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Interaction between photosynthesis and respiration in illuminated leaves

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Abstract

Plants are sessile organisms that often receive excessive amounts of light energy. This excess energy can be exported from the chloroplasts and dissipated by the mitochondrial respiratory chain. The inner membrane of plant mitochondria possesses unique non-phosphorylating pathways, involving alternative oxidase and type II NAD(P)H dehydrogenases. There are accumulating amounts of evidence showing that these energy-wasteful pathways are up-regulated under excess light conditions, suggesting that they play key roles in efficient photosynthesis. Based on recent advances in our understanding about the metabolic interaction between chloroplasts and mitochondria, we discuss the importance of the respiratory chain for stabilizing the photosynthetic system.

Keywords: Alternative oxidase (AOX); Excess energy dissipation; Illuminated leaves; Interaction between chloroplast and mitochondria; NAD(P)H dehydrogenases (NDs); Non-phosphorylating pathway

1. Introduction

For the last two decades, metabolic interaction between photosynthesizing chloroplasts and oxidative-respiring mitochondria has been intensively studied (Noctor et al., 2007). In illuminated leaves, intracellular metabolism is dynamically modulated depending on environmental changes. Under such conditions, the function of chloroplasts and mitochondria is closely coordinated. Photosynthesis fixes atmospheric carbon dioxide and produces carbohydrates, part of which are catabolized into ATP and reductants by respiration in response to cellular energy

Abbreviations: 2-OG, 2-oxoglutarate; 3-PGA, 3-phosphoglyceric acid; AAC, ATP/ADP carrier; AAT, aspartate aminotransferase; AOX, alternative oxidase; Asp, aspartate; C, carbon; COX, cytochrome *c* oxidase; CP, cytochrome pathway; CS, citrate synthase; DCT, dicarboxylate transporter; DHAP, dihydroxyacetone phosphate; DTC, dicarboxylate–tricarboxylate carrier; FAD-GPDH, FAD-dependent glycerol-3-phosphate dehydrogenase; Fd, Ferredoxin; GDC, glycine decarboxylase complex; GGAT, glutamate glyoxylate aminotransferase; Glu, glutamate; Gly, glycine; GOGAT, glutamine:2-oxoglutarate aminotransferase; GP, glycerol-3-phosphate; GPDHc, cytosolic glycerol-3-phosphate dehydrogenase; GS, glutamine synthetase; H_2O_2, hydrogen peroxide; HNE, 4-hydroxy-2-nonenal; HPR, hydroxypyruvate reductase; IMS, inter membrane space; Mal, malate; MCF, mitochondrial inner membrane carrier family; N, nitrogen; NAD-G3PDH, phosphorylating NAD-dependent glyceraldehyde 3-phosphate dehydrogenase; NAD-IDH, NAD-dependent isocitrate dehydrogenase; NAD-MDH, NAD-dependent malate dehydrogenase; NAD-ME, NAD-malic enzyme; NADP-G3PDH, non-phosphorylating NADP-dependent glyceraldehyde 3-phosphate dehydrogenase; NADP-ICDH, NADP-dependent isocitrate dehydrogenase; NADP-MDH, NADP-dependent malate dehydrogenase; NDex, external type II NAD(P)H dehydrogenase; NDin, internal type II NAD(P)H dehydrogenases; NDs, type II NAD(P)H dehydrogenases; NiR, nitrite reductase; NPQ, non-photochemical quenching; NR, nitrate reductase; O_2^-, superoxide; OAA, oxaloacetate; OGDC, 2-oxoglutarate decarboxylase complex; OMT, 2-oxoglutarate/malate transporter; PDC, pyruvate dehydrogenase complex; PDK, pyruvate dehydrogenase kinase; PEP, phospho*enol*pyruvate; PEPCase, phospho*enol*pyruvate carboxylase; PGK, phosphoglycerate kinase; PGly, 2-phosphoglycolate; P_i, phosphate; PK, pyruvate kinase; PS, photosystem; PSI-CEF, photosystem I cyclic electron flow; Pyr, pyruvate; ROS, reactive oxygen species; Ser, serine; SHAM, salicylhydroxamic acid; SHMT, serine hydroxymethyl transferase; SPS, sucrose phosphate synthase; TP, triose phosphate; TPT, triose phosphate–phosphate transporter; Trx, thioredoxin; UCP, uncoupling protein.

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demand. Under light conditions, other metabolic pathways, such as photorespiration and nitrogen (N) assimilation, occur. These pathways take place partly in the chloroplasts and partly in the mitochondria. Thus, the interactions between chloroplasts and mitochondria are indispensable to carbon (C) and N assimilation, and ultimately beneficial for plant survival (Raghavendra and Padmasree, 2003).

The mitochondria in higher plants and green algae possess many unique components, which do not exist in mammalian mitochondria. For example, the mitochondrial matrix of plants contains a glycine decarboxylase complex (GDC), which is involved in the photorespiratory pathway. In addition, the respiratory electron transport chain in higher plants consists of not only the phosphorylating pathway (containing complex I, III, and IV), but also several non-phosphorylating pathways, such as those involving type II NAD(P)H dehydrogenases (NDs) and the cyanide-resistant alternative oxidase (AOX) (Plaxton and Podestá, 2006). The physiological significance of these energy-wasteful non-phosphorylating respiratory pathways is still not fully understood. Plants are immobile, and as such are critically different from animals. Therefore, plants develop many biochemical strategies to withstand long-term exposure to various environmental stresses. Evidence is accumulating showing that many components of these non-phosphorylating pathways are induced and/or up-regulated under stress conditions (Noctor et al., 2007), which suggests that they play an important role for plant acclimation. This induction has been observed in leaves under excess light intensity (Noguchi and Terashima, 2006; Yoshida et al., 2007), implying that the non-phosphorylating pathways can have an important function in the interaction between chloroplasts and mitochondria.

Under high light conditions, the respiratory chain is thought to dissipate excess reductants produced in the chloroplasts (Raghavendra and Padmasree, 2003). The non-phosphorylating pathways in the respiratory chain are considered to be efficient dissipation systems for these reductants, because the electron flow through these pathways is not limited by adenylate control. Thus, the non-phosphorylating pathways may function as a mechanism for plant photo-protection, but the components of this mechanism have not been characterized in detail. In this review, we will summarize recent advances on the export of reductants from the chloroplasts to the cytosol, and on the dissipation of these reductants via the mitochondrial non-phosphorylating pathways in illuminated leaves. In addition, some issues that need to be characterized in the future will be discussed. We will also briefly review a number of other metabolic interactions between chloroplasts and mitochondria in illuminated leaves, but more detailed information on this topic can be found in the following reviews (Atkin et al., 2000; Gardeström et al., 2002; Raghavendra and Padmasree, 2003; Nunes-Nesi et al., 2007; Noctor et al., 2007).

2. Export of excess reductants from chloroplasts

2.1. Dissipation systems for excess light energy in chloroplasts

Light intensity varies temporally and spatially in plant habitats. Leaves often receive amounts of light intensity much greater than the requirement of photosynthetic CO_2 fixation. In particular, under stress conditions, such as low temperature or drought, even low light intensity overflows photosynthetic demands. Excess light energy induces generation of reactive oxygen species (ROS), such as superoxide (O_2^-) and hydrogen peroxide (H_2O_2). Although ROS can act as signal molecules in the cell and function as environmental sensors, excess ROS imposes photo-oxidative damage to the photosynthetic apparatus. In the chloroplasts, ROS production is avoided by several systems dissipating excess light energy, such as thermal dissipation of light energy by the conformational changes in photosystem II (PSII), and by the xanthophyll cycle which is induced by PSI cyclic electron flow (PSI-CEF, Niyogi, 2000; Shikanai, 2007). The photosynthetic electron transport carrier protein, ferredoxin (Fd), can supply its reducing power to nitrite reductase (NiR) and/or glutamine:2-oxoglutarate aminotransferase (GOGAT) in the chloroplasts. The reducing power of NADPH, the final product of linear photosynthetic electron transport, can be exported from the chloroplasts to other cellular compartments. This system is also important for the maintenance of the redox balance in the chloroplasts (Scheibe et al., 2005).

2.2. Mechanisms for excess reductant export from chloroplasts

Several transporters localized on the chloroplast envelope have been identified (Fig. 1; Gardeström et al., 2002). It has been assumed that some of them are involved in the reductant-export system from the chloroplast, referred to as ''shuttle machinery''. This system is essential when reductants are in excess in the chloroplasts, because NADPH cannot pass through the chloroplast envelope directly. In this section, we focus on some transporters that are related to the shuttle machinery and on the importance of this system in plant photo-protection. However, in the available studies on the interaction between chloroplasts and mitochondria, approaches from the standpoint of transporters are few. More research is needed to identify transporters functioning in the shuttle machinery.

2.2.1. The malate–oxaloacetate shuttle

The malate–oxaloacetate (Mal–OAA) shuttle is considered to be an important system for reductant transport (Fig. 1). Similar to several Calvin cycle enzymes, NADP-dependent malate dehydrogenase (NADP-MDH) in the chloroplast stroma is activated by the light-dependent Fd-thioredoxin (Trx) system (Miginiac-Maslow et al.,

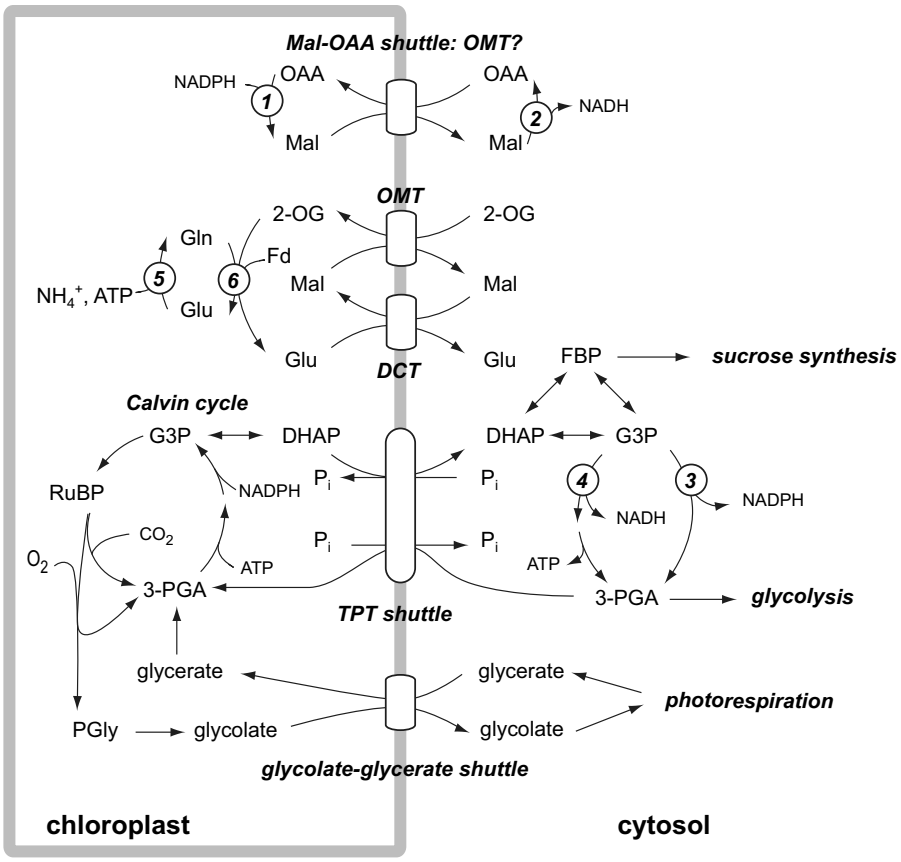

Fig. 1. Metabolic transport across the chloroplast envelope in illuminated leaves. The malate–oxaloacetate shuttle (Mal–OAA shuttle) is considered to function as an efficient exporter of excess reductants from the chloroplasts. NADP-dependent malate dehydrogenase (NADP-MDH, 1) in the chloroplast stroma is activated by light. Exported Mal can be converted to OAA by cytosolic NAD-dependent MDH (NAD-MDH, 2). The triose phosphate–phosphate transporter (TPT) exchanges TP (dihydroxyacetone phosphate, DHAP) with phosphate (P_i), facilitating optimal photosynthesis. TPT can also indirectly exchange TP with 3-phosphoglycerate (3-PGA). In this case, excess reductants can be exported from the chloroplasts. Exported TP is oxidized by non-phosphorylating NADP-dependent glyceraldehyde 3-phosphate dehydrogenase (NADP-G3PDH, 3) or by phosphorylating NAD-dependent glyceraldehyde 3-phosphate dehydrogenase (NAD-G3PDH, 4). 2-oxoglutarate/malate transporter (OMT), and dicarboxylate transporter (DCT) cooperatively import 2-oxoglutarate (2-OG) into chloroplasts, and export Glu into the cytosol. Imported 2-OG is used in the assimilation of NH_4^+ via glutamine synthetase (GS, 5) and glutamine:2-oxoglutarate aminotransferase (GOGAT, 6). The glycolate–glycerate transporter exchanges glycolate with glycerate in the photorespiratory pathway. FBP, fructose bisphosphate; G3P, glyceraldehyde 3-phosphate; Gln, glutamine; Glu, glutamate; PGly, 2- phosphoglycerate.

2000). The activated NADP-MDH produces Mal and $NADP^+$ from NADPH and OAA. Mal can be exported to the cytosol via Mal transporters on the chloroplast membrane. It has been demonstrated that the acceptor side of PSI was more oxidized in NADP-MDH over-expressing potato leaves than in wild type and NADP-MDH under-expressing ones (Backhausen et al., 1998). In potato leaves under high CO_2 conditions, NADPH is more efficiently consumed by the CO_2 assimilation process, thereby decreasing the NADP-MDH activation state and the reduced fraction of the acceptor side of PSI (Backhausen and Scheibe, 1999). These results support the possibility that NADP-MDH plays a key role in recycling NADPH, especially when its production is in excess of the requirement for the photosynthetic CO_2 assimilation (Scheibe et al., 2005).

Results presented by Hatch et al. (1984) indicated that a highly active Mal–OAA exchange exists in the chloroplasts. In *Arabidopsis*, the 2-oxoglutarate/malate transporter (OMT [DiT1]), which has a high affinity to Mal, OAA, and 2-oxoglutarate (2-OG), is a suitable candidate as a Mal–OAA exchanging transporter (Fig. 1, Taniguchi et al., 2002). In addition, OMT also can exchange Mal for 2-OG, and the dicarboxylate transporter (DCT [DiT2]) can subsequently re-import Mal by exporting glutamate (Glu). The Glu export is essential for efficient assimilation of NH_4^+ generated from photorespiration and NO_2^- reduction. The slow growth of OMT-deficient *Arabidopsis* can be recovered be growing the plants under high CO_2 conditions (Taniguchi et al., 2002). In addition, allocation of carbon to amino acid synthesis was impaired, and protein contents decreased, in OMT (DiT1)-antisense tobacco (Schneidereit et al., 2006). These reports suggest that OMT has a function in the transport of the carbon skeleton to N assimilation. However, it is still unclear whether OMT is truly involved in the Mal–OAA shuttle and functions as the exporter of reductants. Furthermore, although DCT (DiT2) can transport Mal and OAA, it is

likewise unknown if this function is important *in vivo*. The exported Mal is oxidized by NAD-dependent MDH (NAD-MDH) localized in the cytosol, peroxisomes, and mitochondria, which generates NADH. This NADH is subsequently consumed by the reduction of NO_3^-, photorespiration, and mitochondrial respiration (Scheibe et al., 2005).

A number of findings support the idea that reductant export in the form of Mal is important for achieving an optimal rate of photosynthesis. Firstly, Igamberdiev et al. (2001) showed that leaves of a GDC-deficient barley mutant have a lower capacity for photorespiration and chloroplast ATP/ADP and NADPH/NAD ratios were higher than those in wild type. The NADP-MDH activation state increased in this mutant, suggesting that reductants are exported from the chloroplasts via a Mal–OAA shuttle. Secondly, non-photochemical quenching (NPQ, thermal dissipation of excess light energy) under high light condition was not induced in the *Arabidopsis* pgr5 mutant, as a result of the lack of the Fd-dependent PSI-CEF pathway (Munekage et al., 2002). In this mutant, both the activity and activation state of NADP-MDH were higher when compared to wild-type plants (Yoshida et al., 2007). The results of these two mutants indicate that the Mal–OAA shuttle plays a role in the dissipation of excess reductants in the chloroplasts.

2.2.2. The triose phosphate–phosphate transporter

The triose phosphate–phosphate transporter (TPT) exchanges TP (e.g., dihydroxyacetone phosphate, DHAP) with phosphate (P_i). In illuminated leaves, TP is exported from the chloroplasts to the cytosol and used for sucrose synthesis (Fig. 1). In leaves of an *Arabidopsis* TPT-lacking mutant, starch accumulated in the chloroplasts, and both photosynthesis and growth decreased (Walters et al., 2004). TPT imports P_i into the chloroplast, and this is essential for optimal photo-phosphorylation in the chloroplasts of illuminated leaves (Sharkey and Vanderveer, 1989).

TPT can also indirectly exchange TP with 3-phosphoglyceric acid (3-PGA). In this case, excess reductants are exported from the chloroplasts in the form of TP (Gardeström et al., 2002). The exported TP can be oxidized by the cytosolic non-phosphorylating NADP-dependent glyceraldehyde 3-phosphate dehydrogenase (NADP-G3PDH), which produces 3-PGA and NADPH. Alternatively, 3-PGA, ATP and NADH can be produced from TP via the phosphorylating NAD-dependent glyceraldehyde 3-phosphate dehydrogenase (NAD-G3PDH) and phosphoglycerate kinase (PGK) in the cytosol (Gardeström et al., 2002; Plaxton and Podestá, 2006). However, NADP-G3PDH has a higher affinity for TP than NAD-G3PDH. Although NADP-G3PDH is inhibited by high NAD(P)H concentrations (Scagliarini et al., 1990), the cytosolic NAD(P) pool is more oxidized under light than dark conditions (Igamberdiev and Gardeström, 2003). Thus, excess reductants in the chloroplasts can be exported to the cytosol via TPT and are then metabolized mainly by the high affinity NADP-G3PDH. 3-PGA produced by G3PDH is partly imported into the chloroplasts via the TPT shuttle, the rest is catabolized in glycolysis.

Other transporters may play important roles in plant photo-protection through the photorespiratory metabolism (Fig. 1). Under atmospheric CO_2 concentrations, OMT and DCT cooperatively import 2-OG into the chloroplasts, and export Glu into the cytosol, allowing continued assimilation of NH_4^+ generated from photorespiration and NO_2^- reduction (see also above). The glycolate–glycerate transporter exchanges glycolate with glycerate in the photorespiratory pathway (Flügge and Heldt, 1991). These transporters do not directly export reductants from the chloroplasts, but both N reduction and photorespiration can be involved in the dissipation of excess light energy.

Many parts of the shuttle system for the export of excess reductants from the chloroplasts are still unknown in detail, but, in illuminated leaves, this export of excess reductants may well be indispensable for maintenance of the redox balance in the chloroplasts.

3. The reductants in mitochondria in illuminated leaves

In illuminated leaves, light-dependent processes, including photosynthesis, alter the whole cellular metabolism and cause large changes in the mitochondrial redox state. In this section, we discuss the mitochondrial redox state and shuttle-mechanisms in illuminated leaves.

3.1. Substrates and reductants imported from the cytosol to the mitochondria

In glycolysis, phospho*enol*pyruvate (PEP) is converted to pyruvate (Pyr) via pyruvate kinase (PK, Fig. 2). Pyr is imported into the mitochondria as a respiratory substrate. Also, PEP carboxylase (PEPCase) converts PEP to OAA, which is subsequently catalyzed into Mal by the cytosolic NAD-MDH. Both OAA and Mal can be imported into the mitochondria as substrates (complemental pathway). When intermediates in the TCA cycle are exported and consumed in several other pathways (e.g., amino acid biosynthesis), OAA or Mal from the complemental pathway is required for the progression through part of the TCA cycle (Atkin et al., 2000; Gardeström et al., 2002). In illuminated leaves, PEPCase is thought to be activated rather than PK. This is because PEPCase is activated by glycine (Gly), an intermediate of photorespiration, as well as by light (Jeanneau et al., 2002). In contrast, PK is inhibited by NH_4^+, a product of photorespiratory GDC, and by a high ATP/ADP ratio. Leaf growth did not decrease under normal conditions in PK-deficient tobacco (Knowles et al., 1998), whereas leaf respiration rate increased in tobacco in which PEPCase was over-expressed (Hausler et al., 1999). These studies seem

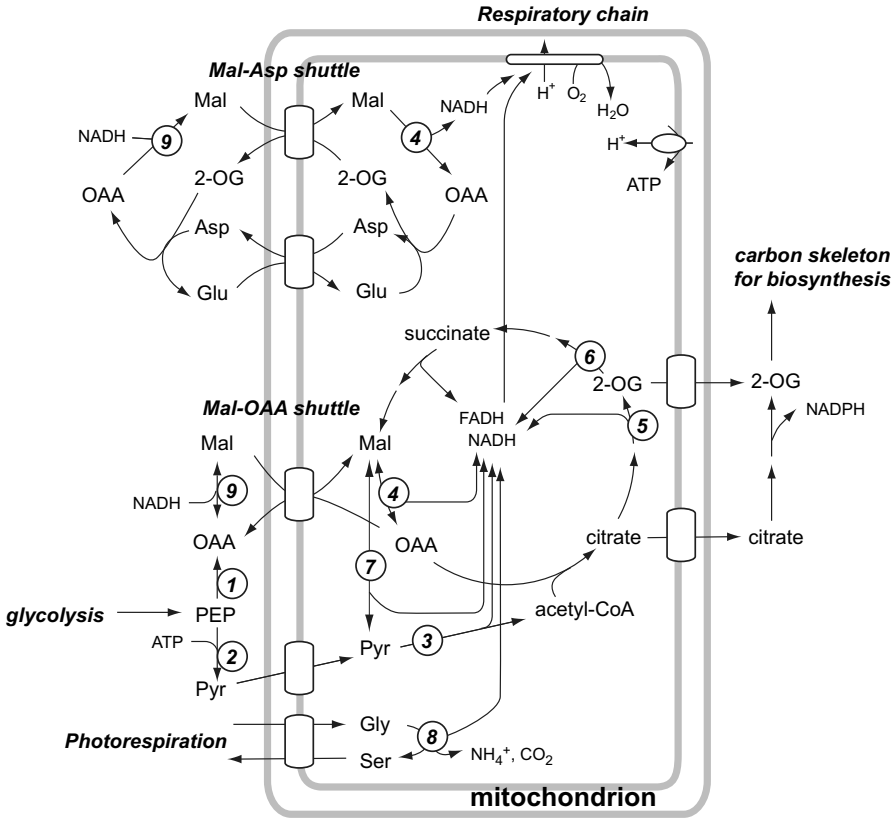

Fig. 2. Respiratory systems and metabolite transporters in the mitochondria of higher plants. In illuminated leaves, phospho*enol*pyruvate carboxylase (PEPCase, 1) is more active than pyruvate kinase (PK, 2). In mitochondria, NADH is produced from the pyruvate dehydrogenase complex (PDC, 3; NAD-MDH, 4), NAD-isocitrate dehydrogenase (NAD-IDH, 5), 2-oxoglutarate decarboxylase (OGDC, 6), and NAD-malic enzyme (NAD-ME, 7). Also, the glycine decarboxylase complex and serine hydroxymethyl transferase (GDC/SHMT, 8) supply NADH. TCA cycle intermediates are exported from the mitochondria for supply of carbon skeletons, and in this situation a partial TCA cycle requires the complemental pathway for the supply of OAA. The Mal–OAA shuttle can transport reductants between the cytosol and the mitochondria, and the Mal–aspartate (Asp) shuttle also functions as a reductant transporter. Glu, glutamate; Gly, glycine; Pyr, pyruvate; Ser, serine.

to support the idea that PEPCase activity is more significant compared to PK in illuminated leaves.

Transport of respiratory substrates into the mitochondria is regulated at the level of the mitochondrial inner membrane (Fig. 2). Here, dicarboxylate–tricarboxylate carrier (DTC) functions as a general carrier for transport of respiratory substrates, including OAA and Mal. DTC belongs to the mitochondrial inner membrane carrier family (MCF). The carriers belonging to MCF are proteins with a mass less than 30 kDa and with six *trans*-membrane domains. The functions of several carriers of MCF have been identified, such as the uncoupling protein (UCP), the ATP/ADP carrier (AAC), and the phosphate carrier (Picault et al., 2004).

In plant cells, the following shuttle-mechanisms can transfer excess reductants between the cytosol and the mitochondria: the Mal–OAA shuttle, the Mal–aspartate (Asp) shuttle (Dry et al., 1987), the glycerol-3-phosphate (GP)–DHAP shuttle (Shen et al., 2006). In the first two shuttles (Fig. 2), Mal is exchanged for a counter metabolite across the mitochondrial membrane, which can maintain an optimal redox poise in the cytosol and mitochondria, especially during the light period. It is still unclear what is the predominant direction of reductant transport in illuminated leaves. In the mitochondria of illuminated leaves, a large amount of NADH is produced by GDC, and this should be exported from the mitochondria to prevent over-reduction (see Section 3.3). On the other hand, an *in vitro* study demonstrated that mitochondria of etiolated durum wheat shoots have the potential to exchange Mal for a counter metabolite (Pastore et al., 2003). This Mal exchange across the mitochondrial membrane may be required for a series of reductant transports from the chloroplasts and dissipation by the respiratory chain under excess light (Yoshida et al., 2007), although the reductants in the cytosol can also be oxidized by the respiratory chain via external ND (NDex) and/or the GP–DHAP shuttle. In the GP–DHAP shuttle, the cytosolic GP dehydrogenase (GPDHc) and FAD-dependent GP dehydrogenase (FAD-GPDH) can transfer reductants to ubiquinone (UQ, Fig. 3; Shen et al., 2006). Under light conditions, the Mal import to the mitochondria may also be important for the redox-regulation of some respiratory enzymes via the NADP-Trx system (Balmer et al., 2004). This is because NADPH can be produced from Mal via NAD-malic enzyme (ME, Agius et al., 1998).

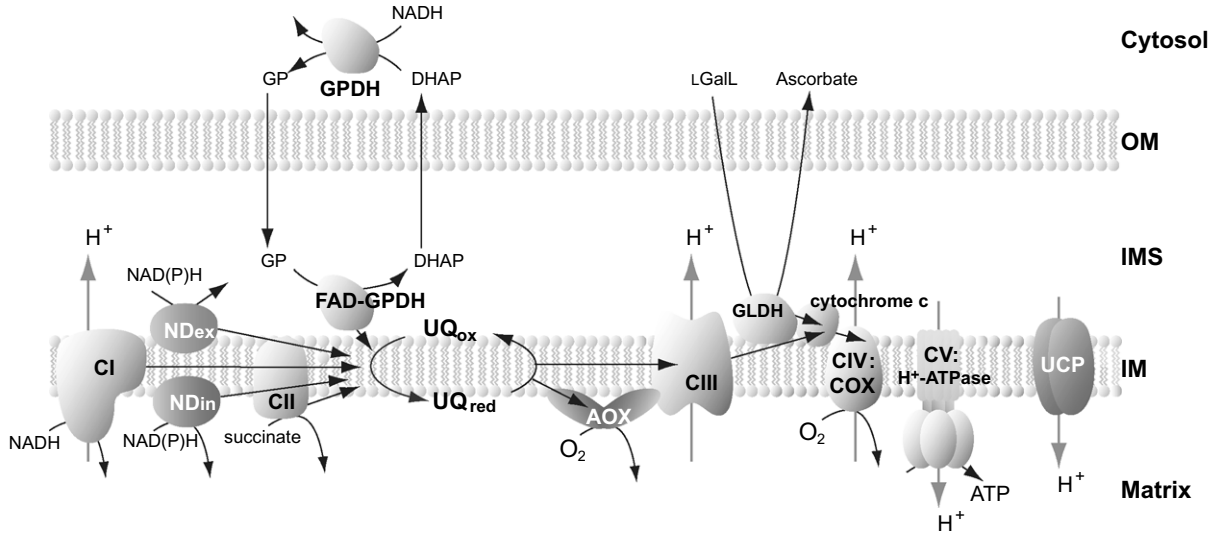

Fig. 3. The respiratory chain in higher plant mitochondria. Localized on the inner membrane are not only the classical phosphorylating pathways (complexes I, III, IV) but also the unique non-phosphorylating pathways, such as external type II dehydrogenase (NDex), internal ND (NDin) and alternative oxidase (AOX), and active uncoupling protein (UCP). L-Galactono-1,4-lactone dehydrogenase (GLDH) catalyzes the final steps in ascorbate synthesis. GLDH transfers electrons to cytochrome *c*. In the glycerol-3-phosphate-dihydroxyacetone phosphate (GP–DHAP) shuttle, cytosolic GP dehydrogenase (GPDHc) and FAD-dependent GP dehydrogenase (FAD-GPDH) transfer reductants to ubiquinone (UQ). COX, cytochrome *c* oxidase.

3.2. The TCA cycle

In the TCA cycle, NADH is produced via several reactions, involving the pyruvate dehydrogenase complex (PDC), NAD-ME, NAD-dependent isocitrate dehydrogenase (NAD-IDH), the 2-oxoglutarete decarboxylase complex (OGDC), and NAD-MDH (Fig. 2). NADPH is supplied by NAD-ME (Agius et al., 1998) or NADP-dependent isocitrate dehydrogenase (NADP-ICDH, Gálvez et al., 1999). The PDC supplies acetyl-CoA, NADH, and CO_2 from Pyr, CoA, NAD^+, and is regulated by allosteric control and phosphorylation/dephosphorylation. NH_4^+ and ATP activate pyruvate dehydrogenase kinase (PDK), which inactivates PDC. In illuminated leaves, photorespiratory GDC produces NH_4^+. Therefore, PDC in illuminated leaves is considered to be inactive (Atkin et al., 2000; Tovar-Méndez et al., 2003). Also, the mitochondrial NAD(P)H/NAD(P) ratio increases in illuminated leaves due to photorespiration (Igamberdiev and Gardeström, 2003). The high NAD(P)H/NAD(P) ratio inhibits PDC and the other matrix dehydrogenases, such as NAD-IDH.

However, PDC is not thought to be completely inactive in the light. Feeding of $^{13}C_1$-pyruvate or $^{13}C_2$-pyruvate into a leaf can distinguish between CO_2 efflux from PDC and other TCA-cycle enzymes. The results of this experiment suggested that in bean leaves under illuminated conditions, the whole TCA-cycle activity is inhibited by 95%, but PDC activity is inhibited only by 27% (Tcherkez et al., 2005). Studies of gas exchange also indicate that leaf respiratory CO_2 efflux in light is slower than in the dark, but is not completely suppressed (Atkin et al., 2000). Not only PDC, but also the other enzymes of the TCA cycle, at least those involved in citrate synthesis, function in the light, because citrate or 2-OG, intermediates of the TCA cycle, are used as carbon skeleton for N assimilation and other biosynthesis processes (Fig. 2). In higher plants, citrate synthase (CS) is under redox control (Stevens et al., 1997), probably via the NADP-dependent Trx system. The NADP-Trx system regulates many mitochondrial enzymes, including several TCA-cycle enzymes (Balmer et al., 2004). These two reports also supported that a partial TCA cycle can supply carbon skeletons for other metabolic pathways (e.g., N assimilation) in illuminated leaves. This partial TCA cycle in the light requires the complemental pathway for supply of OAA. OAA is derived from NAD-MDH or imported from the cytosol via the Mal (or citrate)–OAA exchange (Gardeström et al., 2002).

3.3. Photorespiration

Under ambient CO_2 concentrations, Rubisco oxygenation produces 2-phosphoglycolate (PGly), which can be converted to glycerate in successive reactions of photorespiration in the chloroplast, peroxisome, and mitochondria. It has been shown that photorespiration is required for optimal photosynthesis (Keys and Leegood, 2002). In illuminated leaf mitochondria, GDC is strongly induced, and GDC and serine hydroxymethyl transferase (SHMT) convert Gly and NAD^+ to serine (Ser), NADH, CO_2 and NH_4^+. Gly is imported from the peroxisome, and Ser and NH_4^+ are exported from the mitochondria (Oliver, 1994). Since Rubisco oxygenation rate is assumed to be 20–35% of the CO_2 fixation rate (Atkin et al., 2000), a large amount of NADH is produced by photorespiration in illuminated leaves. Although hydroxypyruvate reductase

(HPR) in the peroxisome needs the same amounts of NADH as produced by GDC, only 30–50% of NADH is exported from the mitochondria to the peroxisome (Krömer et al., 1992). The rest of NADH required by HPR must be transported from the chloroplasts. Photorespiratory NADH in the mitochondria can be exported in the form of Mal from the mitochondria via the Mal–OAA shuttle or the Mal–Asp shuttle. The rest of photorespiratory NADH is thought to be oxidized by the respiratory chain. Indeed, results using a GDC inhibitor and GDC mutant indicated that a large amount of photorespiratory NADH is consumed by the respiratory chain (Wigge et al., 1993; Igamberdiev et al., 2001). In particular, internal ND (NDin) and AOX are important for the oxidation of photorespiratory NADH (Igamberdiev et al., 1997). Since GDC is inhibited by low concentrations of NADH ($K_i = 15$ μM, Oliver, 1994), it is important that NADH is rapidly recycled via export from the mitochondria or via oxidation by non-phosphorylating pathways.

In the mitochondria of illuminated leaves, NADH is generated from GDC and part of the TCA cycle. A part of this NADH is subsequently exported to the cytosol, and the rest is oxidized by the respiratory chain. The reductants in the cytosol are not transported into the mitochondria, but the redox state of the cytosol may influence the redox state of the mitochondrial matrix and influence transport activities from the mitochondria to the cytosol.

4. Components of the respiratory chain, and effects of light

On the mitochondrial inner membrane of higher plants, not only the classical phosphorylating pathways but also several non-phosphorylating pathways, involving NDex, NDin, and AOX, and active UCP exist (Fig. 3; Plaxton and Podestá, 2006). In this section, we briefly introduce these pathways, and summarize and discuss various studies in relation to the possibility that the respiratory chain dissipates excess reductants in the chloroplast under excess light. The regulation and expression of each component of the respiratory chain have been reviewed in detail by Millenaar and Lambers (2003), Finnegan et al. (2004), Rasmusson et al. (2004), Vercesi et al. (2006), Møller (2007), and Rasmusson et al. (in press).

The respiratory chain is also linked with various other metabolic pathways in the mitochondria, such as the ascorbate synthesis (Bartoli et al., 2000) and the GP–DHAP shuttle (Shen et al., 2006). Detail information on these pathways can be found in Sweetlove et al. (2007) and Nunes-Nesi et al. (2007).

4.1. The respiratory chain in higher plants, and its regulation by light

4.1.1. The phosphorylating pathway: complexes I, III, and IV

Complexes I, III, and IV export H^+ from the mitochondrial matrix to the inter membrane space (IMS), and are coupled with ATP synthesis (Fig. 3). These complexes and H^+-ATPase (complex V) are assumed to form a super complex (OXPHOS system, Dudkina et al., 2006). The advantage of such a system would be that it sustains a smooth electron flow and efficient energy transduction.

Gene expression and protein regulation of components of the phosphorylating pathway do not always depend on light (Rasmusson and Escobar, 2007). In leaves of *Alocasia odora*, a shade species, the respiration rate quickly acclimated to changes in light intensity, and the maximal activity of NAD-IDH also acclimated, whereas cytochrome *c* oxidase (COX, complex IV) did not show a clear acclimation response (Noguchi et al., 2001). The protein amount of two subunits of complex I (a 76 kDa subunit and NAD9) in potato leaves, and the gene expression of a 76 kDa subunit in *Arabidopsis* leaves were both independent of light (Svensson and Rasmusson, 2001; Escobar et al., 2004). The phosphorylating pathway is regulated by adenylate levels, and consists of a rather large number of subunits. Therefore, the pathway cannot respond to rapid changes of the environment. However, the following non-phosphorylating pathways can easily and inexpensively respond to environmental fluctuations. The rapid responses to changes in the light environment would facilitate a change of the demands for TCA-cycle intermediates, and dissipate excess reductants.

4.1.2. AOX

AOX expressed in higher plants, some algae and fungi, is a terminal oxidase of 30–36 kDa, which oxidizes ubiquinol and produces water (Fig. 3). AOX does not affect H^+ transport, and thereby is not linked to ATP production. AOX is activated by the reduction of a disulfide bridge linking the AOX homodimer and by alpha-keto acids, e.g., pyruvate (Finnegan et al., 2004). The reduction of AOX is supposed to be regulated by the NADP-Trx system (Gelhaye et al., 2004). Activated AOX competes with complex III of the cytochrome pathway (CP) for electrons (Hoefnagel et al., 1995). This energy-wasteful pathway is assumed to consume excess reductants and avoid ROS production, especially under stressful conditions (Maxwell et al., 1999; Umbach et al., 2005).

In *Arabidopsis*, five genes encode AOX (*AtAOX1a-1d*, and *AtAOX2*), and *AtAOX1a* is mainly expressed in green leaves (Clifton et al., 2006). Leaf AOX is induced by light (AOX protein in potato leaves, Svensson and Rasmusson, 2001; *AOX2* in soybean leaves, Finnegan et al., 1997). During greening of soybean cotyledons, the electron partitioning shifted from CP to AOX (Ribas-Carbo et al., 2000). It is unknown whether light directly regulates *in vivo* AOX activity, but the redox state of AOX in *Alocasia* leaves changed depending on growth light intensity (Noguchi et al., 2005). Since the NADPH/NADP ratio in the mitochondrial matrix of leaves in the light was higher than that in the dark (Igamberdiev and Gardeström, 2003), AOX may be activated by the NADP-Trx system in a light-dependent manner. However, a clear correlation between

the redox state of AOX and the *in vivo* electron flux to AOX has not always been observed (Millenaar et al., 2001; Vidal et al., 2007). A detailed analysis on the relationship between AOX activation state and electron flow to AOX is required.

4.1.3. Type II NAD(P)H dehydrogenases

Type II NAD(P)H dehydrogenases (NDs) do not transport H^+ and are characterized by insensitivity to rotenone, an inhibitor of complex I. NDs exist on the inner membrane facing the IMS (NDex) or the mitochondrial matrix (NDin), bypassing complex I (Fig. 3). In *Arabidopsis*, NDex proteins are encoded by *AtNDB1-4* and NDin proteins are encoded by *AtNDA1*, *AtNDA2*, and *AtNDC1* (Rasmusson et al., 2004, in press; Elhafez et al., 2006). Both NDex and NDin showed higher values of K_m for NADH than complex I (Rasmusson et al., 2004).

In leaf mitochondria, a high activity of rotenone-insensitive respiration was observed (Igamberdiev et al., 1997). NDA1, one of the NDin proteins, is assumed to account for this activity (Rasmusson and Escobar, 2007). Some studies reported that NDin genes are light-induced (*NDA1*, *NDC1* in *Arabidopsis*, Escobar et al., 2004; Elhafez et al., 2006; *NDA1* in potato, Svensson and Rasmusson, 2001). Since glycine oxidation of pea leaf mitochondria was rotenone-insensitive, NDin is thought to oxidize NADH produced from the photorespiratory GDC (Gardeström et al., 2002). However, NDA1-deficient *Arabidopsis* did not show any phenotype (Moore et al., 2003). Microarray data revealed that *AtNDB2*, NDex gene, and *AtAOX1a* were concomitantly expressed (Clifton et al., 2006), allowing the cytosolic NADH to be oxidized, perfectly independent of adenylate control. Instead of dissipation by NDex, the cytosolic NADH could also be consumed by nitrate reductase (NR). In *Arabidopsis* seedlings transferred from a NO_3^- medium to a NH_4^+ medium, NDex genes, *AtNDB2* and *AtNDB4*, were induced, and NDex activity was also increased (Escobar et al., 2006).

4.1.4. UCP

UCP belongs to MCF, similar to DTC and AAC (Picault et al., 2004), and in the presence of free fatty acids, facilitates the re-entry of H^+ into the mitochondrial matrix, bypassing ATP synthesis (Fig. 3). UCP is also activated by ROS and inhibited by purine nucleotides, such as GTP, GDP, ATP (Hourton-Cabassa et al., 2004; Vercesi et al., 2006). In *Arabidopsis*, six genes are reported to encode UCPs (Vercesi et al., 2006), but, except for UCP1, their roles are still ambiguous. UCP is generally induced by stress conditions, but *AtUCP1* and *AtUCP2* did not show light-induction (Escobar et al., 2004). The role of UCP in illuminated leaves is still unclear, partly as a result of the absence of specific inhibitors. However, there is some data that may suggest a function for UCP. For example, in mitochondria of UCP-over-expressed potato tubers, the TCA-cycle activity from Pyr to citrate was increased (Smith et al., 2004). Also, it was found that, in leaves of *UCP1*-deficient *Arabidopsis*, the rate of photorespiratory flow from Gly to Ser and the rate of photosynthesis were suppressed (Sweetlove et al., 2006). These reports suggest that UCP allows NADH to be consumed more efficiently in the mitochondrial matrix, leading to optimal rates of respiration and photorespiration.

4.2. Dissipation of excess chloroplast reductants by respiration

The studies summarized in the above section suggest that the non-phosphorylating pathways can efficiently respond to changes in the light environment. Direct interaction between photosynthesis and the respiratory chain has been observed in studies using specific inhibitors or mutants (Krömer, 1995; Gardeström et al., 2002; Raghavendra and Padmasree, 2003; Yoshida et al., 2006). Antimycin A, an inhibitor of complex III, and salicylhydroxamic acid (SHAM), an inhibitor of AOX, decreased the rate of photosynthesis in pea mesophyll protoplast. In the presence of antimycin A, the TP/3-PGA ratio was increased rather than the Mal/OAA ratio, whereas the Mal/OAA ratio was strongly increased in the presence of SHAM (Padmasree and Raghavendra, 1999). These results suggest that the CP maintains the export of TP, and the supply of ATP to the cytosolic sucrose synthesis (see Section 5.1.), whereas AOX maintains the oxidation of Mal in the light. The *Nicotiana sylvestris* cytoplasmic male-sterile CMSII mutant misses a functional complex I in the respiratory chain. This mutant has been intensively examined with respect to the relationship between respiration and other metabolisms (Noctor et al., 2007). CMSII mutants show a higher rate of respiration and a lower rate of photosynthesis and growth (Sabar et al., 2000; Dutilleul et al., 2003). In the CMSII mutant, NAD^+, NADH and NADPH accumulated, and the C/N ratio decreased (Dutilleul et al., 2005). The capacities of ND and AOX enzymes in this mutant were increased (Sabar et al., 2000). A redox unbalance as a result of a complex I deficit may have stimulated the expression of components of non-phosphorylating pathways, and this would partially compensate for the impaired activity of complex I (Noctor et al., 2007).

The above results suggest that NAD(P)H oxidation by the respiratory chain may regulate cellular redox balance and maintain optimal photosynthesis in illuminated leaves. In order to examine this possibility, we set up a system, in which photosynthetic O_2 evolution rate, chlorophyll fluorescence, and PSI redox state were concomitantly measured using an intact leaf. Using this system, photosynthetic electron transport of broad bean leaves were analyzed in the presence or absence of respiratory inhibitors. In the presence of CP inhibitors, photosynthetic rates were decreased only at high light conditions, whereas in the presence of an AOX inhibitor, photosynthetic rate and PSII operating efficiency were decreased even under low light. Also, the relationship of operating efficiency between PSI and PSII was unbalanced in the presence of an AOX inhibitor

(Yoshida et al., 2006). In leaves of the *Arabidopsis* PSI-CEF mutant, excess reductants accumulated in the chloroplast stroma, and NADP-MDH in the chloroplasts was activated under high light (Yoshida et al., 2007). In this situation, there were high NAD-MDH and AOX capacities, which increased after transfer to high light, whereas maximal activities of TCA-cycle enzymes and COX did not differ compared to the wild type (Yoshida et al., 2007). In drought-stressed wheat leaves, SHAM decreased the PSII operating efficiency, even under low light conditions (Bartoli et al., 2005).

These studies indicate that energy dissipation via AOX may control the redox balance in the chloroplasts and optimize the rate of photosynthesis, rather than the phosphorylating pathway. The excess reductants accumulated in the chloroplasts may be exported to the cytosol via the Mal/OAA shuttle, in accordance with the results of Padmasree and Raghavendra (1999). Fig. 4a shows this possibility. On the other hand, Sweetlove et al. (2006) suggested that UCP allows NADH to be efficiently consumed in the mitochondrial matrix, leading to optimal photorespiration. It is still unclear what the significance is of the redundancy between these two energy-wasteful pathways (AOX and UCP). However, a few studies partially addressed this question. Potato UCP was activated by 4-hydroxy-2-nonenal (HNE), which is derived from mitochondrial ROS (Smith et al., 2004), whereas *Arabidopsis* AOX was inhibited by HNE (Winger et al., 2005). These results suggest that plants can induce UCP or AOX differentially, depending on the severity of the stress condition.

4.3. Oxidative stress regulates respiratory gene expression

AOX is preferentially induced under excess light, when reductants accumulate in the chloroplasts (Yoshida et al., 2007). In this section we examine what signals can influence this preferential induction. Excess reductants in the chloroplasts induce ROS production, which can subsequently affect nuclear-encoded gene expression (Beck, 2005). However, there are no reports showing that plastid signals, such as ROS, redox state or intermediates of tetrapyrrole synthesis, can induce the expression of nuclear-encoded genes involved in respiration. On the other hand, studies using cell cultures indicate that signals from the mitochondria can induce nuclear-encoded respiratory gene expression (mitochondrial retrograde regulation, MRR, Møller, 2001; Rhoads and Subbaiah, 2007). The signal molecules are suggested to be O_2^- produced at complex I or III,

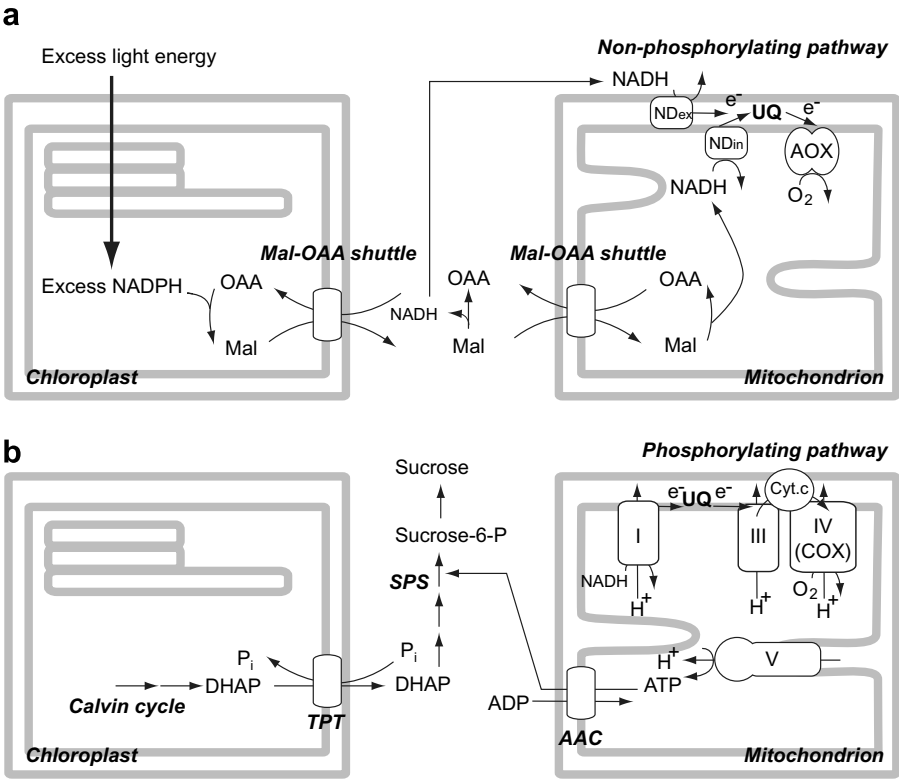

Fig. 4. Possible interactions between photosynthesis and respiration in illuminated leaves. (a) Dissipation of excess light energy by the non-phosphorylating respiratory chain. Under high light conditions, excess reductant accumulates in chloroplasts, and can be exported from the chloroplasts to the cytosol via the Mal–OAA shuttle. Excess reductants in the cytosol are oxidized by the external type II NAD(P)H dehydrogenase (NDex). In addition, these reductants can be imported into the mitochondria via the Mal–OAA shuttle, and dissipated by non-phosphorylating pathways, such as those involving internal type II NAD(P)H dehydrogenese (NDin) and alternative oxidase (AOX). (b) Respiratory ATP supply for cytosolic sucrose synthesis. Respiratory ATP is exported to the cytosol via the ATP/ADP carrier (AAC), and supplied to the sucrose phosphate synthase (SPS) reaction. TPT, triose phosphate–phosphate translocator.

and H_2O_2 produced from O_2^- by the mitochondrial superoxide dismutase. In addition to ROS, some TCA cycle intermediates (Mal, citrate and 2-OG) are thought to affect AOX gene expression in tobacco suspension cell cultures (Gray et al., 2004). In *Chlamydomonas*, a nuclear-encoded photosynthetic gene, *psaE*, was induced in response to the activation of CP (Matsuo and Obokata, 2006). This gene also responds to changes in photosynthetic electron transport (Matsuo and Obokata, 2004). However, it is still unclear how the interaction between photosynthetic electron transport and the respiratory chain occurs in illuminated leaves, which signals induce the genes of the non-phosphorylating pathways, and whether signals from photo-receptors (Rasmusson and Escobar, 2007) are independent of the retrograde signals.

5. Other mechanisms of interaction between photosynthesis and respiration

In the previous sections, we summarized the functions of the respiratory chain as the energy-dissipation system for excess reductants that accumulate in the chloroplasts. In this section, we briefly present other interactions between photosynthesis and respiration.

5.1. Respiratory ATP supply for cytosolic sucrose synthesis

Under high light and high CO_2 conditions, optimal photosynthesis requires a continuous flow of metabolites through the Calvin cycle and cytosolic sucrose synthesis. Several studies indicated that respiratory ATP is consumed by sucrose synthesis, leading to an optimal rate of photosynthesis. For example, in barley protoplasts, a low concentration of oligomycin, which inhibits only respiratory phosphorylation, decreases the rate of photosynthesis, the ATP/ADP ratio, and the TP/3-PGA ratio (Krömer et al., 1988, 1993). Activities and activation state of sucrose phosphate synthase (SPS) were also found to be decreased (Krömer et al., 1993). Rotenone, a complex I inhibitor, decreased photosynthesis and the cytosolic ATP/ADP ratio in barley protoplasts (Igamberdiev et al., 1998). In leaves of a starchless tobacco mutant, the rate of photosynthesis was inhibited by oligomycin, but oligomycin had no effect in wild-type plants (Hanson, 1992). These results indicate that the respiratory ATP is exported to the cytosol, and supplied to the SPS reaction, leading to optimal photosynthesis (Fig. 4b; Krömer, 1995; Gardeström et al., 2002).

However, in leaves of aconitase-deficient tomato, *Aco-1* mutant, the ATP/ADP ratio was lower, but the sucrose synthesis activity was higher than wild type (Carrari et al., 2003). In SPS-over-expressing tobacco, photosynthetic rate was increased only in the older leaves, not in young ones (Baxter et al., 2003). According to the C_3 photosynthesis model, the photosynthetic rate is limited by sucrose synthesis rate only under high light and high CO_2 concentration. Thus, it is still ambiguous if an optimal rate of photosynthesis requires the supply of respiratory ATP to cytosolic sucrose synthesis.

5.2. Supply of carbon skeleton from respiration to NH_4^+ assimilation

In illuminated leaves, glutamine synthetase (GS)/GOGAT localized in the chloroplasts produces Glu from NH_4^+ and 2-OG (Fig. 1). Glu is converted to other amino acids (Gardeström et al., 2002). 2-OG, which functions as a carbon skeleton for NH_4^+ assimilation is thought to originate from the following pathways: (1) 2-OG that is produced as an intermediate of TCA cycle, (2) 2-OG that is produced from citrate, derived from the TCA cycle, by cytosolic aconitase and NADP-ICDH, (3) 2-OG that is a product from cytosolic aspartate aminotransferase (AAT), and (4) 2-OG that is a product from glutamate glyoxylate aminotransferase (GGAT) in the peroxisome. In the first two pathways, a partial TCA cycle is required (Fig. 1; see Section 3.2). It is unclear whether the first two pathways are redundant, but they support *de novo* NH_4^+ assimilation. On the other hand, the last two pathways are also important in mature leaves, because NH_4^+ assimilation is needed for N recycling. In NADP-ICDH antisense potato, growth and photosynthesis were similar to that in control plants (Kruse et al., 1998). In addition, NAD-IDH-deficient *Arabidopsis* showed similar growth rates and no difference in amounts of sucrose and amino acids compared to wild-type plants (Lemaitre et al., 2007). On the other hand, both AAT-deficient *Arabidopsis* (Schultz et al., 1998) and GGAT-deficient *Arabidopsis* (Igarashi et al., 2003) showed severely reduced growth rates. Thus, a partial TCA cycle in illuminated mature leaves may also supply carbon skeletons to other biosynthetic processes, including NH_4^+ assimilation.

6. Summary

The metabolic interactions between chloroplasts and mitochondria have been closely examined in the last two decades. Among these interactions, the dissipation system of excess reductants is important in illuminated leaves. Under excess light, reductants are exported from the chloroplasts to the cytosol, probably via the NADP-MDH and Mal–OAA shuttle, and the mitochondrial non-phosphorylating pathways may facilitate the dissipation of these excess reductants in the cell. Further investigations are needed to examine the expression, regulation, and *in vivo* activity of these enzymes and transporters in detail. In addition, the signal transduction pathways responsible for the induction of the non-phosphorylating respiratory pathways need to be further clarified. Leaf mitochondria are often located adjacent to chloroplasts, and these organelles show dynamic movements in plant cells (Logan, 2006). Further study is needed for understanding the relationship between such dynamic movements and metabolic interactions.

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