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Review

Discovering the role of mitochondria in the iron deficiency-induced metabolic responses of plants

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ABSTRACT

In plants, iron (Fe) deficiency-induced chlorosis is a major problem, affecting both yield and quality of crops. Plants have evolved multifaceted strategies, such as reductase activity, proton extrusion, and specialised storage proteins, to mobilise Fe from the environment and distribute it within the plant. Because of its fundamental role in plant productivity, several issues concerning Fe homeostasis in plants are currently intensively studied. The activation of Fe uptake reactions requires an overall adaptation of the primary metabolism because these activities need the constant supply of energetic substrates (i.e., NADPH and ATP). Several studies concerning the metabolism of Fe-deficient plants have been conducted, but research focused on mitochondrial implications in adaptive responses to nutritional stress has only begun in recent years. Mitochondria are the energetic centre of the root cell, and they are strongly affected by Fe deficiency. Nevertheless, they display a high level of functional flexibility, which allows them to maintain the viability of the cell. Mitochondria represent a crucial target of studies on plant homeostasis, and it might be of interest to concentrate future research on understanding how mitochondria orchestrate the reprogramming of root cell metabolism under Fe deficiency. In this review, I summarise what it is known about the effect of Fe deficiency on mitochondrial metabolism and morphology. Moreover, I present a detailed view of the possible roles of mitochondria in the development of plant responses to Fe deficiency, integrating old findings with new and discussing new hypotheses for future investigations.

Contents

Introduction

Iron (Fe) is an essential element for all living organisms because it is a cofactor for fundamental biochemical activities, such as energy metabolism, oxygen transport and DNA synthesis. Because of its redox reactivity [Fe shuttles between the reduced ferrous (Fe^{2+}) and the oxidised ferric (Fe^{3+}) forms], which allows it to associate with proteins and bind to oxygen, Fe transfers electrons and mediates catalytic reactions (Aisen et al., 2001). Fe is also potentially toxic because it can catalyse the propagation of reactive oxygen species (ROS) and the generation of highly reactive radicals (such as the hydroxyl radical) through Fenton chemistry (Koppenol, 1993). The ensuing oxidative stress is associated with damage to cellular macromolecules, tissue injury and

Abbreviations: mETC, mitochondrial electron transport chain; ND_{in}, internal type II NADPH dehydrogenases; ND_{ex}, external type II NADPH dehydrogenases.

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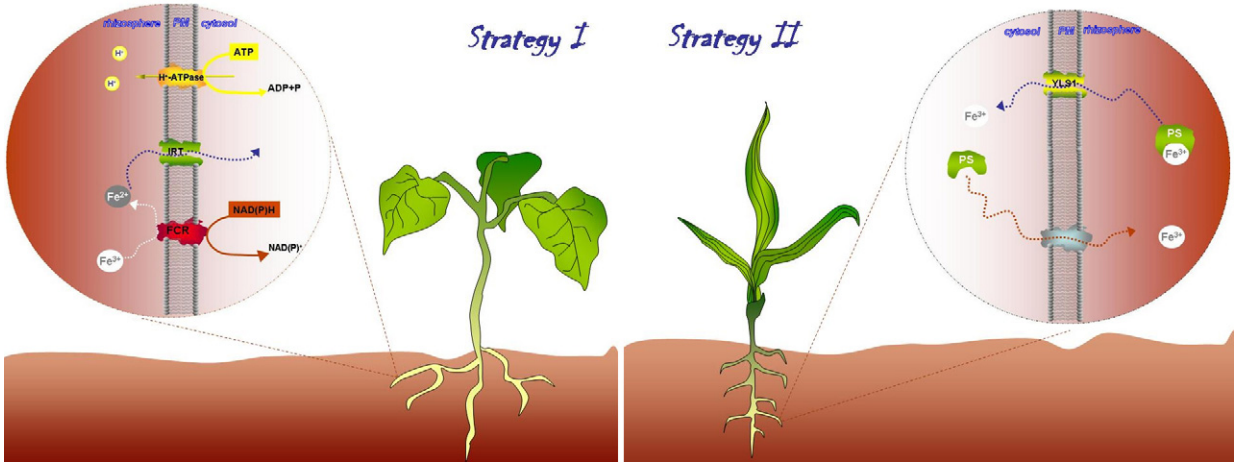

Fig. 1. Schematic representation of the mechanisms of iron uptake in plant roots. Left panel: Dicotyledonous plants induce the so-called Strategy I mechanism, characterised by the induction of the FCR, IRT1 and H^+-ATPase at the plasma membrane level. Right panel: monocotyledonous plants induce the so-called Strategy II mechanism, characterised by the production and the extrusion of the PS in the rhizosphere, and the uptake of the Fe^{3+}–PS complexes by the plants. *Abbreviations*: FC-R, ferric chelate-reductase; IRT1, iron-regulated transporter; PM, plasma membrane; PSs, phytosiderophores; YSL1, yellow strip-like 1.

disease (Galaris and Pantopoulos, 2008; Kell, 2009). Notably, the bioavailability of oxidised Fe^{3+} is poor due to the limited solubility of its compounds. Thus, the acquisition, usage and detoxification of Fe poses a considerable challenge for cells and organisms, which have evolved sophisticated mechanisms to satisfy their metabolic needs and, concomitantly, minimise the risk of toxicity (Andrews, 2008; De Domenico et al., 2008; Hentze et al., 2010). In plants, most of the concern about Fe nutrition is related to its low availability in soil solutions. In fact, notwithstanding its abundance, Fe exists in well-aerated soils as scarcely soluble oxides and oxyhydroxides and is therefore not freely available for plant uptake. To cope with this problem, plants have developed two main strategies: graminaceous plants use a chelation-based strategy (Strategy II), whereas the majority of plants, dicotyledons and non-graminaceous monocotyledons, use a reduction-based strategy (Strategy I) (Schmidt, 1999; Curie and Briat, 2003; Abadía et al., 2011) (Fig. 1). The chelation-based mechanism (Strategy II) has evolved in grasses, which includes most of the world's staple grain crops. Grasses produce molecules of the mugineic acid family called phytosiderophores (PSs). The PSs form a stable, hexadentate complex with Fe^{3+}, which is the predominant form of Fe found in aerobic soils. PSs are secreted into the rhizosphere where they chelate and help to solubilise Fe^{3+}. The Fe (III)–PS complex is then absorbed by the root cells through the action of Yellow Stripe1 (YS1) proteins (von Wiren et al., 1999; Curie et al., 2001; Abadía et al., 2011) (Fig. 1, right panel). In Strategy I, Fe acquisition is mediated by a reduction-based mechanism: a ferric chelate reductase (FCR) converts Fe (III)-chelates to Fe (II), and an iron regulated transporter (IRT1) moves the ion across the plasma membrane (PM) into the cell. Additionally, in most of the Strategy I plants studied to date, there is an associated increase in the activity at the PM, i.e., H^+-ATPase transports ions across the membrane, which lowers the pH of the growth medium, increases the Fe solubility and generates the electrochemical proton gradient necessary to drive ion uptake (Rabotti and Zocchi, 1994; Dell'Orto et al., 2000; Santi et al., 2005; Santi and Schmidt, 2008, 2009) (Fig. 1, left panel). The genes encoding these enzymes have been identified in several plants (Eide et al., 1996; Robinson et al., 1999; Eckhardt et al., 2001; Waters et al., 2002, 2007; Santi et al., 2005). Additionally, in Strategy I plants a plethora of organic compounds, including carboxylates, phenolics, and flavonoids, are increased under Fe deficiency (Cesco et al., 2010; Tomasi et al., 2008).

Under Fe-limited growth conditions, the induction of the Strategy I mechanism is not restricted to the activation of FCR, IRT1 and H^+-ATPase. A metabolic reprogramming of plant root cells is necessary; the activation of reduction processes and the enhanced proton extrusion require an adequate energy supply in the form of NADPH and ATP. The recharging of such substrates requires the acceleration of the metabolism that is strictly linked to energy production (Zocchi, 2006; Vigani and Zocchi, 2009). In fact, several papers published in the last 30 years revealed that almost all enzymes belonging to the glucose catabolism, mainly at the glycolysis level, are increased in several Strategy I plants (Table 1). Among the metabolic activities that increase in Fe–deficient plants, phospho*enol*pyruvate carboxylase (PEPC) is one of the most important (see references in Table 1). PEPC is a ubiquitous enzyme in plants that catalyses the fixation of bicarbonate to phospho*enol*pyruvate (PEP) to produce oxaloacetate (OAA) and Pi. To maintain an

Table 1
Experimental evidences about the increase of some cytosolic enzymes of glucose catabolism under Fe deficiency in several Strategy I plants. References are tabulated according to the type of induction (activity and/or protein content and/or transcript) observed under Fe deficiency with regard the specific enzyme. The numbering of references is specified below.

Enzyme	Activity	Protein	Transcript
HK	7		
PGI			
ATP-PFK	5; 7; 11; 12	14; 15	
PP-PFK		14	
F1,6BPA		8; 13; 14; 15	6
G3PDH	1; 2; 5; 11; 12	12; 13; 15	6
TPI		8; 13	6
PGK		13; 14	6
PGM		8; 14; 15	6
Enolase		8; 13; 14; 15	6
PK	5; 7; 11; 12		6
PEPC	2; 3; 4; 7; 9; 10; 11; 12; 16	3; 8; 16	6; 10; 17
G6PDH	1; 2; 4; 11; 12	14; 15	

(1) Sijmons and Bienfait (1983); (2) Rabotti et al. (1995); (3) De Nisi and Zocchi (2000); (4) López-Millán et al. (2000a,b); (5) Espen et al. (2000); (6) Thimm et al. (2001); (7) Zocchi et al. (2007); (8) Li et al. (2008); (9) López-Millán et al. (2009); (10) Andaluz et al. (2009); (11) M'Sehli et al. (2008); (12) Jelali et al. (2010); (13) Rellán-Álvarez et al. (2010); (14) Donnini et al. (2010); (15) Rodríguez-Celma et al. (2011); (16) Slatni et al. (2011); (17) De Nisi et al. (2010). *Abbreviations*: ATP-PFK, ATP-dependent phosphofructokinase; F1,6BPA, fructose 1,6-bisphosphate aldolase; G6PDH, glucose-6-phosphate dehydrogenase; GAPDH, glyceraldehyde 3-phosphate dehydrogenase; HK, hexokinase; PEPC, phospho*enol*pyruvate carboxylase; PGI, phosphoglucoisomerase; PGK, phosphoglycerate kinase; PGM, phosphoglycerate mutase; PK, pyruvate kinase; PP-PFK, PP-dependent phosphofructokinase; TPI, triosephosphate isomerise.