Chapter 12

Diversity in Photoprotection and Energy Balancing in Terrestrial and Aquatic Phototrophs

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I. Introduction

Photosynthesis likely evolved because it gave living systems the survival advantages of solar energy, obviating the need for localized energy sources. At the same time, nature places very specific constraints on photosynthesis because the light reactions involve highly energetic intermediates that can generate highly toxic side products, especially reactive oxygen species (ROS), that can kill the organisms it powers. Thus, the energy input into photosynthesis must be tightly regulated by photoprotective nonphotochemical quenching (NPQ) and control of electron transfer, to balance the need for efficient energy conversion with the avoidance of photodamage (Gust et al. 2008; Aro et al. 1993; Raven 2011; Sonoike 2010; Long et al. 1994; Kanazawa et al. 2017; Davis et al. 2016, 2017; Alboresi et al. 2018). A related constraint on photosynthesis is the need to balance energy storage into ATP and NADPH to *precisely* meet biochemical

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A. W. D. Larkum et al. (eds.), *Photosynthesis in Algae: Biochemical and Physiological Mechanisms*, Advances in Photosynthesis and Respiration 45, https://doi.org/10.1007/978-3-030-33397-3_12

demands. If this balancing does not occur, the system will fail, leading to metabolic congestion and photodamage (Kramer and Evans 2011). It is also becoming clear that energy balancing mechanisms must not only be robust, but respond to rapid and unpredictable fluctuations in environmental conditions that are encountered in the field, and that lagging responses can lead to severe photodamage (e.g. when light is suddenly increased) (Davis et al. 2016) or substantial losses of photosynthetic efficiency (e.g. when light is suddenly decreased) (Kromdijk et al. 2016). This chapter discusses the impact of recent research that reveals unexpected connections between photoprotection and energy balancing, focusing on the impact of photoprotection on energy balance and *vice versa*.

II. Energy Storage and Regulation in Oxygenic Photosynthesis

In its simplest formulation, oxygenic photosynthesis consists of the light-driven transfer of electrons from water to CO_2, to form O_2 and reduced forms of carbon. This reaction could also be performed in two discrete steps by human-made "artificial leaves," using a set of appropriate catalysts and photovoltaic cells to generate electrical potential, to directly oxidize water at one electrode, and reduce CO_2 at another. By contrast, natural photosynthesis is much more complex, mainly because it evolved within pre-existing living systems. For example, natural photosynthesis systems are quite sensitive to ROS so it is critical for photosynthesis to minimize the production of high energy excited states or electron transfer intermediates that can interact with O_2 (Foyer and Shigeoka 2011; Rutherford et al. 2012) As will be discussed below, minimizing ROS-producing side reactions involves downregulation of energy capture that necessarily results in loss of energy, leading to tradeoffs between energy efficiency and the avoidance of ROS. These tradeoffs are compounded by the fact that natural photosynthesis evolved to "plug into" ancient biochemical pathways that require energy in bio-compatible forms, including redox energy stored in the NAD(P)H/O_2 couple, and phosphorylation potential stored in the ATP/ADP + P_i couple (Gust et al. 2008). As discussed below, the ratios of the production of ATP and NADPH must be balanced to match metabolic needs and thus avoid metabolic congestion (Kramer and Evans 2011).

We first describe the well-known, and highly conserved core components of the light reactions, and then discuss why a photosynthetic system based solely on these components will fail under real world conditions. We then introduce a series of additional components that have evolved that act to "tune" the activities of the core components, making them biocompatible. The text will focus mainly on how the interactions of electron, proton and counterion transport play pivotal roles in tuning both energy capture and energy balancing, and how diverse tuning mechanisms impose different constrains on these interactions.

The core reactions of oxygenic photosynthesis involve a processes called "linear electron flow" (LEF), in which light is used to extract electrons from water and transfer them to $NADP^+$ while generating ATP from ADP and P_i (reviewed in e.g. Kramer et al. 2004a; Ort and Yocum 1996). Light is initially captured by an array of pigments in light harvesting protein complexes, and delivered to a special subset of chlorophyll molecules in photosystem I (PSI) and photosystem II (PSII). The absorption of a photon of visible light result in the photochemical excitation of chlorophylls in PSII, producing a state termed P_{680}^*, which allows for the formation of a charge separated state, with an electron being transferred through a series of electron carriers within PSII to a plastoquinone (PQ) molecule, Q_B, and an electron hole remaining on P_{680}. The oxidized P_{680}^+ is a very strong electron oxidant (~1.25 eV (Grabolle and Dau 2005)), that extracts electrons from a redox active tyrosine, which in

turn allows oxidation of the manganese cluster of the oxygen evolving complex, releasing one molecule of O_2 and 4 H^+ into the lumen for each 4 turnovers of PSII. Upon successive excitation of PSII, electrons on Q_B are transferred through a cascade of exergonic electron transfer reactions through a mobile pool of PQ, the cytochrome b_6f complex, plastocyanin (PC), and to the primary electron donor in PSI, P_{700}^+ (orange arrows in Fig. 12.1). An additional photon is required to excite an electron on P_{700} to reach the P_{700}^* state, which initiates a second cascade of energetically downhill electron transfer reactions in PSI, then to ferredoxin (Fd) and finally to NADPH.

Through these core processes, energy is initially stored in two forms. Extracting electrons from water and transferring them to $NADP^+$, energy is stored in the two redox half reactions $4H^+ + O_2$ / H_2O and $NADP^+ + H^+$/NADPH. In addition, the transfer of electrons results in the storage of an electrochemical gradient of protons across the thylakoid membrane, termed the proton motive force (*pmf*). The vectorial electrogenic transfer (from the lumenal to the stromal face of the thylakoid membrane, within PSII, the cytochrome b_6f complex—via the Q-cycle mechanism (reviewed in Cape et al. 2006) and PSI) results in the formation of the electric field component of *pmf*. The *pmf* represents the driving force for moving protons across the thylakoid membrane, which can have two distinct energetic components. The ΔpH component, sometimes called the proton gradient, is the free energy stored in the difference of free proton concentrations between the lumen and stroma. Because protons are charged, they will also tend to move

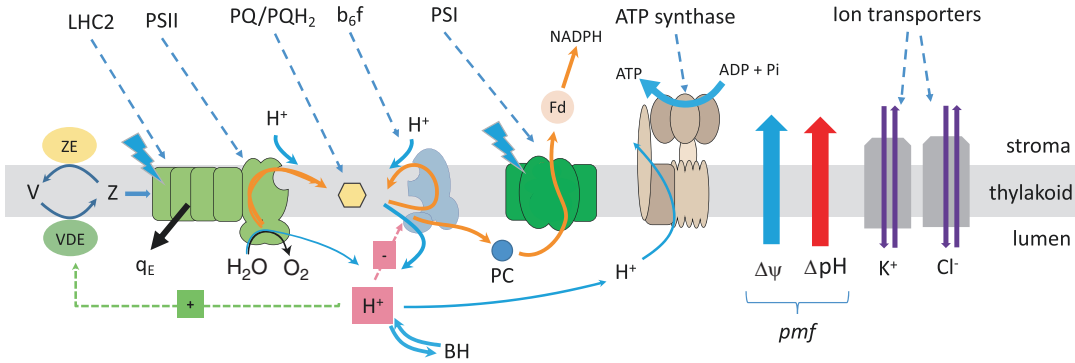

Fig. 12.1. **Basic Z-scheme model for the electronic and protonic circuits of the light reactions of photosynthesis, and the pmf paradigm for regulation of the light reactions.** *Scheme of linear electron flow (LEF) in oxygenic photosynthesis, in which light energy is captured by light harvesting complexes associated with photosystem II (PSII light green) and photosystem I (PSI, dark green) initiate electron flow (orange arrows) from PSII, through the cytochrome* ***b6f*** *complex (dark grey), plastocyanin (PC, turquois), to PSI and ferredoxin (brown) and finally to NADPH.* LEF is coupled to proton flow (blue arrows) at PSII and the cytochrome b_6f complex, storing energy in the thylakoid proton motive force (*pmf*). Transfer of electrons from the lumenal (pink) to the stromal side of the thylakoid (grey) forms a transmembrane electric field (Δψ, blue arrow), while proton uptake from the stroma and deposition in the lumen lead to the formation of a transthylakoid pH gradient (ΔpH, red arrow), which together drive the synthesis of ATP from ADP + P_i at the thylakoid ATP synthase, storing energy in ΔG_{ATP}. The orange and blue lines describe the pathways of electron and proton fluxes, leading to the reduction of NADPH and the synthesis of ATP. The acidification of the lumen (indicated by the H^+ in the red box) activates violaxanthin deepoxidase (VDE) which converts violaxanthin (V) to zeaxanthin (Z) and protonates the PsbS protein, which triggers the photoprotective dissipation of light energy by the q_E (black arrow). Lumen pH also regulates electron flow (red box with '-') to PSI by slowing the rate of PQH_2 oxidation at the cytochrome b_6f complex. Abbreviations: ATP synthase, chloroplast ATP synthase; b_6f, cytochrome b_6f complex; LHC2, light harvesting complexes associated with PSII; PSI/PSII, photosystem I and photosystem II; PQ/PQH_2, plastoquinone, plastoquinol; *pmf*, proton motive force, ZE: zeaxanthin epoxidase

down an electric field (from more positively to more negatively charged faces of the membrane), and thus a second term, $\Delta\psi$, represents the differences in charge between the lumenal and stromal faces. The ATP synthase is driven by the sum of $\Delta\psi$ and ΔpH, and both forms are energetically equivalent (Fischer and Graber 1999; Hangarter and Good 1982), so that

$$pmf = \Delta\psi\left(1-s\right) - 2.3RT / F\,\Delta pH\left(1-s\right)a_b$$

where l and s are the lumenal and stromal facing sides of the thylakoid membrane, R is the universal gas constant, F is Faraday's constant, and T is the absolute temperature in Kelvin. Note that the RT/F term simply converts the energies of two components of *pmf* into the same units.

The $\Delta\psi$ component is sometimes called the "electric field," though a field is defined with units of V/m or N/C, whereas $\Delta\psi$ is typically expressed simply as V. If we assume that the thylakoid membrane is approximately 5 nm thick (Daum et al. 2010), then a typical $\Delta\psi$ of 60 mV would be approximately 120,000 V/cm. This calculation demonstrates that the electric fields experienced by the photosynthetic machinery are not trivial. Indeed, the fields are strong enough to shift the redox potentials of electron transfer components (Drachev et al. 1989; van Gorkom 1996) as well as the absorption spectra (colors) of pigments (via Stark shift) of light harvesting complexes embedded in the thylakoid membranes (Kakitani et al. 1982; Witt and Zickler 1974; Bailleul et al. 2010).

III. The pmf Paradigm for Regulation of the Photosynthetic Light Reactions

A series of mechanisms for photoprotection have evolved to avoid over-excitation of reaction centers that can lead to accumulation of electrons on highly reducing carriers and production of ROS, when light input exceeds the capacity of system to process it (e.g. Hideg et al. 2006; Asada 2006; Nishiyama et al. 2006; Tyystjärvi and Aro 1996; Aro et al. 1993). We will focus here on what we call the "*pmf* paradigm" of feedback regulation, in which the *pmf* plays central role in both energy storage and feedback regulation, and has been extensively studied in chloroplasts of higher plants and some algae. A major photoprotective mechanism in chloroplasts which acts to dissipate light energy from antenna complexes is through "non-photochemical quenching" (NPQ) (Anderson and Barber, 1996; Aro et al. 1993; Baker and Bowyer, 1994; Demmig-Adams and Adams, 1996; Müller et al. 2001). There are several NPQ mechanisms, all of which can contribute to photoprotection, and are triggered by diverse regulatory mechanisms, as discussed in more detail in Chap. 10. We will first describe components of this paradigm, focusing on what has been established for plant chloroplasts, in particular the NPQ mechanism termed q_E (for 'energy dependent' quenching), which is triggered by acidification of the lumen imposed by the ΔpH component of *pmf*. Activation of q_E in higher plant chloroplasts involves at least two reactions: the conversion of violaxanthin (V) to antheraxanthin and zeaxanthin (Z) (see Eskling et al. 2001), which is controlled by the lumen pH-sensitive violaxanthin deepoxidase (VDE), and the stromal redox-sensitive zeaxanthin epoxidase, which catalyzes the reverse reaction; and the protonation of the antenna protein PsbS (Li et al. 2002; Niyogi et al. 2004). Various models have been proposed for both the biophysical mechanism of q_E and how the two reactions interact (Kiss et al. 2008; Crouchman et al. 2006; Zaks et al. 2012; Li et al. 2004). For the purposes of the current discussion, we can assume that Z accumulation and PsbS protonation are both required for full q_E activity, and both are controlled by the lumen pH, which is consistent with phenomenological behavior of q_E in plants (Takizawa et al.

2007). The ΔpH component of *pmf* also down-regulates electron flow by slowing PQH_2 oxidation by the cytochrome b_6f complex (in a process termed 'photosynthetic control'), preventing accumulation of electrons on highly reducing components of PSI, and subsequent PSI photodamage (reviewed in Takizawa et al. 2007).

IV. The Need to Coordinate q_E and Photosynthetic Control

Photosynthetic control in the absence of NPQ can lead to buildup of electrons in the PQ pool, leading to accumulation of reduced Q_A, which is expected to accentuate photodamage to PSII (Vass et al. 1992). To prevent this situation, q_E should be initiated prior to the onset of photosynthetic control, by tuning the lumen pH-dependencies of VDE, PsbS and PQH_2 turnover at the b_6f complex. Indeed, detailed *in vivo* spectroscopic characterization (Takizawa et al. 2007) showed that, as the lumen pH decreases, PsbS and VDE are partially activated prior to onset of photosynthetic control at the b_6f complex (Takizawa et al. 2007). By contrast, the *pgr1* mutant in *Arabidopsis thaliana*, which contains a point mutation in the Rieske FeS protein of the b_6f complex conferring altered pH-dependence of PQH_2 oxidation so that photosynthetic control is induced at a more moderate (higher) lumen pH than q_E can be activated (Jahns et al. 2002), leads to the buildup of reduced Q_A^- and accelerated photoinhibition of PSII (Okegawa et al. 2005).

V. The Critical Need to Balance the Chloroplast Energy Budget

The pool sizes of ATP and NADPH are small relative to the high fluxes of energy from the light reactions. Thus, any imbalance in the production and consumption of ATP or NADPH can rapidly lead to "metabolic congestion" (indigestion), depletion or buildup of metabolic intermediates, followed by the accumulation of high energy intermediates of the light reactions (Avenson et al. 2005). A major issue with a simple Z-scheme model for the light reactions is the lack of flexibility in the output ratios of ATP and NADPH because the proton and electron transfer reactions of LEF are tightly coupled, so that the ratio of ATP and NADPH outputs is fixed. For the light reactions, NADPH production cannot take place without ATP synthesis and vice versa (Kramer and Evans 2011). Similarly, metabolic pathways will consume ATP and NADPH at specific, different ratios than the output of LEF. For instance, if the ratio of ATP/NADPH produced by the light reactions is even slightly smaller than that used by downstream biochemistry, ATP will be depleted in a few seconds, shutting down both LEF and the Calvin-Benson-Bassham (CBB) cycle.

The potential problems associated with ATP/NADPH balance, and some solutions, are illustrated in Fig. 12.2. Three protons are expected to be transferred to the lumen for an electron transferred through LEF (Sacksteder et al. 2000). The number of protons required to be transferred through the ATP synthase to generate one ATP is thought to be determined by the stoichiometry of *c*-subunits per ATP synthase. Higher plant chloroplast ATP synthase has 14 *c*-subunits, suggesting that 14 protons are required to make a full rotation, which should produce 3 ATPs (Stock et al. 1999), but see below (Turina et al. 2003)), so that we expect $H^+/ATP = 4.67$ and the ratio of ATP/NADPH produced by LEF is expected to be 2.66, 13% lower than that needed to drive assimilation, requiring supplementary ATP production even under permissive conditions (Avenson et al. 2005; Noctor and Foyer 1998). Active transport of metabolites or ions across the chloroplast envelope can consume additional ATP, either directly as is the case for ATP-binding cassette transporters (Verrier et al. 2008), lipid transporters (Lu et al. 2007), or by consuming the proton motive force across the thylakoid or chloro-

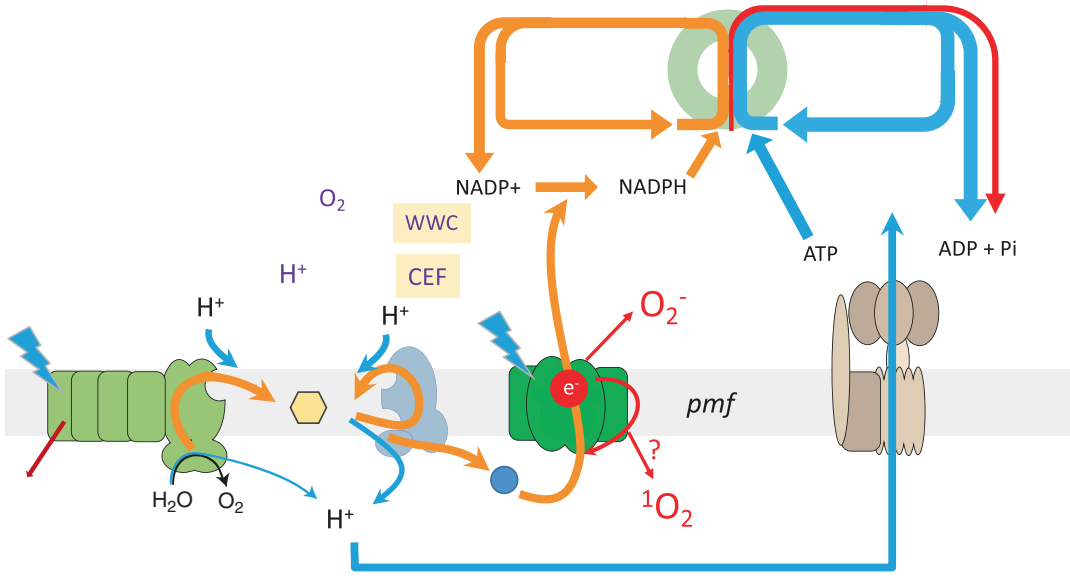

Fig. 12.2. **The problem of ATP/NADPH mismatch and possible solutions.** Alternative electron transfer pathways that can generate ATP without production of NADPH, thus augmenting the ATP/NADPH output ratio to match that of consumption. The orange arrows show the fluxes of energy through the electronic circuit of photosynthesis, starting with electron transfer (ET) through linear electron flow (LEF) to the reduction of substrates in the Calvin-Benson-Bassham (CBB) cycle. The blue arrows show the flux of energy through the proton circuit of photosynthesis, starting with the ET-coupled proton translocation into the lumen, through the ATP synthase to form ATP, which is used in phosphorylation-driven reactions in the CBB. The red arrow shows a mismatch in the supply of ATP relative to NADPH supplied by LEF with the ratio required to run CBB. The purple arrow represent possible mechanisms to augment the production of ATP without net production of NADPH, thus balancing the supply and demand for these energy-containing substrates

plast inner envelope, as is the case with ion transporters involved ion homeostasis (Spetea et al. 2016; Chap. 6). ATP/NADPH demands will be further altered by activation of other metabolic processes, so that adjustments to the energy budget should be quite flexible (Kramer et al. 2004a; Cruz et al. 2005a; Scheibe 2004). Some metabolic processes preferentially oxidize NADPH (e.g. nitrite reduction), while others preferentially consume ATP (e.g. transport of substrates, ions and proteins, and synthesis of DNA or proteins). Thus, the chloroplast needs energy balancing mechanisms that adjust both the production and consumption of ATP/NADPH.

Several processes have been proposed to overcome a deficit of ATP in the chloroplast (Backhausen et al. 1994; Kramer and Evans 2011), including the 'malate valve' (Scheibe 2004), the Mehler peroxidase reaction (MPR) (Asada 1999), the plastid terminal oxidase (PTOX) in some species, (reviewed in Joet et al. 2002b) and cyclic electron flow (CEF) (see Chap. 8). (Note that the MPR is also termed the water-water cycle because electrons are extracted from water, forming O_2, but are returned to O_2, reforming water; because other pathways, including PTOX and FLV, perform the same reaction, we thus prefer to use the term MPR). By far the most studied process is cyclic electron flow around PSI (CEF) (Kramer and Evans 2011). This process involves excitation of PSI, but not PSII, eliminating the participation of PSII (Fig. 12.2, purple curved arrows). Light-driven electron flow from PSI through Fd is shunted back to the PQ pool by several possible PQ reductases. Once PQH_2 is formed by PQ reductase activity, it is oxidized

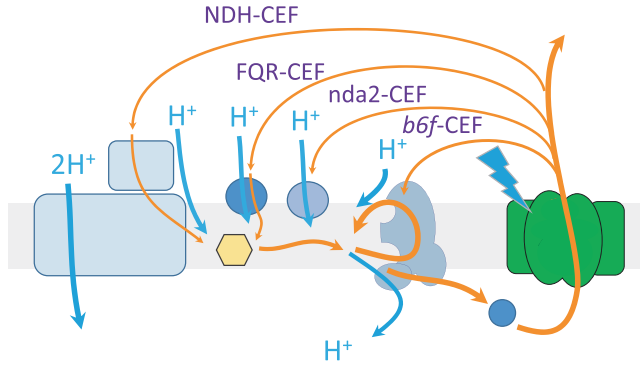

Fig. 12.3. **Cyclic Electron Flow (CEF) electron flow routes.** NDH-CEF, CEF through the NDH complex; FQR-CEF, CEF through the antimycin-A sensitive PGR5/PGRL1 pathway; nda2-CEF, CEF through the type 2 (non-proton pumping) PQ reductase; b_6f-CEF, CEF directly to redox carriers in the cytochrome b_6f complex

through the Q-cycle via the cytochrome b_6f complex, resulting in proton translocation, and delivery of electrons back to PSI. The specific mechanisms for CEF have been under intense debate, and at least four pathways have been proposed, differing at the PQ reductase step (Fig. 12.3). These pathways may operate in parallel (Scheller 1996; Holser and Yocum 1987; Munekage et al. 2004; Ducruet et al. 2005) or in species-specific ways (Havaux et al. 2005). Thylakoids from higher plants contain a type 1 (proton pumping) NADPH/Fd: plastoquinone reductase, termed the NDH complex (also NDH-like or chloroplast Complex I), which is proposed to participate in CEF. NDH is homologous to bacterial and mitochondrial (respiratory) complex I (Lascano et al. 2003). However, it is important to note that chloroplast NDH has a number of distinct properties. For example, it lacks the complex I NAD(P)H binding site, and uses Fd, rather than NAD(P)H, as its substrate (Ifuku et al. 2011). Recent work demonstrated that the higher plant NDH complex is a thermodynamically reversible proton pump that enables highly efficient ATP production by CEF (Strand et al. 2017), with a H^+/e^- stoichiometry matching that predicted for respiratory complex I (Burrows et al. 1998; Friedrich et al. 1995) that pumps up to two protons across the energetic membrane for each electron transferred to quinone (Efremov et al. 2010). The added proton pumping through NDH is expected to double the proton-to-electron stoichiometry for CEF compared to CEF through non-proton pumping pathways (Strand et al. 2017) allowing for more efficient balancing of the ATP/NADPH budget.

Other CEF pathways have been proposed that likely involve non-proton pumping PQ reductases. (It is important to note that even if these pathways do not involve a proton pumping PQ reductase, CEF will still translocate protons into the lumen through reduction and oxidation of PQ, mediated by the b_6f complex.) A distinct Ferredoxin:PQ oxidoreductase (FQR) was introduced by study of the *pgr5* mutant in *Arabidopsis*, which shows low *pmf* and slow *in vitro* PQ pool reduction by NADPH and Fd (Munekage et al. 2004). It was subsequently found that the PGRL1 protein is required for this activity (DalCorso et al. 2008). It has been proposed that PGR5:PGRL1 may act as the PQ reductase itself (Hertle et al. 2013), though other studies (Nawrocki et al. 2019) argue that PGR5:PGRL1 cannot account for the observed rates of CEF, suggesting that these proteins may play a regulatory role in CEF (Shikanai 2007). In line with this view, *pgr5* also shows a remarkably high ATP synthase activity, preventing the buildup of sufficient

lumen acidification to activate q_E. This observation implies that, while PGR5 is clearly involved in maintaining ΔpH, these effects cannot be completely accounted for by decreased CEF.

In *Chlamydomonas*, a CEF pathway has been proposed that involves a type 2 (non-proton pumping) NADPH:PQ oxidoreductase, termed Nda2, related to those found in bacteria and mitochondria (Desplats et al. 2009). Cramer and co-workers (Zhang et al. 2004), Minagawa and coworkers (Iwai et al. 2010) and Joliot and coworkers (Joliot and Joliot 2006) have proposed that CEF might occur directly through the participation of cytochrome b_6f complex c_i heme, after formation of a supercomplex containing FNR and cytochrome b_6f complex (Furbacher et al. 1989), with electrons being passed from the bound FNR to the Q_i (PQ reductase) site. Consistent with these models, the crystal structure of the b_6f complex (Kurisu et al. 2003; Stroebel et al. 2003) showed that heme c_i, to which PQ is presumably bound and reduced, is located very near the stromal surface, suggesting that it might be able to conduct electron transfer from stromal reductants. On the other hand, efforts to directly detect the reduction of the c_i/b hemes of b_6f in plant chloroplasts found only very slow rates (Fisher et al. 2018), implying that this pathway may not be rapid enough to account for all CEF.

In all cases, *pmf* produced by CEF can drive ATP synthesis to increase the ATP/NADPH output ratio, and thus activation of CEF is primarily thought to act as an energy balancing mechanism (Strand et al. 2016b). However, excessive activation of CEF will produce imbalance in the ATP/NADPH output ratio (i.e. excess ATP or NADPH deficit), that can also lead to metabolic congestion. It is thus essential that CEF be finely tuned to produce the correct amount of ATP, but not more. CEF may also play a regulatory role via its acidification of the thylakoid lumen (Kramer et al. 2004a; Golding et al. 2004; Joliot and Johnson 2011), but the coupling of any increased CEF to ATP production may also restrict the extent to which CEF can induce sufficient ΔpH to initiate q_E, without also leading to ATP/NADPH imbalances, suggesting that CEF may play a predominant role in ATP/NADPH balancing (but see below).

Consistent with the view that CEF is highly regulated, measurements in C3 plants show only small rates of CEF under steady-state, non-stressed conditions when presumably LEF by itself can nearly meet the ATP/NADPH demand required for assimilation (Laisk et al. 2007; Cruz et al. 2005a; Livingston et al. 2010b; Livingston et al. 2010a; Harbinson et al. 1989; Avenson et al. 2005). CEF is observed to increase under conditions of high ATP demand, e.g. under environmental stress (e.g. Rumeau et al. 2007; Kohzuma et al. 2009; Jia et al. 2008), when carbon concentrating mechanisms or C_4 photosynthesis are engaged (Finazzi et al. 2002; Kubicki et al. 1996), or during induction of photosynthesis in dark-adapted plants (Joet et al. 2002a; Joliot et al. 2004). These ATP/NADPH balancing and regulatory roles for CEF are not independent, since ATP/NADPH balance is critical for maintaining *pmf*, and thus regulating q_E or b_6f complex. However, CEF is clearly not essential for q_E since it is observed in its absence (Avenson et al. 2005; Ishikawa et al. 2008), while other processes can contribute to the regulation of q_E responses (Kanazawa and Kramer 2002; Kramer et al. 2004b; Avenson et al. 2004; Cruz et al. 2005b).

VI. Regulation of CEF

A previously accepted model for CEF regulation (Finazzi et al. 2002; Iwai et al. 2010) stated that CEF is linked to antenna state transitions, and is inactive in state 1, but activated in state 2. However, more recent work (Takahashi et al. 2013; Shikanai 2010) showed that CEF can occur in the complete absence of state transitions, both in algae

(Lucker and Kramer 2013; Takahashi et al. 2013) and plants (Strand et al. 2013) and that state transitions, which occur on the minutes timescale, are too slow to account for rapid regulation of CEF required to respond to abrupt changes in metabolic demands (Lucker and Kramer 2013). These results suggested CEF is regulated by other signals, such as redox status (Breyton et al. 2006), ATP levels (Joliot and Joliot 2002, 2006), and ROS production (Livingston et al. 2010b). It is also possible that these processes differentially affect different CEF pathways (Livingston et al. 2010b). A detailed regulatory pathway, a chloroplast-localized Ca^{2+} sensor (CAS) protein has been proposed for *Chlamydomonas reinhardtii*, and is posited to interact with the PGRL1 protein (and also control expression of the LHCSR3 antenna protein) (Hochmal et al. 2015; Terashima et al. 2012), but the means by which CEF is regulated in plants and cyanobacteria is, in general, not well understood. Very recently we found (Fisher et al. 2019) that, in plants, the chloroplast NDH and FQR pathways are inhibited by stromal adenylates (ATP and ADP) at physiologically relevant concentrations (K_i (ATP) *in vitro* ≈ 0.5 mM), with ADP a two- to threefold weaker inhibitor of these pathways. Importantly, ATP appears a stronger inhibitor of FQR compared to NDH, such that the more energy efficient CEF pathway afforded by NDH ($8H^+/2e^-$, compared to the predicted $4H^+/2e^-$ for the FQR pathway) would be operational under conditions when ATP concentrations may be expected to be limiting for downstream metabolism.

In our previous work, we identified a series of mutants with constitutively high rates of CEF, and used these to assess possible regulatory mechanisms (Strand et al. 2015, 2016c; Livingston et al. 2010a, b). We noticed a striking pattern in which essentially all of these mutants showed elevated production of H_2O_2 and pondered whether these effects were functionally linked. We thus tested the hypothesis that CEF is activated by H_2O_2 *in vivo* (Strand et al. 2015). We found that CEF through the type 1 NDH pathway was strongly increased by H_2O_2, both by infiltration or *in situ* production by chloroplast-localized glycolate oxidase, implying that H_2O_2 can activate CEF either directly by redox modulation of key enzymes, or indirectly by affecting other photosynthetic enzymes. We propose that H_2O_2 may be a "missing link" between environmental stress, metabolism, and redox regulation of CEF in higher plants. Relatedly, we have found recently that including reduced dithiothreitol (DTT) in spinach thylakoid preparations (where the FQR-mediated CEF pathway is dominant) preserves *in vitro* CEF activity (where it is normally rapidly lost on tissue disruption). The midpoint potential of this preservatory component was determined to be −306 mV at pH 7.6, titrating as an $n = 2(e^-)$ species, therefore most likely a thiol (Strand et al. 2016a). As such, the apparent thiol-mediated regulation of FQR CEF would allow this particular pathway to respond more rapidly to metabolic demands under conditions of oxidative stress given the slower activation of NDH CEF in response to exposure to H_2O_2 (halftime for activation of approximately 20 min) (Strand et al. 2015).

VII. Modulation of *pmf* Feedback Regulation and Its Impact on Energy Balancing

In the simplest formation of the *pmf* paradigm, lumen acidification is determined by the rate of proton translocation, explaining why q_E responds to increasing light, i.e. higher increased proton flux, which increases lumen acidification. However, photoprotection must also respond to changes in downstream capacity to utilize the products of the light reactions. Indeed, increased q_E is observed under adverse environmental conditions when the LEF and associated proton fluxes decrease (Kramer et al. 2004a). These observations imply that *pmf* feedback is modulated (or regulated) by downstream pro-

cesses either by modulating the extent or sensing of lumen acidification. For the purposes of this review, we divide these processes into two distinct classes of mechanism (Kramer et al. 2004a): Type 1 modulation involves processes that change the production or sensing of lumen pH without affecting the ratios of production of ATP and NADPH. For example, altering the concentrations of Z by regulating biosynthesis of xanthophylls or the activities of VDE or ZE should change the activation of q_E at a particular lumen pH without directly affecting the relative output of ATP/NADPH, as is seen in a number of mutants affecting these enzymes (Zhang et al. 2009; Murchie and Niyogi 2011; Li et al. 2004), or under diverse environmental conditions (Demmig-Adams et al. 2017). Another type 1 mechanism involves adjusting the activity of the ATP synthase, which is known to be modulated by metabolic or environmental factors (e.g. low CO_2 levels (Kanazawa and Kramer 2002), drought (Kohzuma et al. 2009), and feedback limitations (Takizawa et al. 2008)). Slowing the ATP synthase decreases the conductivity of the thylakoid to protons (g_{H^+}), leading to buildup of the *pmf* even at similar proton input rates, and subsequent acidification of the lumen and enhanced q_E and photosynthetic control (Avenson et al. 2005; Cruz et al. 2005a; Kanazawa and Kramer 2002).

Type 2 mechanisms modulate lumen pH by activating non-LEF light-driven processes, such as CEF and MPR. These processes necessarily alter the ratio of ATP/NADPH produced by the light reactions. In principle, the acidification of the lumen associated with these processes should activate q_E or induce photosynthetic control (Shikanai and Yamamoto 2017; Shikanai 2007; Johnson 2004). However, they also alter the ATP/NADPH output ratio of the light reactions, which as discussed above, can lead to mismatches in the energy budget of the chloroplast, likely limiting the degree to which these processes can be activated, as will be discussed in more detail below.

A special case of feedback modulation is partitioning of *pmf* into $\Delta\psi$ and ΔpH. Because ΔpH, but not $\Delta\psi$, results in lumen acidification, changing the fraction of *pmf* stored in these two forms should control the regulatory impact of a particular extent of *pmf* (Kramer et al. 2003; Avenson et al. 2004). For instance, suppose that the steady state *pmf* is held at 180 mV. If this *pmf* was stored exclusively as $\Delta\psi$, then ΔpH = 0. With no ΔpH, and stromal and lumen pH of 7.8, no q_E should be activated. If, instead, this *pmf* is equally partitioned, $\Delta\psi$ and ΔpH should be approximately 90 mV and 1.5 pH units respectively, and lumen pH should thus reach about 6.3, where q_E should be substantially activated (Takizawa et al. 2007). If all *pmf* is stored as ΔpH, lumen pH should reach 4.8, which should induce maximal activation of q_E as well as very strong slowing of PQH_2 oxidation, such that LEF would become very slow (Kramer et al. 2003), and possible acid-induced photodamage to PSII centers (Krieger and Weis 1993; Spetea et al. 1997; Kramer et al. 1999). Further, the need to sustain a sufficiently large *pmf* to maintain stromal ΔG_{ATP} should preclude 100% ΔpH storage, as this would lead to induction of q_E in the dark and such extreme lumen acidities that the system could not work efficiently (Kramer et al. 1999). It is thus likely that *pmf* partitioning is adjusted to balance the needs for energy storage and regulation, as is seen under adverse environmental conditions (e.g. Avenson et al. 2004; Zhang et al. 2011; Davis et al. 2017).

It was originally suggested (Kramer et al. 2003) that *pmf* partitioning would act as a Type 1 regulator, because under steady state conditions, changing the fraction of *pmf* stored as ΔpH should not affect ATP/NADPH output. However, a detailed computational model of the light reactions (Davis et al. 2017) showed that abrupt changes in light intensity should transiently affect ATP/NADPH output. As illustrated in Fig. 12.4, the simulations showed that, under many conditions, the rate of q_E formation will

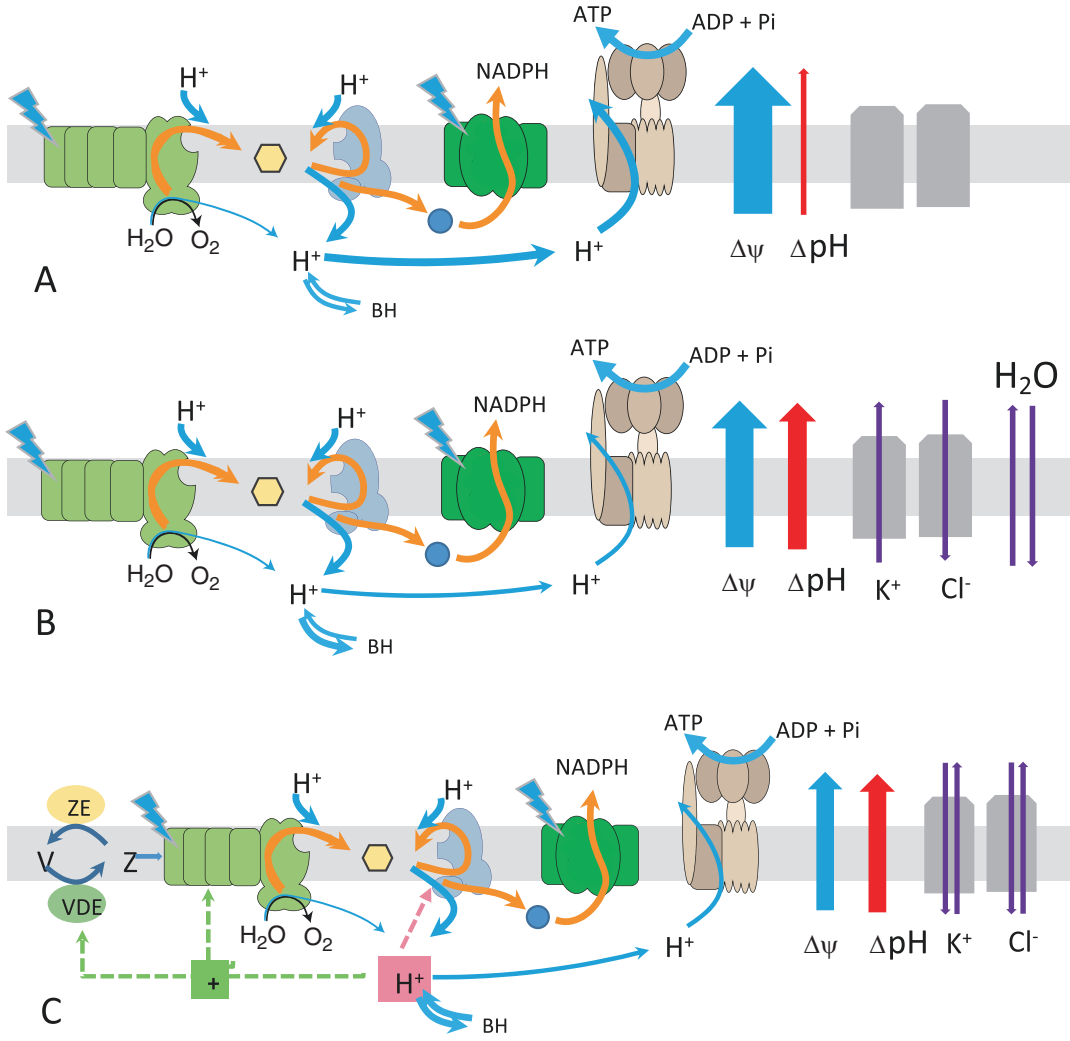

Fig. 12.4. **Counterion exchange modulates Δ*p*H, resulting in activation of qE, but imposing deficits in proton translocation and ATP synthesis.** *The blue arrows on the upper thylakoid membrane show the directions of transmembrane electron flow that generates Δψ, while the red arrows show the uptake and deposition of protons that generates ΔpH.* The semi-transparent arrows passing over the ATP synthase indicate the relative contributions of Δψ (blue) and ΔpH (red) to the ATP synthase reaction. (**a**) At early times after illumination, *pmf* is stored predominantly in Δψ, owing to the low electrical capacitance of the thylakoid membrane and the large proton buffering capacity of the lumen. The high Δψ effectively drives the efflux of protons from the lumen through the ATP synthase, maintaining a low ΔpH. Without counterion fluxes, the *pmf* would remain predominantly in Δψ. (**b**) Activating counter-ion fluxes dissipates a fraction of Δψ, allowing additional proton influx and less efflux, which gradually protonates lumenal buffering groups, forming a ΔpH. (**c**) Counter-ion fluxes establish ion gradients that eventually reach a local equilibrium with Δψ, leading to a steady-state ratio of Δψ: ΔpH. The acidification of the lumen due to the generation of ΔpH can then downregulate the cytochrome b_6f turnover (triangle) and activate q_E (dark blue circle). However, the dissipation of Δψ by counterion transport results in loss of *pmf*. We propose that the proton (ATP) deficit is filled by activating CEF.

likely be limited by protonation of PsbS, which is determined by ΔpH formation, in turn controlled by the (slow) rates of counter-ion fluxes across the thylakoid (Davis et al. 2016, 2017). Owing to the low electrical capacitance of the thylakoid membrane (Junge and Witt 1968) and the large proton buffering capacity of the lumen (Junge et al. 1979), the initial charge separations at PSI and PSII induce a large *pmf* almost exclusively in the form of Δψ (Cruz et al. 2001). The large Δψ is sufficient to force protons to exit through the ATP synthase, preventing the formation of ΔpH, and limiting the rate of q_E activation (Fig. 12.4, panel A). Only when counterions are allowed to pass across the thylakoid membrane (Fig. 12.4, panel B), collapsing part of Δψ, can protons accumulate to overcome the lumen buffering capacity (Fig. 12.4, panel C). Thus, counter-ion translocation that dissipates Δψ is not only *essential* for lumen acidification-induced photoprotection, it is likely the limiting step under many conditions (Davis et al. 2017). Dissipation of Δψ requires either an influx of anions (probably Cl^-) into the lumen, or effluxes of cations (K^+, Na^+, Mg^{2+}, Ca^{2+} etc.) from the lumen (Pottosin and Schönknecht 1996; Cruz et al. 2001; Barber 1976). The extent and kinetics of Δψ dissipation are finely controlled by a series of additional ion transporters, including K^+ and Cl^- channels and a K^+/H^+ antiporter (Spetea et al. 2016; Armbruster et al. 2016). In addition, mobile buffers, such as the polyamine putrescine or bicarbonate may play a role in modulating Δψ/ΔpH by altering the proton buffering capacity of the lumen (Ioannidis et al. 2012).

Along with the metabolic and regulatory processes involved in the generation and dissipation of the *pmf,* the movement of protons and ions into the thylakoid lumen during photosynthesis results in ultrastructural changes in thylakoids (Deamer et al. 1966; Kirchhoff et al. 2011), as illustrated in Fig. 12.4, panel B. Experiments with isolated chloroplasts with addition of various salt concentrations or ionophores to intact or broken chloroplasts showed that the thylakoid was permeable to specific ionic species, and that these had large effects on photosynthetic electron transfer and light absorbing properties of the antenna complexes (Barber 1976), including grana stacking (Mg^{2+}), Rubisco activity (Mg^{2+}), and oxygen evolution (Cl^- and Ca^{2+}). Thus, at a minimum, ion transporter systems must be present to facilitate delivery of necessary ions into the chloroplast compartments. In higher plant chloroplasts, illumination of dark-adapted samples results in expansion of the internal lumen volume of thylakoids (Kirchhoff et al. 2011), likely accomplished by light-induced chloride influx into the lumen (Schönknecht et al. 1988; Cruz et al. 2001), followed by water influx (see arrows indicating H_2O flux in Fig. 12.4, panel B), altering the diffusion rates of soluble lumen proteins. Very recently, a connection has been identified between hyperosmotic stress responses and the chloroplast ionic homeostatic machinery triggered through a Ca^{2+}-signaling pathway (Stephan et al. 2016). Therefore, the chloroplast must simultaneously modulate the Δψ and ΔpH components of *pmf*, prevent fluctuations in osmotic balance, and ensure that the concentrations of ionic cofactors are maintained.

The simulations show an additional effect of ion translocation on energy balance. Overcoming the buffering capacity of the lumen requires the electrogenic transport of an ion, which diverts one proton from ATP synthesis to counterion transport, leading to a deficit of ATP relative to NADPH (see Fig. 12.4 panel B). In other terms, the dissipation of Δψ by counterion fluxes, required to build ΔpH, results in a loss of *pmf* that would otherwise drive the ATP synthase (Davis et al. 2017). The simulations predict that the ATP deficit can be substantial, and generating a ΔpH of one unit should consume the equivalent of ATP needed to fix 140 CO_2 molecules per PSII complex. This deficit must be balanced in some way to prevent metabolic congestion, and we proposed that

CEF may be transiently activated to meet this limitation. Because of the slow relaxation of the $\Delta\psi$ component during photosynthesis, as well as the slow decay of the $\Delta\psi$ in plants or algae under conditions where synthase is inactive (Cruz et al. 2001, 2005b), it can be concluded that the counterion movements across thylakoids in plants and green algae are 2–3 orders of magnitude slower than movements of protons (see Chap. 6), so that ATP deficit induced by ΔpH formation is spread out over a lengthy time (tens of seconds to minutes), requiring only a small contribution from CEF. However, speeding up the formation and recovery of q_E, as has been suggested, could increase plant productivity (Kromdijk et al. 2016), should impose an acute, albeit transient, ATP deficit. Without an equally rapid mechanism to refill the proton pool, this proton drain may disable photoprotection, and lead to the accumulation of electrons on PSI and PSII. Thus, this tradeoff may explain the rather slow onset and decay of q_E. One possible remedy to circumvent this problem would be to introduce a mechanism to replenish the lost protons; for example, a very rapidly activated and robust form of CEF, though it is an open question whether such mechanisms have already evolved.

VIII. How Diverse Photoprotective Mechanisms Challenge the *pmf* Paradigm and Open Up New Questions

Above, we described how cross talk between the two critical roles of *pmf*, storing energy and activating photoprotection, require multiple levels of regulation. A key lesson is that there will be far-reaching and unexpected consequences of changing any one component of this hub. It is also clear from genetic and physiological analyses that some of these components are likely not conserved across the diversity of photosynthetic species, leading to questions about how a *pmf*-feedback regulatory system could work, or if the *pmf* paradigm may be replaced with completely different modes of regulation.

It is important to point out that, the overall *pmf* paradigm can still operate even if specific components behave differently (Goss and Lepetit 2015). Even in plants, different forms of NPQ occur simultaneously with q_E, including the long-lived forms q_I and q_Z, related to the damage-repair cycle of PSII and the accumulation of Z (Adams III et al. 2006). In diverse species, the mechanistic details can vary. For example, q_E in *Chlamydomonas* involves "light stress" forms of light harvesting complexes, LHCSR1, LHCSR3, rather than PsbS (Peers et al. 2009; Tokutsu and Minagawa 2013; Bonente et al. 2011; Chap. 4), though these are similarly responsive to acidification of the lumen, which is controlled by changes in *pmf* partitioning (Cruz et al. 2005a) and activation of CEF (Lucker and Kramer 2013). The moss *Physcomitrella patens* contains both the PsbS and LHCSR3 system (Gerotto et al. 2011). In the stramenopile *Phaeomonas* sp., the extent of NPQ is strictly proportional to the accumulation of Z, i.e. it lacks the more rapid adjustment of NPQ by protonation of PsbS or LHCSR3 (Berne et al. 2018). In diatoms and dinoflagellates, the xanthophyll cycle is replaced by a diadinoxanthin cycle, in which the lumen pH-activated diadinoxanthin de-epoxidase catalyzes the one-step de-epoxidation of diadinoxanthin into diatoxanthin (Goss and Lepetit 2015). In the red alga, *Cyanidioschyzon merolae*, lumen acidification apparently induces NPQ within the reaction center complex itself (Krupnik et al. 2013).

Ion movement across the thylakoid, in general, has been observed for decades (Barber 1976), yet there appears to be evidence for more ion transport activities from experimental data than can be accounted for by the small number of transport proteins thus far identified (Chap. 6). This mismatch means that either the transporters are not very specific or that their low content in the membranes has prevented unam-

biguous identification by bulk proteomic analyses (van Wijk 2004). Genetics and reverse genetics approaches have identified a limited number of mutants in *Arabidopsis thaliana* ion channels and transporters at both the thylakoid and chloroplast inner envelope membranes (Spetea et al. 2017). We know even less about ion transporters in algae and cyanobacteria (Pfeil et al. 2014). In *Chlamydomonas reinhardtii*, in which electrochromic shift measurements of the *pmf* are well-developed, alterations in the $\Delta\psi$:ΔpH ratio have been observed (Cruz et al. 2005b), similar to that seen in higher plant chloroplasts (see above). Comparative genomic analyses suggest that *Chlamydomonas* contains ion channels with similar sequences to those in Arabidopsis (Marchant et al. 2018), but their activities have yet been demonstrated. In the model cyanobacterium *Synechocystis* sp. PCC 6803, the demonstration of ion channel activities have been limited to observations that altering the expression of a putative potassium channel affects the turnover rate of b_6f complex, consistent with a role in lumen pH homeostasis (Checchetto et al. 2012).

Prokaryotic phototrophs have evolved a wide range of antenna systems, some of which are unlikely to interact with the *pmf* in the same way as those in plants (Kirilovsky and Kerfeld 2012). In some cyanobacteria, light capture by the cytosolic phycobilisomes (PBS) is regulated by the orange carotenoid protein (OCP) and the fluorescence recovery protein (FRP) system, in which strong excitation with blue-green light leads to the conversion of the orange to the red form of the protein, allowing it to attach to PBS and quench excitons (Wilson et al. 2006). The process is reversed by the activity of the FRP, which converts OCP back from the red to the orange form (Kirilovsky and Kerfeld 2016). PBS can also shunt energy to either PSII or PSI through state transitions that appear to be responsive to the redox state of the cell (Ranjbar Choubeh et al. 2018).

Photoprotection in these systems are not directly induced by lumen acidification, opening the question of whether the *pmf* paradigm is needed. Of particular interest is whether photosynthetic control in cyanobacteria is needed to prevent PSI photodamage. Evidence for a substantially acidified lumen and subsequent photosynthetic control was presented by Trubitsin et al. (2005) and it was further suggested that the formation of ΔpH involves dissipation of $\Delta\psi$ by ion transporters (Checchetto et al. 2012). However, it has also been suggested that, at least in some cases, photosynthetic control is not needed or would hinder efficient photosynthesis (Badger et al. 2000/see also below).

Under some conditions, e.g. low temperature, high temperature (Diaz et al. 2007; Quiles 2006), or high salt (Stepien and Johnson 2009), plant chloroplasts can dissipate small amounts of energy by diverting electrons from the light reactions to O_2 but this mechanism appears to be much more robust in cyanobacteria and algae (Houille-Vernes et al. 2011; Peltier et al. 2010), and can account for a substantial fraction photoprotection in green algae, red algae, and dinoflagelletes (Badger et al. 2000). There are at least three mechanisms for dissipating photosynthetic energy to O_2, the Mehler-peroxidase or Water-Water cycle (Asada 1999), oxidation of PQH_2 by the plastid terminal oxidase (PTOX) (Krieger-Liszkay and Feilke 2015), and the flavodiiron proteins (FLV) (Chukhutsina et al. 2015; Allahverdiyeva et al. 2013).

The FLV system is found in the cytoplasm of many (but not all) cyanobacterial species, in most eukaryotic algae icluding green algae and in moss stroma (Chaux et al. 2017; Dang et al. 2014). FLV1- and 3, functioning as a heterodimeric enzyme, can accept four electrons (sequentially) from PSI-reduced Fd, and reduce O_2 to two molecules of water. Much like NPQ, which dissipates light energy, the diversion of electron flow from PSI to O_2 effectively prevents the buildup of highly reducing electron carriers, minimiz-

ing production of ROS (Jokel et al. 2018). In contrast to the Mehler-peroxidase cycle, the 4-electron oxygen reductase chemistry of FLV1/3 probably results in the formation of water, not superoxide, though there is some question about the precise mechanism (see Chap. 8 by Raven, Beardall and Quigg). It has been proposed that upwards of 30% of the electrons derived from water oxidation may be shunted through the FLV1/3 system under high light conditions in cyanobacteria (Helman et al. 2003). It has been demonstrated in both cyanobacteria and green algae that knocking out FLV proteins can lead to severe photodamage, especially under high or fluctuating light (Shimakawa et al. 2016; Jokel et al. 2018). It can also be argued that electron flow through FLV can augment ATP synthesis and replace the need for CEF (Dang et al. 2014). Intriguingly, the FLV2- and − 4 proteins, which are unique to β-cyanobacteria, have been posited to have a photoprotective role for PSII, either *via* direct oxidation of the Q_B-site semiquinone (i.e. functioning as an electron sink) or through modification of PBS association with PSII (Zhang et al. 2012).

In higher plant tissues, PTOX plays an essential role in plastid development as its plastoquinol oxidase activity is required for carotenoid biosynthesis (a role which is undertaken by the cytochrome b_6f complex in mature chloroplasts) (Josse et al. 2000). PTOX is also likely to participate in a 'chlororespiratory' pathway within the thylakoid (the light-independent linear electron flow of electrons from NADPH (or Fd) to oxygen), of which NDH has also been proposed to be a component of in plants (Bennoun 2002). While a role for PTOX in plastid development has been established, its role in photosynthesis and stress responses remains unclear, likewise the importance of chlororespiratory processes within the chloroplast. PTOX has been proposed to function as an alternative electron sink under conditions where the acceptor side of PSI is limited (McDonald et al. 2011), and it has been shown to be induced by a variety of stress conditions, including cold, heat, salinity, drought (Diaz et al. 2007; Quiles 2006; Ibanez et al. 2010), low temperature and high light (Ivanov et al. 2012), and combinations of low temperature and high UV exposure (Streb et al. 2005; Laureau et al. 2013). According to the original model proposed (Huner et al. 1998), a single stress alone does not induce the response. Rather, the acclimation response occurs when a high excitation pressure is coupled with an environmental stress.

Several groups have proposed that PTOX in eukaryotic algae and higher plants acts as a safety valve in the light under abiotic stress conditions by preventing the "over-reduction" of the PQ pool, which would lead to accumulation of electrons on PSII (see Chap. 8). For example, when PTOX was found to be upregulated in sun vs. shade leaves in the alpine plant *Ranunculus glacialis* L., it was concluded that the adaptation may help ameliorate the rapid light and temperature changes that occur in the plant's climate. The work showed that PTOX content correlated with the magnitude of electron flow unrelated to the carboxylation and oxygenation of rubisco, strongly suggesting that PTOX acts as safety valve to relieve pressure on the electron transport chain when the PQ pool is highly reduced (Laureau et al. 2013). Similarly, when rice PTOX was overexpressed in the cyanobacteria *Synechocystis* sp. PCC 6803, the NAD(P)H/NAD(P)$^+$ pool became highly oxidized, consistent with the model of PTOX being a redox release valve, although care is needed in interpreting these data due to the highly branched nature of competing electron transfer pathways in cyanobacterial thylakoids, which also contain respiratory complexes (Ermakova et al. 2016). The overexpression of PTOX also led to decreases in the ratio of PSII/PSI suggesting that PTOX may play a role in (direct or indirect) signaling that regulates PSI and PSII biosynthesis, perhaps by altering the thiol redox state (Feilke et al. 2017). PTOX has also been shown to func-

tion as an electron sink in Chlamydomonas and its loss decreases fitness under low (40 μmol photons m^{-2} s^{-1}) light (Houille-Vernes et al. 2011).

Other studies cast some doubt on the PTOX is a safety valve model, or at least call into question whether introducing PTOX into exotic species will lead to "safer" photosynthesis. A study in tomato demonstrated that the enzymatic properties of PTOX are not compatible with high fluxes required for a robust safety valve (Trouillard et al. 2012). Overexpression of PTOX in *A. thaliana* did not decrease light-induced photodamage (Rosso et al. 2006), and over-expression in tobacco (Krieger-Liszkay and Feilke 2015) resulted in higher photoinhibition and ROS production under high light. Also, PTOX overexpressing plant thylakoids showed decreases in oxygen evolving activity beyond that accounted for by diversion of electrons to O_2 through PTOX, and higher levels of superoxide ($O_2^{\cdot -}$) production. Experiments with *E. coli* expressing PTOX showed that PTOX activity leads to the generation of ROS when the quinone pool is over-reduced (Heyno et al. 2009). Overall, these results suggest that in plants, PTOX mainly plays a role in maintaining the redox state of the PQ pool during chloroplast biogenesis and the assembly of the photosynthetic apparatus, but in high light may result in $O_2^{\cdot -}$ production (Heyno et al. 2009).

The emerging picture seems to be that, in higher plants, PTOX is highly regulated to operate only under specific stress conditions (Stepien and Johnson 2018; Feilke et al. 2016). This view reconciles discrepancies between earlier work because PTOX activities are kept low, either by low expression levels of regulation at the enzyme level, under non-stressed conditions (Feilke et al. 2016). Consistent with this view, overexpressing PTOX does not necessarily increases its overall activity (Stepien and Johnson 2018), whereas exposure to abiotic stresses does (Feilke et al. 2016). In dark adapted tomato plants, PTOX showed sluggish activity (Trouillard et al. 2012), but under abiotic stress, its activity increases to the point where it could account for a substantial fraction of LEF (Feilke et al. 2016) and in stressed *Extrema* (previously *Thellungiella halophila*), PTOX-dependent electron flow accounted for up to 30% of the total LEF (Stepien and Johnson 2009).

The mode of regulation of PTOX is not yet settled, but it has been proposed that the association of PTOX (which is a monotopic membrane protein) with the stromal face of the thylakoid membrane, and therefore its activity, is controlled by pH, which in turn is responsive to the metabolic state of the stroma, especially the activity of the Calvin-Benso-Bassham cycle (Feilke et al. 2016). Another hypothesis is that PTOX regulation involves translocation of the protein from the stroma lamellae into proximity with the PQ pool associated with PSII (Stepien and Johnson 2018). In any case, the flux of electrons through PTOX should also be responsive to the redox state of the PQ pool, which is determined by the flux of electrons into the pool through PSII and out of the pool through the b_6f complex, which are both regulated by the *pmf* through effects on NPQ and photosynthetic control (Strand and Kramer 2014).

IX. Coping with ATP Excess or NADPH Deficit

The previous section emphasizes an important theme of this paper: that while O_2 reduction by PTOX, FLV and MPR have been considered to act as safety valves for the electronic circuit of photosynthesis, these valves may themselves be unsafe in that they perturb the energy balance of the photosynthetic system, and thus may impose the need for a second safety valves for the protonic circuit. Badger et al. (2000) recognized this potential problem, and suggested that, in algae and cyanobacteria that have larger capacity for O_2 reduction, the additional LEF needed to rebalance the energy budget may not be uncoupled from ATP pro-

duction, e.g. if electron transport through the cytochrome b_6f complex was not controlled by lumen pH, but by stromal side regulatory processes, such as the redox state of NADPH or the presence of CO_2/bicarbonate. Indeed, one could imagine adaptations that decreased the canonical photosynthetic control mechanisms, e.g. changing the pH dependence of the cytochrome b_6f complex or storing more *pmf* in the form of $\Delta\psi$. However, in our view, the thylakoid would still need to dissipate $\Delta\psi$ to prevent accelerated recombination reactions that produce $^1O_2$ (Rutherford et al. 2012; Davis et al. 2016).

While much research has been reported on responses to ATP deficits, little is known about how photosynthetic systems cope with the opposite: excess ATP or limiting NADPH (Fig. 12.5). One way to adjust cellular ATP/NADPH would be to decrease the rate of ATP synthesis under NADPH deficit (or ATP excess) and there is some evidence that mitochondrial respiration through complex IV is inhibited by ATP levels (Ramzan et al. 2010; Bender and Kadenbach 2000; Kadenbach and Arnold 1999), though to our knowledge this has not been tested in photosynthetic systems. Also, thylakoids from cyanobacteria (Vermaas 2001) and (in likelihood) green alga (Bennoun 2002) contain respiratory enzymes and there is evidence that these interact (Pringault et al. 2005; Scherer et al. 1988; Binder 1982) possibly leading to regulation of ATP/NADPH balance. Of course, this type of mechanism could only account for imbalances smaller than ATP formation by these respiratory pathways, and given that for a phototroph, photosynthesis must run faster than respiration, there would seem to be situations where additional balancing is needed.

It may also be that the production of ATP is decreased or that ATP sinks are upregulated. However, because productive sinks are likely to become saturated at high rates, it seems more likely that these processes act more like safety valves that dissipate excess energy, rather than productive energy stor-

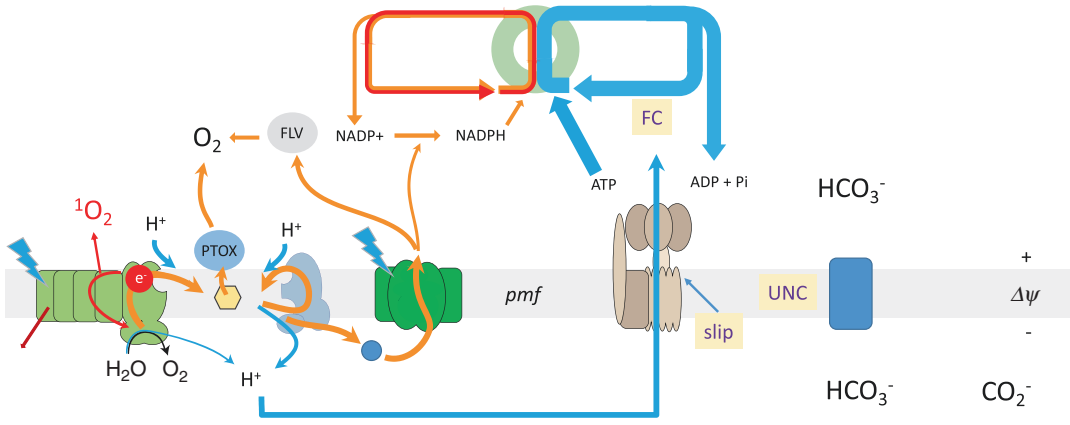

Fig. 12.5. **Energy imbalances caused by excess ATP or insufficient NADPH production.** The production ratio of NADPH/ATP may at times be insufficient to maintain metabolic pathways, e.g. when rates of O_2 reduction by PTOX or FLV are rapid, leading to a decrease in NADPH and increase in ATP, leading to mismatches between production and consumption of ATP (blue arrows) and NADPH (red curves). The depletion of ADP and P_i would be expected to slow the ATP synthase, increase *pmf*, slow electron flow at the cytochrome b_6f complex and lead to accelerated recombination reactions in PSII, leading to $^1O_2$ production (red arrows). Several processes may allow rebalancing of the energy budget (purple arrows) including uncoupling (leakage) protons across the thylakoid or through the ATP synthase, and various *pmf* or ATP-consuming futile cycles, e.g. the pumping of bicarbonate into the lumen, followed by escape of CO_2

age. Early evidence in favor of high rates of slippage (Schonfeld and Neumann 1977; Braun et al. 1991) were shown to be artifacts of isolation in the absence of nucleotides and Mg^{2+} (Groth and Junge 1993), though it has also been argued that such conditions are physiologically relevant (Feniouk et al. 1999). Indeed, the structural/mechanistic basis for proton-driven ATP synthesis involves mechanical coupling between two rotating subcomplexes, and it seems conceivable that slippage could occur, and it has been proposed that such slippage may be beneficial (Nelson et al. 2002). Another possibility is partially uncoupling the thylakoid membrane, as seen with uncoupler proteins (UCPs) (Jarmuszkiewicz 2001), which allow protons to pass through a membrane, bypassing ATP synthesis. UCPs have been found in animals, higher plants and some microorganisms, but as far we know, not in thylakoids. In plants, mitochondrial UCPs have been shown to be important for responses of photosynthesis to abiotic stresses, most likely by relieving oxidative stress during photorespiration (Sweetlove et al. 2006), but a recent paper showed that these proteins are more likely to act as transporters of aspartate, glutamate and dicarboxylates (Monne et al. 2018). To our knowledge, no canonical UCPs have not been found in cyanobacteria, though it is possible that unrelated proteins with similar functions have evolved.

Futile cycling may also be induced to consume ATP. One possible futile cycle involves the carbon concentrating mechanism, which under certain conditions may conduct a futile cycle that dissipates *pmf* and thus ATP (Fig. 12.5, purple arrows). In green algae, such a futile cycle could involve transport of bicarbonate across the thylakoid into the lumen (via an electrogenic transporter), followed by hydrolysis in the lumen of bicarbonate + H^+ to CO_2 + H_2O, and escape of the CO_2 across the non-pyrenoid segments of the thylakoid membrane (Wang et al. 2011; Badger 2003). Similarly, the uptake of HCO_3^- by cyanobacteria involves active transport down the ATP-hydrolysis-driven electrochemical potential gradient of the cell membrane, and therefore will consume ATP (Mangan and Brenner 2014). Metabolic futile cycles are also possible. For example we previously proposed (Livingston et al. 2010b) that in the Calvin-Benson cycle, 3-phosphoglycerate (3-PGA) is phosphorylated to 1,3-bisphosphoglycerate (1,3-BPG), which is reduced to glyceraldehyde-3-phosphate (GAP) via GAP dehydrogenase (GAPDH). However, 1,3-BPG is very unstable and if GAPDH activity is low, e.g. when NADPH is limiting, should decay with loss of P_i, to reform 3-PGA, forming an effective ATP-consuming futile cycle activated when NADPH content is low. It is unclear whether any of these reactions occur in cyanobacteria.

A. Energy Balancing by Interactions Between Photosynthetic and Respiratory Machinery

For cyanobacteria, it is likely that when Flv1/3 complexes are functional, other respiratory chain oxidases are not essential (Ermakova et al. 2016). However, under conditions of increased ATP requirements, e.g. under fluctuating light, when presumably repair of cellular machinery is required, the cytochrome c oxidase (COX) and/or the cytochrome bd quinol oxidase (Cyd), two membrane-localized respiratory terminal oxidases (RTOs), are necessary for survival. The Cyd oxidizes PQ/PQH_2 directly, and is not in itself a proton pump, but participates in proton translocation by reducing O_2 on the stromal side of the thylakoid, taking up 1 H^+/e^-. COX oxidizes PC or cytochrome c_6 and reduces O_2 to H_2O, like Cyd, but also directly pumps 2 H^+/e^- (Iwata et al. 1995; Branden et al. 2006). In respiration, PQ is reduced by a non-photoactive reductase, e.g. complex I or II, but because the respiratory enzymes are localized in the thylakoid in cyanobacteria, it is possible that electrons from PSII can also participate in respiration, through the activi-

ties of Cyd or the combination of b_6f complex PC/c6 and COX, leading to the generation of a *pmf* and ATP, without the reduction of $NADP^+$ (essentially another water-water-like cycle). Evidence has been presented that such alternative electron transfer pathways can occur in cyanobacteria, for example when the b_6f complex is blocked, PQH_2 can be oxidized by Cyd, and in mutants lacking PSI electron flow can occur to COX (Ermakova et al. 2016).

There is evidence that some eukaryotic algae have evolved energy balancing mechanisms that rely on cross talk between the chloroplast and the mitochondria. In diatoms, the chloroplast-localized energy balance mechanisms (i.e. CEF, MPR, FLV) do not appear active enough to modulate ATP/NADPH energy fluxes (Wilhelm et al. 2006; Lepetit et al. 2012; Bailleul et al. 2015), but instead appear to channel excess reducing power generated in the chloroplast to the mitochondria, where the reductant is utilized to generate ATP via oxidative phosphorylation. This ATP is then brought back to the chloroplast, to power processes such as the carbon assimilation (Bailleul et al. 2015). This rerouting of the electronic circuit of photosynthesis appears to involve the malate shuttle, which transfers reducing equivalents by transporting the malate/aspartate redox couple across the chloroplast and mitochondrial membranes, allowing equilibration of redox potentials of the $NAD(P)^+$/NAD(P)H couples in the two compartments. The rapid fluxes needed to balance the photosynthetic energy budget may be facilitated by the close proximity of the chloroplast and mitochondria in these organisms (Prihoda et al. 2012).

An intriguing, but relatively unrecognized bioenergetic complex is the the pyridine nucleotide transhydrogenase (PntAB), which catalyzes the reversible hydride transfer between NAD^+ and NADPH, coupled to the pumping of protons across a bioenergetic membrane (Jackson 2012). In mitochondria, PntAB is thought to use the *pmf* to maintain the NADPH and NADH out of equilibrium, favoring the reduction of NADPH, which in heterotrophic organisms is used to power reductive (anabolic) reactions, whereas NADH tends to be involved in breaking down molecules to supply energy for cellular processes. Interestingly, PntAB is also found in some cyanobacteria, in which it is expressed in the same bioenergetic membrane (thylakoid) as both the photosynthetic and respiratory reactions occur. During photosynthesis, LEF will reduce $NADP^+$ to NADPH. Transferring the hydride from NADPH to NAD^+ will result in the pumping of a proton from the lumen to the cytoplasm, storing energy in the *pmf*, and possibly contributing to ATP synthesis, though it is not clear how large fluxes of electrons to NAD^+ could be maintained. It is more likely that PntAB plays a role under respiratory conditions, where *pmf* formed during oxidative phosphorylation will be used to maintain a more reducing NADPH pool, as would be required to power biosynthetic reactions, presumably at night when photosynthesis is not actively reducing NADPH. Consistent with this interpretation, mutants of *Synechocystis* deficient in PntAB exhibit growth defects when grown under hetertropic, low light conditions, but not under autotrophic conditions (Kamarainen et al. 2017).

X. Conclusions and Perspective

The photoprotective and energy balancing mechanisms of photosynthesis are interconnected in complex ways, such that diversity of one component will have far reaching consequences that need to be recognized to understand the efficiency and robustness of photosynthesis in different environments. The *pmf* paradigm is probably partly, but not completely, conserved across evolutionarily diverse phototrophs. For example, while the ΔpH-induced regulation of light capture in plants is not conserved in cyanobacteria, the need to maintain *pmf* in a range

that allows efficient production of ATP but does not induce photodamage still exists. Though relatively little is currently known about these systems in diverse algal organisms, there will almost certainly be unexpected mechanisms.

Acknowledgements

The authors are funded by grant DE-FG02-11ER16220 from the Division of Chemical Sciences, Geosciences, and Biosciences, Office of Basic Energy Sciences of the US Department of Energy with foundations support from grants DE-FG02-04ER15559 (for AK, GD) and Grant DE-FG02-91ER2002 (for AK and NF). A portion of DMK's salary is supported by the MSU AgBioResearch.

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