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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.,