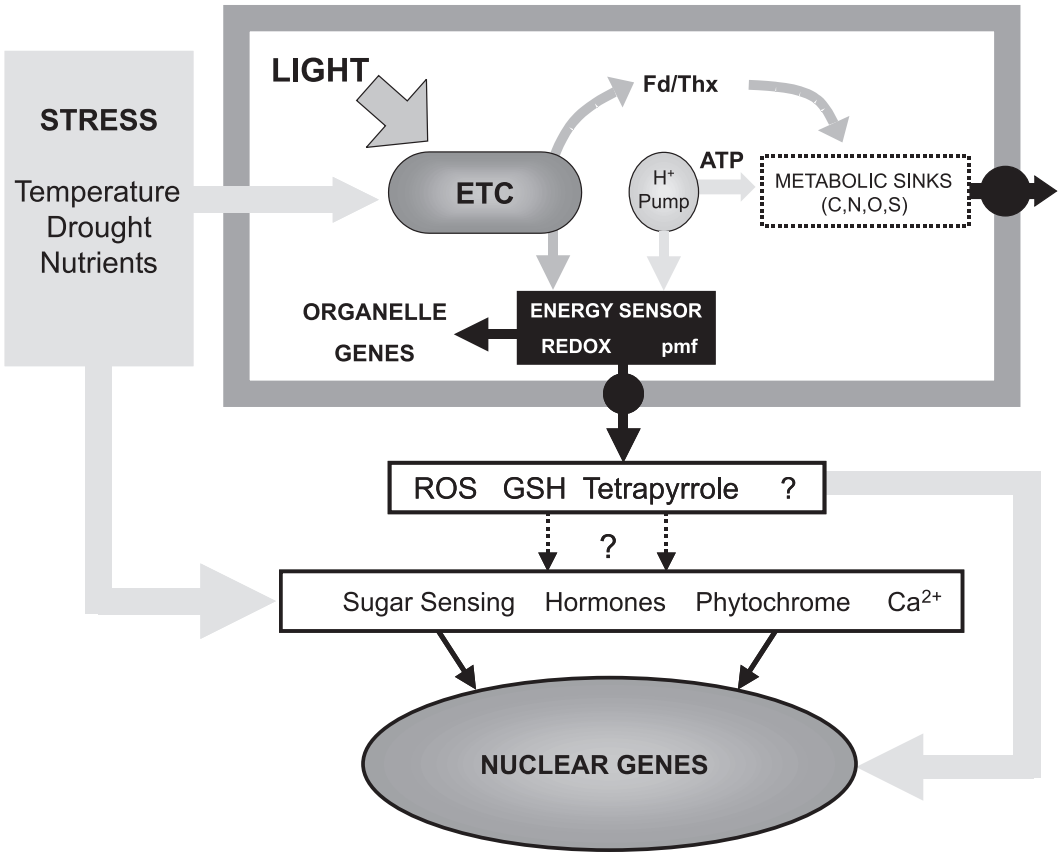
How do cells sense changes in their energy status? Ferguson et al. (1987) and Taylor and Zhulin (1999) have suggested that cellular energy sensing in prokaryotes is linked to the redox status of the energy transducing electron transport chains. The precise mechanism for energy sensing remains a mystery. A number of mechanisms for sensing and signalling environmental redox perturbations have been suggested: dithiol and (or) glutathione levels (Gomez et al. 2004); reactive oxygen species (ROS) (Laloi et al. 2004); ATP or NADPH ratios (Melis et al. 1985; Noctor and Foyer 2000); and carbohydrate levels (Rolland and Sheen 2005). Other mechanisms that may play a role in communicating the metabolic status of the chloroplast to the cytosol and nucleus are Snf1-related kinase (Thelander et al. 2004), isoprenoid biosynthesis (Laule et al. 2003), and chlorophyll biosynthesis (Strand 2004). Thus, a myriad of possibilities exist. The great difficulty for elucidation of the signalling processes is very likely due to the interconnectedness of some or all of these possibilities, as well as complex hormone and photoreceptor signalling processes.

In chloroplasts and mitochondria, it has been proposed that energy sensors may monitor changes in the redox status of specific electron carriers. In particular, the redox status of the quinone pool and (or) one of the components of the pmf ($\Delta\psi$ or ΔpH; Fig. 3) (Ferguson et al. 1987; Allen et al. 1995; Huner et al. 1998; Taylor and Zhulin 1999). In chloroplasts, the key component of such a system is thought to be the cytochrome b_6f complex (Allen 2004). An integral component of the electron transport chain, accepting electrons from the PQ pool and potentially Fd via cyclic electron transport, the cytochrome b_6f complex appears perfectly situated to act as a sensor of electron transport imbalance. Recent structural studies suggest that redox-dependent changes in the secondary structure of cytochrome b_6f components may be involved in this signalling process (Allen 2004). However, strong genetic and biochemical proof is still lacking.

The key role of this type of signal sensing at the PQ redox level is assessing the balance of electron transport. Modulation of either source or sink capacity will modulate the relative redox status of the electron transport chain, which will also affect the pmf, as these two forces are linked by the proton pumping of electron transport reactions. The sensor(s) would then transform either the redox status or the pmf into a biochemical signal that regulates intracellular gene expression (Fig. 3).

The PQ pool redox state appears to regulate both short-term (state transitions and LHC phosphorylation) and long-term (transcriptional regulation of electron transport and light-harvesting components) photoacclimation responses. However, the PQ pool is not the sole regulator of acclimation to light and temperature. As demonstrated by NDong et al. (2001), individual stresses, even when present in combination, can influence gene expression independently. Their study of gene expression in response to 1-qP revealed that some acclimation genes respond to cold only and others respond to light only. A study of high-light inducible genes in *C. reinhardtii* revealed a partial interaction between light and CO_2 levels (Im and Grossman 2002). One could expect combined regulation in this situation, as increased CO_2 would increase τ^{-1}. By treating the cells with DCMU and DBMIB, it was demonstrated that CO_2 affects gene expression in a redox-sensitive manner, while high light has an independent effect (Im and Grossman 2002). Thus, while both

Fig. 3. An illustration reflecting the convergence of retrograde and redox-energy sensing and (or) signalling pathways with those traditionally associated with abiotic stresses. In photosynthetic organisms, light is the ultimate source of energy. It is converted to redox potential energy through electron transport reactions. Various environmental factors will modulate the redox state of the plastoquinone pool because of limitations in diffusion or electron acceptor availability. Thus, the energy balance may be sensed by changes in either the redox status of the electron transport chain components or the proton motive force (pmf) and signals transduced through an unknown signal transduction pathway to regulate organellar as well as nuclear gene transcription. Ultimately acclimation to the environment will be dependent upon the chloroplast sensor(*s*). The idea of cross-talk between the chloroplast and other sensors suggests the same abiotic stresses may also act through independent sensors and signal transduction pathways. The net response of an organism to any abiotic stress represents an integration of these sensing and (or) signalling pathways. Abbreviations: Fd, ferredoxin; Thx, thioredoxin; ETC, electron transport chain; ROS, reactive oxygen species; GSH, glutathione; Phy, phytochrome.



ends of the signalling pathway (photosynthesis-related gene expression and PQ redox status) have been well studied, the steps in between remain to be elucidated.

Signal transduction and "cross-talk"

In a changing environment, all photosynthetic organisms must sense and integrate a myriad of oscillating external signals such as light intensity, light quality, temperature, water availability, and nutrient status. In nature, modulation of any one of these external conditions rarely, if ever, occurs independently of the others. Thus, acclimation to an ever-changing environment must be a stochastic process, whereby a cell or an organism integrates signals from multiple sensors. Subsequently, this information is processed by a multitude of signal transduction pathways that elicit the required molecular and physiological responses necessary for the maintenance of cellular homeostasis (Fig. 3). In our opinion, energy sensing represents an example of a global sensing mechanism. Because cellular energy metabolism integrates the primary redox chemistry of both photosynthesis and respiration connected through C and N metabolism (Padmasree and Raghavendra 1999; Noctor and Foyer 2000; Wilson et al. 2003*a*), chloroplasts and mitochondria may be considered to exhibit dual functions: not only are they essential cellular energy transformers, but they also represent environmental redox sensors through their capacity to detect cellular energy imbalances. There is abundant evidence in the literature to indicate that the redox status of the electron transport chain is involved in regulating the expression of photosynthetic genes in prokaryotes (Bauer et al. 1999; Oh and Kaplan 2001). In eukaryotes, a consensus is emerging that expression of both chloroplastic and mitochondrial genes, as well as retrograde regulation of nuclear genes encoding photosynthetic and nonphotosynthetic proteins, can occur via the modulation of the redox status of the membrane-bound quinone pools associated with photosynthetic and respiratory electron transport (Allen et al. 1995; Escoubas et al. 1995; Poyton 1999; Blackstone 2000; Huner et al. 2002*b*; Pfannschmidt 2003).

As discussed above, intracellular energy imbalance ($\sigma_{PSII} \cdot E_k \neq \tau^{-1}$) can be created by changing either tem-

perature or light intensity. In addition, there is evidence that photosynthetic acclimation to nutrient stress such as iron or phosphate deficiency, at least in part, may also represent adjustment to excitation pressure and the redox status of the PQ pool (Terry 1983; Wykoff et al. 1998; Moseley et al. 2002). How is the redox signal transduced intracellularly? Escoubas et al. (1995) proposed a working model for an intracellular signalling pathway connecting the chloroplastic PQ redox state (as the sensor) to the regulation of nuclear-encoded *Lhc* genes. It is suggested that the reduction of the PQ pool induces the phosphorylation of a chloroplastic phosphoprotein (CPP) that is exported from the chloroplast to the cytosol (Chen et al. 2004). Subsequently, the phosphorylated CPP was proposed to activate a cytosolic protein kinase that phosphorylates an *Lhc* gene transcription factor. Although evidence for a G-like motif in the *Lhc* promoter region(s), which appears to bind protein factors present under high light but not under low light, has been presented (Escoubas et al. 1995; Chen et al. 2004), genetic evidence for a chloroplastic phosphoprotein involved in the regulation of a cytosolic kinase is still lacking.

The identification of plastid factors involved in retrograde regulation between the chloroplast and the nucleus continues to be an active area of research in plant biology (for a recent review, see Beck 2005). Recently, evidence for the involvement of several candidate plastid factors has been reported. Results with the *Arabidopsis* mutant *genomes uncoupled 5* (*gun5*) indicate that perturbations of tetrapyrole biosynthesis generate at least two plastid-nuclear signalling pathways, one of which involves the H-subunit of Mg-chelatase, an enzyme involved in Chl biosynthesis (Mochizuki et al. 2001). Several reports indicate that Mg-protoporphyrin IX may act as a signalling molecule in the repression of *Lhc* transcription in eukaryotes (Kropat et al. 1997). These results are consistent with the role of heme (Fe-protoporphyrin IX) in oxygen sensing and signalling in animal systems (Poyton 1999). In *C. vulgaris*, Mg-protoporphyrin intermediate accumulation occurs in response to low temperature but not high light (Wilson et al. 2003*b*). Thus, these intermediates do not appear to play a significant role in the regulation of Lhcb polypeptide accumulation by excitation pressure in *C. vulgaris* (Wilson et al. 2003*b*), as has been suggested in higher plants (Strand 2004). Furthermore, while Flu and Flp proteins have been suggested to regulate chlorophyll biosynthesis, to ensure proper levels of intermediates (Meskauskiene et al. 2001; Falciatore et al. 2005), how these proteins themselves are regulated remains unclear.

Another class of signal molecules that has been presented as a possible link between excitation pressure and nuclear gene expression are ROS (Karpinski et al. 1999; Dutilleul et al. 2003; Apel and Hirt 2004). In *C. vulgaris*, the accumulation of ROS in vivo is negatively correlated with Lhcb abundance (Wilson et al. 2003*b*). Interestingly, absolute ROS levels are positively correlated with excitation pressure and exhibit a distinctly temperature-dependent component (Wilson et al. 2003*b*). The proposed role of ROS as a redox signal of excitation pressure is consistent with a proposed mechanism of ROS generation, where electrons from reduced electron transport chain components are transferred directly to O_2, thus linking the redox state of electron transport components to signalling molecule production (Apel and Hirt 2004).

There is a consensus in the literature that Ca^{2+} acts as an important second messenger in signal transduction pathways associated with low temperature, heat stress, drought, hypoxia, and phytochrome-regulated plant development (Snedden and Fromm 1998). Research using Arabidopsis indicates that mitogen-activated protein kinase (MAPK) cascades are involved in the signal transduction pathways associated with responses to abiotic stresses (Ichimura et al. 2000). Recently, Kim et al. (2002) concluded that phytochrome plays an important role in the low temperature-induced gene expression mediated by the dehydration responsive element (DRE) in Arabidopsis. Because excitation pressure is modulated by a variety of abiotic stresses, signal transduction pathways likely converge to regulate gene expression (Fig. 3). Most abiotic stress experiments are performed in the light, making it critical that the experimental design includes proper controls to separate the confounding influence of excitation pressure from the specific effects of individual cues on gene expression. An experimental design that successfully differentiates between the effects of excitation pressure, low temperature, and light intensity has been described for *Secale cereale* L. (NDong et al. 2001). In their experimental design, NDong et al. (2001) grew plants under five different conditions: three at 20 °C and two at 5 °C, representing three levels of 1-qP. This was essential for the identification of light, temperature, and excitation pressure regulated genes from a cDNA subtraction library. Their results highlight the requirement for proper experimental design for the interpretation of studies designed to assess transcriptional responses to abiotic stress.

The chloroplast redox sensor(s) not only regulates intracellular processes, it also appears to induce systemic, long-distance signalling. Through an elegant experimental design, Karpinski et al. (1999) demonstrated that primary Arabidopsis leaves exposed to high excitation pressure has altered cytosolic ascorbate peroxidase (Apx) gene expression, making the leaves more tolerant to high light exposure. Most importantly, it was demonstrated that the acclimation process also occurs in secondary leaves that have never been exposed to high light (Karpinski et al. 1999). It was suggested that H_2O_2 may act in long-distance signalling. This has since been supported by examination of H_2O_2 trapping compounds at the cellular and whole-leaf level (Fryer et al. 2002). Thus, excitation pressure-induced signals can be transmitted throughout the leaf.

The compact growth habit of cereals typically associated with development at low temperature is actually a consequence of growth at high excitation pressure (Huner et al. 1998). Thus, a systemic signal must be transported from the leaf to the meristematic regions in the crown to affect tillering and leaf development. Reactive oxygen species such as H_2O_2 may also function in this adaptive process, but it has been proposed that sucrose also possesses the characteristics necessary for a signalling molecule. Sucrose levels could effectively communicate information between source and sink tissues regarding metabolic status. Thus, it appears that an excitation-pressure-related signal, independently or in com-

bination with plant growth regulators, may act as a morphogen (Pollock and Farrar 1996).

Chloroplast redox sensing and (or) signalling appears to interact with cytosolic sugar sensing and (or) signalling pathways to regulate *Lhcb* expression in Arabidopsis (Oswald et al. 2001). As mentioned, many other signalling processes are known to affect *Lhc* expression, for example phytochrome, cytokinins, and brassinosteroids. This highlights one of the primary difficulties in teasing out the cellular processes that connect PQ redox state to the maintenance of energy balance. Despite results that clearly indicate the existence of significant crosstalk between redox sensing and (or) signalling and other sensing and (or) signalling pathways involved in the regulation of Lhcb polypeptide accumulation, the organellar redox signals remain to be dissected from the complex background of plant metabolism and development (Fig. 3).

Clearly, the energy sensing and (or) signalling pathways initiated by the chloroplast (Fig. 3) have global implications with respect to cell and whole-plant energy balance. Thus, we suggest that the chloroplast has a dual function: not only primary energy production, but also global environmental sensor. Modulation of the redox status of the plastoquinone pool affects intracellular acclimation as well as plant growth and development through long-distance systemic signalling, with chloroplast signals regulating both plant metabolism and morphological characteristics throughout the organism. It should not be surprising that the chloroplast plays such a central role, because plants ultimately depend upon light as an energy and information source. Indeed, this is consistent with the notion of a "grand design" for photosynthesis initially proposed by Daniel Arnon (1982).

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