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

This is the author’s manuscript

Original Citation:


Availability:

This version is available http://hdl.handle.net/2318/1655341 since 2018-01-12T14:52:12Z

Published version:

DOI:10.1016/j.jplph.2011.09.008

Terms of use:

Open Access
Anyone can freely access the full text of works made available as "Open Access". Works made available under a Creative Commons license can be used according to the terms and conditions of said license. Use of all other works requires consent of the right holder (author or publisher) if not exempted from copyright protection by the applicable law.

(Article begins on next page)
This is the author's final version of the contribution published as:


The publisher's version is available at:
https://ac.els-cdn.com/S0176161711004093/1-s2.0-S0176161711004093-main.pdf?_tid=458d46ca-f79f-11e7-bf8b-00000aaccb35f&acdnat=1515765090_5d8df8bd1da43bcd03d418d6c205c00

When citing, please refer to the published version.

Link to this full text:

This full text was downloaded from iris-Aperto: https://iris.unito.it/
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.

Abbreviations

mETC: mitochondrial electron transport chain
ND: internal type II NADPH dehydrogenases
ND: external type II NADPH dehydrogenases

Keywords

Energy metabolism, Fe deficiency, Tricarboxylic acid (TCA) cycle, Mitochondria, Respiratory chain

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 (Fe2+) and the oxidised ferric (Fe3+) 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 disease (Galaris and Pantopoulos, 2008; Kell, 2009). Notably, the bioavailability of oxidised Fe3+ 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 Fe3+, which is the predominant form of Fe found in aerobic soils. PSs are secreted into the rhizosphere where they chelate and help to solubilise Fe3+. The Fe (III)–PS complex is then absorbed by the root cells through the action of Yellow Stripe1 (YS1) proteins (von Wirén 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).

![Fig. 1. Schematic representation of the mechanisms of iron uptake in plant roots.](image)

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; Viganì 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, phosphoenolpyruvate 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 phosphoenolpyruvate (PEP) to produce oxaloacetate (OAA) and Pi. To maintain an adequate rate of glycolysis, PEP must be consumed at a high rate. Under conditions of Fe deficiency, it has been postulated that the activation of PEPC could be the downstream driving force for glycolysis that leads to an increase in the rate and production of cytosolic acidification to activate H+–ATPase (Zocchi, 2006). Additionally, the activation of PEPC could be responsible for the production of organic acids. Indeed, when under Fe deficiency, plants produce considerable amounts of organic acids, mainly citrate and malate (Abadía et al., 2002 and references therein). The following is a list of potential role(s) of increased organic acids under Fe deficiency: (i) to control the cytosolic pH or feed the increased activity of H+–ATPase at the PM; (ii) to increase production of oxalacetate (as a product of PEPC) and/or malate to replenish the tricarboxylic acid (TCA) cycle; and/or (iii) to facilitate the acquisition of Fe from the soil by the root cell (Abadía et al., 2002).

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.

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

(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. (2006); (12) Jelali et al. (2010); (13) Rellán-Alvarez 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, phosphoenolpyruvate carboxylase; PGI, phosphoglucoisomerase; PGK, phosphoglycerate kinase; PGM, phosphoglycerate mutase; PK, pyruvate kinase; PP-PFK, PP-dependent phosphofructokinase; TPI, triosephosphate isomerase.

Overall, the upregulation of glycolysis under Fe deficiency provides: (i) ATP synthesis to sustain the major activity of the H+–ATPase; (ii) the formation of reducing equivalents for
the FC-R; (iii) the formation of PEP and (iv) a contribution to the regulation of cytosolic pH. Because Fe deficiency induces an energy demand in the cell, it is critical to understand how this stress affects mitochondria.

Plant mitochondria and iron

Mitochondria play a key role in energy transduction, and are the site of aerobic respiration, a process common to almost all eukaryotic organisms that releases a large amount of free energy in the cell. Mitochondria possess two membranes that divide the organelle into four compartments: (i) the outer membrane (OMM); (ii) the region between the two membranes or intermembrane space (IMS); (iii) a highly invaginated inner membrane (IMM); and (iv) the matrix, which is the aqueous phase contained within the inner membrane (Sweetlove et al., 2007). The IMM is the site of oxidative phosphorylation (OXPHOS) electron transport chain coupling NADH and FADH2 oxidation, and reduction of oxygen, to proton translocation and OXPHOS. The TCA cycle, which is responsible for the oxidative decarboxylation of organic acids leading to reduction of NADP and FAD and substrate-level phosphorylation of ADP to ATP, takes place in the matrix (Millar et al., 2011 and references therein).

The mitochondrion occupies a central place in the metabolic network of eukaryotes. Essential metabolic processes occur within the organelle, and several other metabolic pathways either converge on or derive from the mitochondrion. In plants, mitochondria provide precursors for a number of essential biosynthetic processes (e.g., nitrogen fixation and the biosynthesis of amino acids, tetrapyrroles and vitamin cofactors, Douce, 1985; Douce and Neuberger, 1989; Mackenzie and McIntosh, 1999). Interestingly, mitochondria are also a focal point of Fe metabolism. An analysis of the metallome of mitochondria isolated from Arabidopsis revealed a 26:8:6:1 molar ratio for iron:zinc:copper:manganese (Tan et al., 2010), suggesting that Fe is the primary micronutrient present in the mitochondrion (Nouet et al., 2011). Mitochondria contain a large amount of metalloproteins that require Fe to carry out their function (Bertini and Rosato, 2007). In fact, several enzymes belonging to both the respiratory chain and to the TCA cycle are Fe-containing proteins. Moreover, crucial steps of the Fe–S cluster assembly for the entire cell take place in the mitochondria, suggesting an important role for this compartment in Fe handling by the cell.

Iron deficiency and mitochondria are strongly linked because this stress greatly induces the Strategy I activities that lead to a great energy demand, thus the participation of the mitochondria would be required.

Iron uptake and handling in plant mitochondria

Once inside the root cell, Fe can face different fates: (i) chelation by citrate and stored in vacuole or transported to the shoot; (ii) sequestration by ferritin in the cytosol; or (iii) transportation to organelles, such as mitochondria (Conte and Walker, 2011). The processes of Fe transport into plant mitochondria are partially unknown. The OMM permeability is conferred by a family of porin proteins, forming channels for the free diffusion of a wide range of molecules, and no specific Fe transporter has been identified so far. In the IMM, the Fe importer MIT1 (Mitochondrial Iron Transporter 1) has been recently identified, which is essential for plant growth and development (Bashir et al., 2011). Furthermore, the ABC transporter STA1/AtATM3 (ATP-binding cassette Transporters of Mitochondria 3) has been characterised in Arabidopsis and implicated in the export of Fe–S clusters (Kushnir et al., 2001). In addition, FCR(s), encoded by FRO genes in plants, might be involved in mitochondrial Fe transport because AtFRO3 and AtFRO8 contain mitochondria-targeting sequences and are mainly located in root and shoot veins, respectively (Mukherjee et al., 2006). These findings suggest that a reduction-based uptake could also take place in the IMM of mitochondria. Moreover, the oxidising
conditions found in the mitochondrial IMS (Hu et al., 2008) would indicate the need for a reduction-based mechanism (Abadía et al., 2011). Once Fe is inside the mitochondria, it can be utilised in several pathways. The most important pathways for mitochondrial functionality and cell viability are Fe–S cluster assembly, haem biosynthesis and Fe storage.

Iron–sulphur (Fe–S) clusters are cofactors that are chemically simple but functionally versatile. In fact, they represent a prosthetic group of proteins that are essential to life processes, such as photosynthesis, respiration and nitrogen fixation. Fe–S clusters consist of Fe and acid-labile sulphide (Balk and Lobréaux, 2005). Plant mitochondria operate the so-called ISC (Iron Sulphur Cluster) pathway for the assembly of the Fe–S cluster. Recently, this pathway was extensively reviewed (Balk and Pilon, 2011). Interestingly, the Fe–S cluster assembly capability of plant mitochondria is essential not only for mitochondrial Fe–S proteins but also for cytosolic Fe–S protein synthesis. Indeed, in the cytosol, the so-called Cytosolic Iron–sulphur protein Assembly (CIA) machinery is present. Cytosolic Fe–S protein synthesis depends on ISC components and a mitochondrial ABC transporter (ATM3) that has been proposed to export an unidentified product of the ISC pathway, allowing Fe–S cluster assembly and therefore Fe–S protein synthesis in the cytosol by the CIA machinery (Balk and Pilon, 2011 and references therein).

There is also evidence that mitochondria can synthesise haem. Haem is a prosthetic group formed by an Fe atom that coordinates the binding of a large heterocyclic organic ring called a porphyrin. Not all porphyrins contain Fe, but a substantial fraction of porphyrin-containing metalloproteins has haem as their prosthetic group: the so-called haemoproteins. Haem forms the prosthetic group of photosynthetic and respiratory cytochromes involved in energy transduction and oxidases, such as catalase, peroxidase and NADPH oxidase (Sweetlove et al., 2007 and references therein). In plant cells, the presence of the haem biosynthetic pathway in plant mitochondria is still a matter of debate. However, some authors report that the enzymes that catalyse the final steps of haem biosynthesis, such as ferrochelatase (which catalyses the insertion of ferrous Fe into protoporphyrin IX) and protoporphyrinogen oxidase, are also present in mitochondria and in plastids (Sweetlove et al., 2007 and references therein).

The mitochondrial proteins that are critical for Fe homeostasis are ferritin and frataxin. Generally, ferritin is considered the primary Fe storage protein in the cell. Ferritin provides protection against Fe toxicity and oxidative damage, and its expression is increased upon exposure to an excess of Fe (Eisenstein, 2000; Arosio et al., 2009; Briat et al., 2010a,b). Zancani et al. (2004) reported the mitochondrial localisation of ferritin in both Pisum sativus and Arabidopsis thaliana. Moreover, recently, the ferritin isoform FER4 was identified in the mitochondria in Arabidopsis heterotrophic cells (Tarantino et al., 2010b). A fer4 Arabidopsis mutant displays different rearrangements of its respiratory machinery but is not impaired in its development or response to abiotic stress (Tarantino et al., 2010a,b).

Although the precise role of ferritin in plant mitochondria has not been resolved completely, there is evidence for its role in Fe homeostasis. Frataxin is a conserved mitochondrial protein implicated in cellular Fe homeostasis, which functions as an Fe chaperone that delivers Fe for the Fe–S cluster and haem biosynthesis (Malinardi et al., 2011 and references therein). Frataxin deficiency causes severe disease in both humans and plants; the absence of frataxin induces severe oxidative stress in mitochondria, while its overexpression allows the cell to be more resistant to oxidising agents (Møller et al., 2011 and references therein).

Overall, it is increasingly evident that mitochondria can be considered as an ‘iron-handling area’ within the cell, possessing several specific enzymes for the maintenance of this element. Mitochondria store and process Fe to synthesise a large number of essential
Mitochondrial respiration and Fe deficiency

In the mitochondria of all eukaryotes, the principal respiratory electron transport chain consists of a series of membrane-bound redox centres that catalyse the multi-step transfer of electrons from NADH and FADH2 to oxygen. The mitochondrial electron transfer chain (mETC) facilitates a number of exergonic redox reactions and conserves energy through proton translocation. The standard mETC consists of four multi-centred protein complexes (referred to as Complexes I through IV). In addition, the F1F0-ATP synthase (Complex V), which does not have electron transfer activity, provides a path for passive proton diffusion into the matrix and uses the free energy released by this spontaneous movement to drive the phosphorylation of ADP. In addition to the electron transfer chain shared by all aerobic eukaryotes, plant mitochondria also possess novel pathways for both the oxidation of NADH and the reduction of oxygen. These bypass proteins, the so-called alternative type II NADPH dehydrogenases [located both in the external surface of the IMM (NDex) and the internal surface of the IMM (NDin)] and the alternative oxidase (AOX), do not pump protons, and the free energy released as electrons flow through them is lost as heat. These additional enzymes allow a great deal of flexibility in the oxidation of NADPH in plants. Type II NADPH dehydrogenases reduce ubiquinone and thus circumvent respiratory Complex I, whereas AOX bypasses Complexes III and IV of the cytochrome pathway by directly oxidising ubiquinol. Millar et al. (2011) recently reviewed the specific molecular and genetic features of each enzyme of the respiratory chain.

In the absence of Fe, the levels of some Fe-containing components (cytochromes and Fe-S clusters) were greatly diminished in the mitochondria of Fe-deficient sycamore cells (Pascal and Douce, 1993) and cucumber roots (Vigani et al., 2009). In Fe-deficient sycamore cells, the activity of Complex II was affected, whereas Complexes I, III, and IV, and ATP synthase, were unimpaired. In contrast, in cucumber root, the number of Fe ions required for the correct assembly of each mETC complex (unit) and their relative activities under Fe-deficient conditions are highly correlated: Complex IV, which has only two Fe atoms, showed a decrease in activity of approximately 50%, whereas Complexes I and II, which require at least 20 and 10 Fe atoms, respectively, showed a greater decrease (95% and 77%, respectively). A proteomic investigation of Medicago truncatula roots under Fe deficiency showed a decreased level of some Fe-containing proteins related to the respiratory chain (e.g., 76 kDa mitochondrial Complex I) (Rodríguez-Celma et al., 2011). In contrast, a proteomic study performed on Fe-deficient Arabidopsis roots showed an increased level of several proteins belonging to the respiratory chain. These proteins included cytochrome c oxidase, the ubiquinol-cytochrome c reductase complex ubiquinone-binding protein and three components of mitochondrial Complex I (Lan et al., 2011). The contrasting results obtained in cucumber compared with Arabidopsis might be explained by the different degree of Fe deficiency conditions used in the studies.

Overall, root mitochondria are strongly affected by Fe starvation because they require at least forty 40 Fe atoms per respiratory unit to function (Vigani et al., 2009). Nevertheless, when Fe availability is low, either due to absence, as in the cucumber study, or low supply (Fe content in the nutrient solution is low but not zero), as in the Arabidopsis (Lan et al., 2011) and sycamore cell (Pascal and Douce, 1993) studies, the mitochondria are still working.

Under increased Fe deficiency, it is possible to observe an interesting regulation of the respiratory chain in cucumber root; the dehydrogenase pathways (the enzymes able to reduce the UQ pool) of the mETC are more affected with respect to the cytochrome pathways (the enzymes able to oxidase the UQ pool). The strong decrease of Complex I and Complex II activities in the absence of Fe is compensated by the activation of the type
II alternative NADPH dehydrogenase (see above). In fact, both the enzymatic activity and the protein level of the NDex are highly induced compared with the NDIn (Vigani and Zocchi, 2010). These findings suggest that the respiratory chain could contribute to cytosolic NADPH turnover through the NDex, notwithstanding that the mETC is strongly inhibited by Fe deficiency (Vigani and Zocchi, 2010). Accordingly, Higa et al. (2010) observed an induction of type II alternative NADPH dehydrogenase in the Fe-deficient roots of *Hyoscyamus albus* that was similar to that in cucumber, where low Fe availability decreases the main dehydrogenase enzymes of the mETC (Complexes I and II) and induces the alternative dehydrogenase pathways. Interestingly, Higa and co-workers (2010) stated that Complex I and Complex II require not only a large number of Fe ions but also flavins as cofactors. Interestingly, flavin accumulation and excretion are induced upon Fe deprivation in various plant species (Pound and Welkie, 1958; Nagarajah and Ulrich, 1966; Kannan, 1988; Welkie et al., 1990). It has been suggested that flavins, which accumulate in Fe-deficient roots, could act as electron donors or as cofactors for the FCR (Rodríguez-Celma et al., 2011; López-Millán et al., 2000a,b; Gonzalez-Vallejo et al., 1998; Welkie and Miller, 1993) because this enzyme contains an FAD-binding sequence motif (Schagerlof et al., 2006). However, it is rational to consider that if Complex I and Complex II proteins were prevented from becoming fully functional by Fe deficiency, the unused flavins might be transported outside the mitochondria, reaching the apoplast and the rhizosphere (Higa et al., 2010).

Generally, Fe deficiency affects the respiratory chain by limiting the activity of Fe-containing enzymes and inducing the alternative enzymes. This regulation allows the respiratory chain to partially satisfy the energy demand of the cell.

### TCA cycle and Fe deficiency

The TCA cycle is a universal feature of the metabolism in aerobic organisms. It begins with the condensation of OAA and acetyl-CoA, proceeds via a series of oxidative steps that release two carbon atoms as CO₂, and ends with the regeneration of OAA (Fig. 2). In plants, acetyl-CoA is derived from the end product of glycolysis, pyruvate, by the action of mitochondrial pyruvate dehydrogenase. The pyruvate is either imported directly from the cytosol or synthesised by the mitochondrial NAD-malic enzyme from malate that, in turn, is derived from cytosolic PEP. PEP carboxylase generates cytosolic OAA, which is readily converted to malate and aspartate. All three metabolites can be imported into the mitochondria to provide a source of malate for the NAD-malic enzyme (Sweetlove et al., 2010).

![FIG. 2](https://example.com/fig2.png)

**Fig. 2.** Representation of the changes occurring at the TCA cycle level under Fe deficiency. (A) Hypothetical non-cyclic TCA mode occurring in the case of decrease of aconitase and SDH activities under Fe deficiency. (B) Hypothetical non-cyclic TCA mode occurring in the case of decrease of SDH activity under Fe deficiency.

Several authors have suggested that Fe deficiency induces the activation of the TCA cycle. Li et al. (2008) observed that the expression of several TCA enzymes (isocitrate dehydrogenase, aconitase, succinyl-CoA ligase, and succinate dehydrogenase) was simultaneously upregulated in Fe-deficient tomato roots. López-Millán et al. (2009) showed that the total organic anion concentrations in tomato root tips did not change significantly with Fe deficiency, but their composition was markedly changed. In the control root tips, the major organic anions were oxalate, 2-OG and malate, whereas in Fe-deficient root tips, citrate and malate accounted for 90% of the total organic anion concentrations. Moreover, the authors showed that the activities of five enzymes (ICDH, aconitase, MDH, fumarase and CS) involved in organic acid metabolism increased significantly in the tissue
extracts of Fe-deficient root tips when compared with the Fe-sufficient tips. The authors pointed out that the upregulation of the TCA cycle might have special significance for roots responding to Fe deficiency. The TCA cycle provides abundant organic acids, including citrate and malate, which play an important role as chelators for Fe transport in plants (López-Millán et al., 2000b; Tiffin, 1966). A metabolite study in Beta vulgaris revealed large increases in organic acids related to the TCA cycle (Rellán-Álvarez et al., 2010). Additionally, a proteomic analysis performed on Medicago truncatula roots revealed an increase in the amount of several TCA cycle enzymes under Fe-deficient conditions (Rodríguez-Celma et al., 2011). All these data suggest that the TCA cycle plays an important role in the Fe deficiency response by providing organic acids and protons for the acidification of the rhizosphere.

Nevertheless, none of the studies mentioned above involved the isolation of purified mitochondria. Therefore, they were unable to discriminate between the identical reactions occurring in the mitochondrial matrix and in the cytosol (i.e., reactions catalysed by aconitase, malate dehydrogenase and isocitrate dehydrogenase). One complication inherent in studying the TCA cycle is that almost all the steps in the pathway can be bypassed by steps in the cytosol; however, the reactions catalysed by citrate synthase and succinate dehydrogenase are exceptions.

Like the respiratory chain, the TCA cycle possesses two Fe-containing enzymes [i.e., aconitase and the succinate dehydrogenase (Complex II of the respiratory chain)]. As previously discussed, the succinate dehydrogenase activity in purified mitochondria obtained from cucumber roots (Vigani et al., 2009) and sycamore cells (Pascal and Douce, 1993) strongly decreased under Fe deficiency, suggesting that the TCA cycle was decelerated at least at some point.

Sweetlove et al. (2010) recently reviewed the different carboxylic acid metabolism flux modes predicted from metabolic models and concluded that cyclic TCA could not be just a cycle. They propose that the cyclic mode of TCA provides efficient energy transfer from the carbon bonds of respiratory substrates to ATP, suggesting that it might not be necessary to maintain the cyclic flux if the demand for ATP is low or if alternative sources of ATP are available. Consistent with this argument, a recent flux-balance model of heterotrophic Arabidopsis metabolism demonstrates that a cyclic TCA flux is only established as the demand for ATP increases in a cell (Poolman et al., 2009; Sweetlove et al., 2010).

It was proposed that the effect of Fe deficiency on the TCA cycle could induce different changes. Considering the expected decrease of aconitase and succinate dehydrogenase activities under Fe deficiency, the TCA cycle could shift from a circular to a linear mode, creating at least two metabolic scenarios (Fig. 2). First, if both aconitase and succinate dehydrogenase activities are strongly affected by Fe deficiency, the TCA cycle could similarly work as in the non-cyclic flux mode as predicted from the analysis of an enzyme kinetic model (Steuer et al., 2007); malate is imported into the matrix and then converted to citrate, which is subsequently exported from mitochondria (Fig. 2A). This hypothetical scenario is plausible because there is evidence for the accumulation of the di- and tricarboxylic acid carrier (DTC) protein under Fe deficiency, suggesting an increased transfer of organic acids, mainly malate and citrate, between the mitochondria and cytosol. Additionally, increased citrate export from the mitochondria to the cytosol has been observed in several species under Fe deficiency (Vigani and Zocchi, 2009 and references therein). However, there is no direct evidence that mitochondrial aconitase is affected by Fe deficiency, even if it would be expected.

Indeed, the second scenario implies that Fe deficiency affects only succinate dehydrogenase. In this case, the TCA cycle could work as in the non-cyclic flux mode
predicted under conditions of low ATP demand in a genome-scale metabolic model of Arabidopsis (Poolman et al., 2009; Sweetlove et al., 2010); malate and/or OAA are imported into the matrix and then converted to 2-OG, which is subsequently exported from the mitochondrion together with citrate (Fig. 2B). This hypothesis is also reasonable because there is evidence for the strong inactivation of Complex II under Fe deficiency (Pascal and Douce, 1993; Vigani et al., 2009).

Taking into consideration that the TCA cycle does not necessarily support a cyclic flux and that the actual flux distribution through it will reflect the function of the whole metabolic network, a combination of these non-cyclic TCA modes could occur under Fe deficiency. Furthermore, the presence of a cyclic or non-cyclic mode of TCA might depend on the degree of stress. Indeed, it could be hypothesised that when the amount of Fe in the cell is close to zero, Fe-containing enzyme activities are inhibited, which leads to a non-cyclic TCA mode. When the amount of Fe is low but not depleted, the Fe-containing enzymes might still function, leading only to a slowing of the TCA cycle. In any case, the shift from a cyclic to a non-cyclic TCA mode under Fe deficiency could result from a necessity to maintain a functioning metabolism, even if important Fe-containing enzymes are not synthesised.

Mitochondrial dynamics in Fe-deficient plants

The morphology and ultrastructure of the mitochondria are also affected by Fe deficiency. In fact, mitochondria from Fe-deficient sycamore cells displayed a more dilute matrix with less pronounced cristae (Pascal and Douce, 1993). Interestingly, in Fe-deficient cucumber roots, mitochondria displayed a particular handlebar-like structure and appeared to aggregate (Vigani et al., 2009) (Fig. 3A). However, the particular shape that was observed in Fe-deficient cucumber roots was also observed in the wild-type heterotrophic cells of Arabidopsis grown under control conditions, indicating that this shape is probably not specifically related to Fe deficiency (Tarantino et al., 2010b). A similar handlebar-like structure observed in Fe-deficient cucumber root has been observed in the Arabidopsis drp3a mutant (Logan, 2006, 2010) (Fig. 3). DRP3A is a dynamin-like protein that is required for the maintenance of mitochondrial size and number in Arabidopsis and rice; this protein has been partially localised to the poles of mitochondria and to sites of constriction (Arimura et al., 2004; Fujimoto et al., 2004; Logan et al., 2004). The disruption of DRP3A results in an aberrant mitochondrial morphology that is characterised by an increase in the size of individual mitochondria and a corresponding decrease in the number of mitochondria per cell (Logan, 2010 and references therein). Interestingly, drp3a mutants displayed mitochondria with the presence of a matrixule, a thin protuberance of up to many micrometers in length that extends from one pole of a mitochondrion (Fig. 3B). It has been proposed that the presence of numerous mitochondria with matrixules in drp3a mutants is linked to the function of DRP3A as a component of the mitochondrial division machinery (Logan, 2010 and references therein).
Fig. 3. Morphological comparison of mitochondria from the Fe-deficient cucumber root (Vigani et al., 2009; kind permission to reproduce from John Wiley and Sons, license number: 2740690508918) and dpr3a mutant of Arabidopsis (Logan et al., 2004; kind permission to reproduce from Oxford University Press, license number: 2740640528797). (A) TEM analysis displaying the handlebar-like morphology observed in Fe-deficient cucumber roots (bar = 1 µm). (B) GFP imaging analysis of the mitochondria from Arabidopsis dpr3a mutants (bar = 10 µm). This mutant displayed mitochondria with the presence of matrixule, a thin protuberance of up to many micrometers in length that extends from one pole of a mitochondrion. Interestingly, similar matrixule formation seems to occur in Fe-deficient cucumber plants. Abbreviations: CW, cell wall; M, mitochondrion; V, vacuole.

The higher plant chondriome (all the mitochondria in a cell, collectively) is a highly dynamic structure composed predominantly of physically discrete organelles. This structure contrasts with that of most animal cell types and yeast cells in which the chondriome is frequently organised into long tubules or reticula. An analysis of the mitochondrial morphology in vivo has shown that higher plant mitochondria are highly pleomorphic, although most frequently they are spherical to sausage-shaped organelles. Mitochondria are also dynamic in terms of their movement within cells, moving rapidly along cellular structures, such as actin and microtubule cytoskeletal elements (Logan, 2006, 2010 and references therein). The primary mitochondrial processes that regulate the shape and the movement of the organelles in the cell are fusion and fission. These two processes compensate for one another, leading to the maintenance of a relatively constant number of mitochondria in the cell (Logan, 2006, 2010). Several authors have reported that the number of mitochondria was increased in Fe-deficient cells (Landsberg, 1986, 1994; Pascal and Douce, 1993; Dell’Orto et al., 2002); therefore, it is rational to hypothesise that this nutritional stress could modify the mitochondrial fusion/fission ratio. Because the plant chondriome is organised as a discontinuous whole, proper mitochondrial function requires fine control over the mitochondrial motility and cellular distribution. In fact, it is intrinsic in the discontinuous whole hypothesis that there is a ‘need to meet’, where the need is to provide a mechanism for the exchange of mitochondrial
proteins; the fusion of physically discrete organelles requires mitochondrial movement (Logan, 2010). The control of mitochondrial positioning and direction of movement are the result of the coordinated activity of myosin and the rate of actin turnover (Logan, 2010). Under Fe deficiency, the amount of actin decreases (Donnini et al., 2010), whereas the number of mitochondria increases. Mitochondrial motility has not been studied in Fe-deficient plants, and we do not know exactly how cells organise the cytoskeleton to allow organelle and vesicle trafficking in the cytosol. We might speculate that the increased number of mitochondria observed in Fe-deficient roots in several studies could be in response to an inefficient cytoskeletal structure to enhance the probability of mitochondrial contact, thus maintaining the discontinuous whole structure of the plant chondriome. In this way, the cell could satisfy the “need to meet” hypothesis. Interestingly, under Fe deficiency, mitochondrial contact results from a strongly affected metabolism in which the merging of mitochondria could help them to overcome the lack of Fe and at the same time, facilitate cell survival. Moreover, the probable shift towards a non-cyclic TCA mode increases mitochondrial communication with external components, within the cytosol and with other mitochondria. The fact that under a mild Fe deficiency, one of the first enzymes affected is Complex II (Pascal and Douce, 1993) would mean that mitochondria, perceiving an upcoming Fe deficiency, reprogram their own function, adjusting both the respiratory chain and the TCA cycle by acting on only one enzyme.

Role of mitochondria in controlling the NADPH reduction levels under Fe deficiency

The presence of type II NADPH dehydrogenases with different properties on both sides of the mitochondrial inner membrane allows the mETC to specifically regulate the reduction of either mitochondrial or cytosolic NADH or NADPH (Rasmusson and Wallström, 2010 and references therein). Importantly, metabolite exchangers, together with the activity of the TCA cycle enzymes, mediate reductant shutting across the inner mitochondrial membrane, modulating the turnover of reducing equivalents in both the mitochondria and the cytosol. Indeed, the reduction of NADH in the matrix and the cytosol are believed to equilibrate primarily with the malate/OAA ratios in each compartment, while the reduction of NADPH pools between the matrix and cytosol is equilibrated through the export of reductant from mitochondrion to the cytosol by both a citrate valve that exchanges citrate and 2-oxoglutarate (Igamberdiev and Gardeström, 2003) and a malate valve that exchanges malate and OAA (Scheibe, 2004).

In this context, the reducing power turnover of Fe-deficient cells undergoes a fine and complex regulation to satisfy the metabolic request. Indeed, because both the cytosolic NADPH and the mitochondrial NADPH need to be re-oxidised to keep the catabolic pathways working, a less active respiratory chain could impair this turnover, leading to a higher reduced/oxidised NADP ratio, as shown by the increased NADPH/NADP⁺ ratio in Fe-deficient bean and Plantago lanceolate (Sijmons et al., 1984; Schmidt and Schuck, 1996). However, in Fe-deficient sugar beet and tomato roots, the NADPH/NADP⁺ ratio was lower than the relative control conditions (López-Millán et al., 2000a,b, 2009), suggesting that Fe deficiency might stimulate the NADPH-oxidising activities more that the NADP⁺-reducing activities.

To oxidise the large production of NADPH from metabolism, the cell activates reactions that are able to consume NADPH. López-Millán et al. (2000a,b, 2009) reported that Fe deficiency increased the activities of fermentative enzymes, such as lactate dehydrogenase (LDH), pyruvate decarboxylase (PDC) and several other cytosolic NADH-consuming enzymes, which could contribute to the oxidation of the pyridine nucleotide pool in tomato and sugar beet roots. These data were also confirmed by microarray analysis of Fe-deficient Arabidopsis (Thimm et al., 2001) and proteomic data obtained from cucumber roots, in which putative alcohol dehydrogenase and malate
dehydrogenase proteins accumulated under Fe deficiency (Donnini et al., 2010). The mitochondria might participate in the oxidation of NADPH under Fe deficiency by the activation of their alternative pathways (i.e., NADPH DHs). The external and internal localisation of these enzymes in the inner mitochondrial membrane allows them to oxidise the NADPH on both the cytosol and the matrix sides (Vigani and Zocchi, 2010). Because Fe deficiency leads to a strong energy demand in the cell, the activation of mitochondrial metabolism would represent the perfect solution for the cell. However, the lack of Fe strongly affects mitochondrial functionality. Nevertheless, the activity of the plant mitochondria results in a complicated reprogramming of their own metabolism. At the level of the respiratory chain, alternative pathways are induced, bypassing the impaired complexes. The TCA pathway could shift from a cyclic to a non-cyclic mode, bypassing the limiting step (for instance, succinate dehydrogenase) and improving external communication. This bypass might be accomplished by inducing both the anaplerotic reactions to produce organic acids (which are useful for the responses to Fe deficiency) and the metabolite shuttles to allow the recycling of reducing power. The cell, in some way, helps mitochondria satisfy their “need to meet”, which would be high under Fe deficiency, by increasing the number of mitochondria. To facilitate mitochondrial activity, the cell could obtain useful compounds for rhizosphere acidification and Fe chelation, such as organic acid and flavins, which result from the disuse of Complexes I and II (Fig. 4).
Fig. 4. Schematic representation of the role of mitochondria in the Fe-deficient dicotyledonous root cell. Under Fe deficiency, the activation of Strategy I responses (FCR, IRT1, H+-ATPase) requires a large supply of energetic substrates [i.e., ATP and NADPH]. Mitochondria cannot meet this energetic requirement because Fe deficiency affects several mitochondrial enzymes. Thus, the cell activates other pathways (e.g., glycolysis) to overcome the energetic emergency status. Nevertheless, mitochondria participate in metabolic responses induced by Fe deficiency by activating their own alternative respiratory pathways. Moreover, unused flavin compounds (i.e., FMN) could be transferred to the cytosol and extruded into the rhizosphere, facilitating the Fe reduction activity. Overall, the metabolic alteration of Fe-deficient root cells depends on the mitochondrial functionality. This organelle, which represents the “Iron-Area” of a root cell, seems to be the main target of the Fe deficiency, which might also become a starting point for whole cellular metabolic reprogramming to overcome this emergency energetic status (Vigani and Zocchi, 2010). The Fe deficiency establishes a cellular effort in which mitochondria activate alternative pathways to sustain NADPH turnover and organic acid synthesis. In this way, mitochondria facilitate the increase of glycolysis, which becomes a strong source
of energy substrates for the activation of the response to Fe deficiency. Could mitochondria be involved in the signal transduction of the Fe-deficient status of the cell? The mitochondrion occupies an integrated position in the metabolic network of plants, reflecting the evolutionary origins of the organelle. Indeed, through its integration into the ancestral eukaryotic cell, the mitochondrion provided a new arsenal of metabolic capabilities (Martin and Russell, 2003; Sweetlove et al., 2007). To communicate with external compartments, mitochondria generate several compounds as signals, such as ROS and organic acid. It is well known that mitochondria produce ROS that not only cause damage to cellular components but are also involved in intracellular signalling; H$_2$O$_2$ is considered the most likely messenger (Neil et al., 2002; Møller et al., 2011). Importantly, abiotic stress influences mitochondrial H$_2$O$_2$ production (Møller, 2001), which could generate secondary signals and therefore transduce the oxidative damage of the mitochondrial component to the cell (Halliwell, 2006; Rhoads et al., 2006). Indeed, because H$_2$O$_2$ does not have the required specificity to regulate specific genes, it has been proposed that (carbonylated) peptides deriving from the proteolytic degradation of oxidised protein could irreversibly be the specific secondary ROS messenger from mitochondria to regulated source-specific genes (Møller and Sweetlove, 2010; Møller et al., 2011). The relationships between Fe deficiency and secondary oxidative stress remain controversial because iron is either a constituent or a cofactor of many antioxidant enzymes and simultaneously acts as a prooxidant through the Fenton reaction (Halliwell, 2006). However, Fe deficiency is associated with an accumulation of ROS (Donnini et al., 2011). In addition to ROS, it has been suggested that TCA-cycle intermediates might be potent regulators of gene expression in a manner analogous to sugars (Finkemeier and Sweetlove, 2006; Sweetlove et al., 2007). Indeed, the intermediates of the TCA cycle could communicate the mitochondrial metabolic status to the cytosol by virtue of the presence of di- and tri-carboxylic acid transporter (DTC) proteins located in the inner mitochondrial membrane (Picault et al., 2002). The exogenous application of citrate, malate and 2-oxoglutarate could induce AOX expression (Gray et al., 2004), suggesting that it is the extra-mitochondrial levels of the TCA cycle metabolites that are sensed. Under Fe deficiency, a number of organic acids, such as citrate, are synthesised in the root cells from the TCA cycle. Moreover, DTC proteins accumulate in Fe-deficient cucumber roots (Vigani et al., 2009). The multiple roles for citrate are: Fe chelation, Fe transport, the regulation of cellular pH, and as a carbon skeleton source. Because of its multiple functions, citrate exported from the mitochondrion might be both in response to the Fe deficiency and an indicator of the metabolic status of the cell, together with the induction of several mitochondrial metabolic carriers that are able to strongly activate the exchange of organic acids and reducing equivalent compounds with the cytosol. Indeed, the cytosolic reducing equivalents might be transferred to the mitochondria through both specific reducing equivalent shuttles, such as glycerol-3-phosphate (G-3-P) shuttle (Shen et al., 2003, 2006) and several metabolite shuttles that operate between the two compartments, including shuttles for malate/OAA (Ebbighausen et al., 1985) and malate/aspartate (Journet et al., 1981). There is accumulating evidence for calcium (Ca$^{2+}$) involvement in mitochondrial signalling. In fact, Ca$^{2+}$ itself might be an important part of the signal transduction pathways that originate from the mitochondria. Mitochondria contain relatively high concentrations of Ca$^{2+}$ (Logan and Knight, 2003) and can be considered intracellular Ca$^{2+}$ stores. Moreover, mitochondrial Ca$^{2+}$ might be released into the cytosol during abiotic stress (Subbaiah et al., 1994), and there is evidence that mitochondrial Ca$^{2+}$ levels respond to environmental signals (Logan and Knight, 2003). However, there is no clear
evidence for Ca2+ involvement in the transduction of Fe deficiency-inducible responses. However, indirect findings suggest that Ca2+ might act as a second messenger in targeting the Fe deficiency signal in roots. In fact, Buckhout et al. (2009) reported the induction of genes encoding 14-3-3 proteins and some protein kinases in Fe-deficient Arabidopsis roots, which would be target proteins of the Ca2+-calmodulin-transduced signal. Moreover, in Fe-deficient cucumber roots, it has been observed that a gene encoding calmodulin was overexpressed and its relative protein level was increased (Vigani et al., in press). Overall, these data suggest that Ca2+ is potentially involved in the transduction of Fe deficiency signals and that the mitochondria could participate in targeting the signal of altered Fe nutritional status.

Another well-known second messenger that could also be important in mitochondrial signal transduction is nitric oxide (NO). It is well documented that plants generate NO and that NO can act as an important signal molecule in a variety of scenarios (Wendehenne et al., 2004; Shapiro, 2005). In plants, there are reports suggesting the existence of NO synthase (NOS)-like activities in various developmentally and physiologically stressful situations (Corpas et al., 2009). However, the existence of NOS in plant mitochondria is doubtful (Gupta et al., 2010). Indeed, it has been recently shown that plant mitochondria might be involved in NO turnover by the inter-conversion of nitrite and NO at the site of complex IV. Cytochrome c oxidase could represent the most plausible and established candidate for the nitrite-to-NO conversion, although other components of mETC could perform this reaction (Gupta and Igamberdiev, 2011 and references therein).

Importantly, NO has also recently been demonstrated to be involved in the induction of Fe-deficiency responses. Indeed, it has been shown that the NO level in roots was rapidly and continually elevated when plants were transferred to an Fe-deficient growth medium (Graziano and Lamattina, 2007; Jin et al., 2009; Chen et al., 2010). Furthermore, it has been observed that NO is required for the molecular events involved in controlling the development of lateral (Correa-Aragunde et al., 2004; Mendez-Bravo et al., 2010) and cluster roots (Wang et al., 2010), which are typical morphological changes that occur under Fe deficiency in several plants (Landsberg, 1994; Dell’Orto et al., 2002).

Thus, it is clear that mitochondria have several signal types that might be involved in Fe deficiency signal transduction.

Concluding remarks

As in other organisms, changes in the availability of Fe in plant mitochondria determine the reorganisation of cellular metabolism. The complex alteration of root metabolism under Fe deficiency has been reviewed here, discussing old and new findings. It is clear that mitochondria represent a central site of the metabolic regulation in the cell. In the case of low Fe availability, plant mitochondria become both a victim and a crucial site of fascinating alternative processes that make them unique in their role. The mitochondrion is probably the most affected compartment of the root cell under Fe deficiency. Nevertheless, it is not destined just to succumb but to play a central role, through the effective modulation of the respiratory chain and the TCA cycle, in the Fe-deficient metabolic network. The mitochondrial flexibility in the development of the adaptive response to Fe deficiency is not only related to metabolism but also in mitochondrial dynamics. Moreover, the importance of mitochondria as possible signal-producing sites suggests an intriguing hypothesis that the mitochondrion might act as a cellular-stress sensor of Fe deficiency for the induction of the metabolic reprogramming of the cell. Therefore, it would be of interest to concentrate future research on understanding how Fe deficiency influences mitochondria and vice versa, that is, how mitochondrial remodelling facilitates the reprogramming of root cell metabolism.
Acknowledgments
I wish to thank Prof. Graziano Zocchi and Dr. Marta Dell’Orto for critically reading the manuscript. I also wish to thank the anonymous reviewers for their precious comments to improve the manuscript. The author was supported by “Dote Ricerca”: FSE, Regione Lombardia.

References

Abadía et al., 2002
J. Abadía, A.F. López-Millán, A. Rombolà, A. Abadía
Organic acids and Fe deficiency: a review
Plant Soil, 241 (2002), pp. 75-86
CrossRef View Record in Scopus

Abadía et al., 2011
Towards a knowledge-based correction of iron chlorosis

Aisen et al., 2001
P. Aisen, C. Enns, M. Wessling-Resnick
Chemistry and biology of eukaryotic iron metabolism
Article PDF (216KB) View Record in Scopus

Andaluz et al., 2009
S. Andaluz, J. Rodríguez-Celma, A. Abadía, J. Abadía, A.F. López-Millán
Time course induction of several key enzymes in Medicago truncatula roots in response to Fe deficiency
Plant Physiol Biochem, 47 (2009), pp. 1082-1088
Article PDF (286KB) View Record in Scopus

Andrews, 2008
N.C. Andrews
Forging a field: the golden age of iron biology
Blood, 112 (2008), pp. 219-230
CrossRef View Record in Scopus

Arimura et al., 2004
S. Arimura, G.P. Aida, M. Fujimoto, M. Nakazono, N. Tsutsumi
Arabidopsis dynamin-like protein 2a (ADL2a), like ADL2b, is involved in plant mitochondrial division
Plant Cell Physiol, 45 (2004), pp. 236-242
CrossRef View Record in Scopus

Arosio et al., 2009
P. Arosio, R. Ingrassi, P. Cavadini
Ferritins: a family of molecules for iron storage, antioxidation and more
Biochim Biophys Acta, 1790 (2009), pp. 589-599
Article PDF (626KB) View Record in Scopus

Balk and Lobréaux, 2005
J. Balk, S. Lobréaux
Biogenesis of iron–sulfur proteins in plants
Trends Plant Sci, 10 (2005), pp. 324-331
Article PDF (239KB) View Record in Scopus

Balk and Pilon, 2011
J. Balk, M. Pilon

**Ancient and essential: the assembly of iron–sulfur clusters in plants**

**Bashir et al., 2011**


**The rice mitochondrial iron transporter is essential for plant growth**

**Bertini and Rosato, 2007**

I. Bertini, A. Rosato

**From genes to metalloproteins: a bioinformatic approach**
Wiley-VCH Verlag (2007)
p. 2546–2555

**Briat et al., 2010a**

J.F. Briat, C. Duc, K. Ravet, F. Gaymard

**Ferritins and iron storage in plants**
Biochim Biophys Acta, 1800 (2010), pp. 806-814

**Buckhout et al., 2009**

T.J. Buckhout, T.J. Yang, W. Schmidt

**Early iron-deficiency-induced transcriptional changes in Arabidopsis roots as revealed by microarray analyses**
BMC Genomics, 10 (2009), p. 147

**Cesco et al., 2010**

S. Cesco, G. Neumann, N. Tomasi, R. Pinton, L. Weisskopf

**Release of plant borne flavonoids into the rhizosphere and their role in plant nutrition**

**Chen et al., 2010**


**Nitric oxide acts downstream of auxin to trigger root ferricchelate reductase activity in response to iron deficiency in Arabidopsis**
Plant Physiol, 154 (2010), pp. 810-819

**Conte and Walker, 2011**

S.S. Conte, E.L. Walker

**Transporters contributing to iron trafficking in plants**

---

*iris-AperTO*

University of Turin’s Institutional Research Information System and Open Access Institutional Repository
F.J. Corpas, J.M. Palma, L.A. Del Rio, J.B. Barroso
Evidence supporting the existence of l-arginine-dependent nitric oxide synthase activity in plants
New Phytol, 184 (2009), pp. 9-14
CrossRef View in Scopus
Correa-Aragunde et al., 2004
N. Correa-Aragunde, M. Graziano, L. Lamattina
Nitric oxide plays a central role in determining lateral root development in tomato
Planta, 218 (2004), pp. 900-905
CrossRef View in Scopus
Curie and Briat, 2003
C. Curie, J.F. Briat
Iron transport and signalling in plants
CrossRef View in Scopus
Curie et al., 2001
Maize yellow stripe1 encodes a membrane protein directly involved in Fe(III) uptake
CrossRef View in Scopus
De Domenico et al., 2008
I. De Domenico, D. McVey Ward, J. Kaplan
Regulation of iron acquisition and storage: consequences for iron-linked disorders
Nat Rev Mol Cell Biol, 9 (2008), pp. 72-81
CrossRef View in Scopus
De Nisi et al., 2010
P. De Nisi, G. Viganì, G. Zocchi
Modulation of iron responsive gene expression and enzymatic activities in response to changes of the iron nutritional status in Cucumis sativus L.
(2010)

De Nisi and Zocchi, 2000
P. De Nisi, G. Zocchi
Phoephophenolphpyruvate carboxylase in cucumber (Cucumis sativus L.) roots under iron deficiency: activity and kinetic characterization
View Record in Scopus
Dell’Orto et al., 2002
M. Dell’Orto, L. Pirovano, J.M. Villalba, J.A. Gonzalez-Reyes, G. Zocchi
Localization of the plasma membrane H+-ATPase in Fe-deficient cucumber roots by immunodetection
Plant Soil, 241 (2002), pp. 11-17
CrossRef View in Scopus
Dell’Orto et al., 2000
Development of Fe deficiency responses in cucumber (Cucumis sativus L.) roots: involvement of plasma membrane H+-ATPase activity
CrossRef View in Scopus
Donnini et al., 2010
S. Donnini, B. Prinsi, A.S. Negri, G. Vigani, L. Espen, G. Zocchi
Proteomic characterization of iron deficiency responses in Cucumis sativus L. roots
BMC Plant Biol, 10 (2010), p. 268
CrossRef

Donnini et al., 2011
S. Donnini, M. Dell’Orto, G. Zocchi
Oxidative stress responses and root lignifications induced by Fe deficiency condition in pear and quince genotype
Tree Physiol, 31 (2011), pp. 102-113
CrossRef

Douce and Neuberger, 1989
R. Douce, M. Neuberger
The uniqueness of plant mitochondria
CrossRef

Douce, 1985
R. Douce
Function of plant mitochondrial membranes; mitochondria in higher plants
p. 77–153

Ebbighausen et al., 1985
H. Ebbighausen, J. Chen, H.W. Heldt
Oxaloacetate translocator in plant mitochondria
Biochim Biophys Acta, 810 (1985), pp. 184-199
Article
PDF (1MB)
View Record in Scopus

Eckhardt et al., 2001
U. Eckhardt, A. Mas Marques, T.J. Buckhout
Two iron-regulated cation transporters from tomato complement metal uptake-deficient yeast mutants
Plant Mol Biol, 45 (2001), pp. 437-448
CrossRef
View Record in Scopus

Eide et al., 1996
D.J. Eide, M. Broderius, J. Fett, M.L. Guerinot
A novel iron-regulated metal transporter from plants identified by functional expression in yeast
Proc Natl Acad Sci USA, 93 (1996), pp. 5624-5628
CrossRef
View Record in Scopus

Eisenstein, 2000
R.S. Eisenstein
Iron regulatory proteins and the molecular control of mammalian iron metabolism
CrossRef
View Record in Scopus

Espen et al., 2000
L. Espen, M. Dell’Orto, P. De Nisi, G. Zocchi
Metabolic responses in cucumber (Cucumis sativus L.) roots under Fe-deficiency: a 31P-nuclear magnetic resonance in vivo study
CrossRef
Finkemeier and Sweetlove, 2006
I. Finkemeier, L.J. Sweetlove
**Signalling in primary metabolism**

CrossRef
Fujimoto et al., 2004
M. Fujimoto, S. Arimura, M. Nakazono, N. Tsutsumi
**A rice dynamin-like protein, OsDRP3A, is involved in mitochondrial fission**

CrossRef
Galaris and Pantopoulos, 2008
D. Galaris, K. Pantopoulos
**Oxidative stress and iron homeostasis: mechanistic and health aspects**

CrossRef
Gonzalez-Vallejo et al., 1998
E.B. Gonzalez-Vallejo, S. Susín, A. Abadía, J. Abadía
**Changes in sugar beet leaf plasma membrane Fe(III)-chelate reductase activities mediated by Fe deficiency, assay buffer composition, anaerobiosis and the presence of flavins**
Protoplasma, 205 (1998), pp. 163-168

CrossRef
Gray et al., 2004
G.R. Gray, D.P. Maxwell, A.R. Villarimo, L. McInotosh
**Mitochondria/nuclear signalling of alternative oxidase gene expression occurs through distinct pathways involving organic acids and reactive oxygen species**
Plant Cell Rep, 23 (2004), pp. 497-503

CrossRef
Graziano and Lamattina, 2007
M. Graziano, L. Lamattina
**Nitric oxide accumulation is required for molecular and physiological responses to iron deficiency in tomato roots**
Plant J, 52 (2007), pp. 949-960

CrossRef
Gupta et al., 2010
K.J. Gupta, A.U. Igamberdiev, W.M. Kaiser
**New insights into the mitochondrial nitric oxide production pathways**
Plant Signal Behav, 5 (2010), pp. 999-1001

CrossRef
Gupta and Igamberdiev, 2011
K.J. Gupta, A.U. Igamberdiev
**The anoxic plant mitochondrion as a nitrite: NO reductase**
Mitochondrion, 11 (2011), pp. 537-543

Article
PDF (513KB)

Halliwell, 2006
B. Halliwell
**Reactive species and antioxidants. Redox biology is a fundamental theme of aerobic life**
Plant Physiol, 141 (2006), pp. 312-322
Hentze et al., 2010
M.W. Hentze, M.U. Muckenthaler, B. Galy, C. Camaschella
Two to tango: regulation of mammalian iron metabolism
Cell, 142 (2010), pp. 24-38
ArticlePDF (2MB) View Record in Scopus

Higa et al., 2010
A. Higa, Y. Mori, Y. Kitamura
Iron deficiency induces changes in riboflavin secretion and the mitochondrial electron transport chain in hairy roots of Hyoscyamus albus
ArticlePDF (916KB) View Record in Scopus

Hu et al., 2008
J. Hu, L. Dong, C.E. Outten
The redox environment in the mitochondrial intermembrane space is maintained separately from the cytosol and matrix
CrossRef View Record in Scopus

Igamberdiev and Gardeström, 2003
A.U. Igamberdiev, P. Gardeström
Regulation of NAD- and NADP-dependent isocitrate dehydrogenases by reduction levels of pyridine nucleotides in mitochondria and cytosol of pea leaves
Biochim Biophys Acta, 1606 (2003), pp. 117-125
ArticlePDF (236KB) View Record in Scopus

Jelali et al., 2010
N. Jelali, W. M’sehli, M. Dell’Orto, C. Abdelly, M. Gharsalli, G. Zocchi
Changes of metabolic responses to direct and induced Fe deficiency of two Pisum sativum cultivars
Environ Exp Bot, 68 (2010), pp. 238-246
ArticlePDF (1MB) View Record in Scopus

Jin et al., 2009
Elevated carbon dioxide improves plant Fe nutrition through enhancing the Fe-deficiency-induced responses under Fe-limited conditions in tomato
Plant Physiol, 150 (2009), pp. 272-280
CrossRef View Record in Scopus

Journet et al., 1981
E.P. Journet, M. Neuburger, R. Douce
The role of glutamate oxaloacetate transaminase and malate dehydrogenase in the regeneration of NAD+ for glycine oxidation by spinach leaf mitochondria
CrossRef View Record in Scopus

Kannan, 1988
S. Kannan
Physiological-responses associated with Fe-deficiency stress in different plant species
CrossRef View Record in Scopus

Kell, 2009
D.B. Kell
Iron behaving badly: inappropriate iron chelation as a major contributor to the aetiology of vascular and other progressive inflammatory and degenerative diseases
BMC Med Genomics, 2 (2009), p. 2
View Record in Scopus
Koppenol, 1993
W.H. Koppenol
The centennial of the Fenton reaction
ArticlePDF (596KB)View Record in Scopus
Kushnir et al., 2001
A mutation of the mitochondrial ABC transporter Sta1 leads to dwarfism and chlorosis in the Arabidopsis mutant starik
Plant Cell, 13 (2001), pp. 89-100
CrossRefView Record in Scopus
Lan et al., 2011
iTRAQ protein profile analysis of Arabidopsis roots reveals new aspects critical for iron homeostasis
CrossRefView Record in Scopus
Landsberg, 1986
E.C. Landsberg
Function of rhizodermal transfer cells in the Fe stress response mechanism of Capsicum annuum L.
Plant Physiol, 82 (1986), pp. 511-517
CrossRefView Record in Scopus
Landsberg, 1994
E.C. Landsberg
Transfer cell formation in sugar beet roots induced by latent Fe deficiency
Plant Soil, 165 (1994), pp. 197-205
CrossRefView Record in Scopus
Li et al., 2008
J. Li, X. Wu, S. Hao, X. Wang, H. Ling
Proteomic response to iron deficiency in tomato root
Proteomics, 8 (2008), pp. 2299-2311
CrossRefView Record in Scopus
Logan, 2006
D.C. Logan
The mitochondrial compartment
CrossRefView Record in Scopus
Logan, 2010
D.C. Logan
Mitochondrial fusion, division and positioning in plants
CrossRefView Record in Scopus
Logan and Knight, 2003
D.C. Logan, M.R. Knight
Mitochondrial and cytosolic calcium dynamics are differentially regulated in plants
Plant Physiol, 133 (2003), pp. 21-24
CrossRef View Record in Scopus
Logan et al., 2004
D.C. Logan, I. Scott, A.K. Tobin
ADL2a, like ADL2b, is involved in the control of higher plant mitochondrial morphology
CrossRef View Record in Scopus
López-Millán et al., 2000a
Responses of sugar beet roots to iron deficiency. Changes in carbon assimilation and oxygen use
CrossRef
López-Millán et al., 2000b
A.F. López-Millán, F. Morales, A. Abadia, J. Abadia
Effects of iron deficiency on the composition of the leaf apoplastic fluid and xylem sap in sugar beet. Implications for iron and carbon transport
CrossRef View Record in Scopus
López-Millán et al., 2009
A.F. López-Millán, F. Morales, Y. Gogorcena, A. Abadía, J. Abadía
Metabolic responses in iron deficient tomato plants
J Plant Physiol, 166 (2009), pp. 375-384
Article PDF (256KB) View Record in Scopus
M'Sehli et al., 2008
W. M'Sehli, M. Dell’Orto, S. Donnini, P. De Nisi, G. Zocchi, C. Abdelly, et al.
Variability of metabolic responses and antioxidant defence in two lines of Medicago ciliaris to Fe deficiency
Plant Soil, 320 (2008), pp. 219-230
Mackenzie and Mcintosh, 1999
S. Mackenzie, L. Mcintosh
Higher plant mitochondria
CrossRef
Malinardi et al., 2011
M.V. Malinardi, M.V. Busi, V.R. Turowski, L. Leaden, A. Araya, D.F. Gomez-Casati
The mitochondrial protein frataxin is essential for heme biosynthesis in plants
FEBS J, 278 (2011), pp. 470-481
Martin and Russell, 2003
W. Martin, M.J. Russell
On the origin of the cells: a hypothesis for the evolutionary transitions from abiotic geochemistry to chemoautotrophic prokaryotes, and from prokaryotes to nucleated cells
View Record in Scopus
Mendez-Bravo et al., 2010
A. Mendez-Bravo, J. Raya-Gonzalez, L. Herrera-Esresta, L. Lopez-Bucio
Nitric oxide is involved in alkamide-induced lateral root development in Arabidopsis
Plant Cell Physiol, 51 (2010), pp. 1612-1626
CrossRef View Record in Scopus
Millar et al., 2011
A.H. Millar, J. Whelan, K.L. Soole, D.A. Day
Organization and regulation of mitochondrial respiration in plants
CrossRef View Record in Scopus
Møller et al., 2011
I.M. Møller, A. Rogowska-Wrzesinska, R.S.P. Rao
Protein carbonylation and metal-catalyzed protein oxidation in a cellular perspective
J Prot (2011), 10.1016/j.jprot.2011.05.004
Møller and Sweetlove, 2010
I.M. Møller, L.J. Sweetlove
ROS signalling-specificity is required
ArticlePDF (229KB)View Record in Scopus
Møller, 2001
I.M. Møller
Plant mitochondria and oxidative stress: electron transport, NADPH turnover, and metabolism of reactive oxygen species
CrossRef View Record in Scopus
Mukherjee et al., 2006
I. Mukherjee, N. Campbell, J. Ash, E. Connolly
Expression profiling of the Arabidopsis ferric chelate reductase (FRO) gene family reveals differential regulation by iron and copper
Planta, 223 (2006), pp. 1178-1190
CrossRef View Record in Scopus
Nagarajah and Ulrich, 1966
S. Nagarajah, A. Ulrich
Iron nutrition of the sugar beet plant in relation to growth, mineral balance, and riboflavin formation
Soil Sci, 102 (1966), pp. 399-407
CrossRef View Record in Scopus
Neil et al., 2002
S. Neil, R. Desikan, J. Hancock
Hydrogen peroxide signalling
Nouet et al., 2011
C. Nouet, P. Motte, M. Hanikenne
Chloroplastic and mitochondrial metal homeostasis
ArticlePDF (441KB)View Record in Scopus
Pascal and Douce, 1993
N. Pascal, R. Douce
Effect of iron deficiency on the respiration of sycamore (*Acer pseudoplatanus* L.)
calls
Plant Physiol, 103 (1993), pp. 1329-1338
CrossRefView Record in Scopus
Picault et al., 2002
N. Picault, L. Palmieri, L. Pisano, M. Hodges, F. Palmieri
Identification of a novel transporter for dicarboxylates and tricarboxylates in plant
mitochondria. Bacterial expression, reconstitution, functional characterization, and
tissue distribution
CrossRefView Record in Scopus
Picault et al., 2002
N. Picault, L. Palmieri, L. Pisano, M. Hodges, F. Palmieri
Identification of a novel transporter for dicarboxylates and tricarboxylates in plant
mitochondria. Bacterial expression, reconstitution, functional characterization, and
tissue distribution
CrossRefView Record in Scopus
Poolman et al., 2009
M.G. Poolman, L. Miguet, L.J. Sweetlove, D.A. Fell
A genome-scale metabolic model of *Arabidopsis* and some of its properties
Plant Physiol, 151 (2009), pp. 1570-1581
CrossRefView Record in Scopus
Rabotti et al., 1995
G. Rabotti, P. De Nisi, G. Zocchi
Metabolic implications in the biochemical responses to iron deficiency in cucumber
(*Cucumis sativus* L.) roots
CrossRefView Record in Scopus
Rabotti and Zocchi, 1994
G. Rabotti, G. Zocchi
Plasma membrane-bound H+-ATPase and reductase activities in Fe-deficient
cucumber roots
CrossRefView Record in Scopus
Rasmusson and Wallström, 2010
A.G. Rasmusson, S.V. Wallström
Involvement of mitochondria in the control of plant cell NAD(P)H reduction levels
CrossRefView Record in Scopus
Rellán-Álvarez et al., 2010
R. Rellán-Álvarez, S. Andaluz, J. Rodríguez-Celma, G. Wohlgrmuth, G. Zocchi, A.
Álvarez-Fernández, *et al.*
Changes in the proteomic and metabolic profiles of *Beta vulgaris* root tips in
response to iron deficiency and resupply
BMC Plant Biol, 10 (2010), pp. 120-134
CrossRef
Rhoads et al., 2006
D.M. Rhoads, A.L. Umbach, C.C. Subbaiah, J.N. Siedow
Mitochondrial reactive oxygen species. Contribution to oxidative stress and
interorganellar signalling
Robinson et al., 1999
N.J. Robinson, C.M. Procter, E.L. Connolly, M.L. Guerinot
A ferric-chelate reductase for iron uptake from soils

Rodriguez-Celma et al., 2011
J. Rodríguez-Celma, G. Lattanzio, M.A. Grusak, A. Abadía, J. Abadía, A.F. López-Millán
Root responses of Medicago truncatula plants grown in two different iron deficiency conditions: changes in root protein profile and riboflavin biosynthesis
J Proteome Res, 10 (2011), pp. 2590-2601

Santi et al., 2005
S. Santi, S. Cesco, Z. Varanini, R. Pinton
Two plasma membrane H+ -ATPase genes are differentially expressed in iron-deficient cucumber plants
Plant Physiol Biochem, 43 (2005), pp. 287-292

Santi and Schmidt, 2009
S. Santi, W. Schmidt
Dissecting iron deficiency-induced proton extrusion in Arabidopsis roots
New Phytol, 183 (2009), pp. 1072-1084

Schagerlof et al., 2006
U. Schagerlof, G. Wilson, H. Hebert, S. Al-Karadaghi, C. Hagerhall
Transmembrane topology of FRO2, a ferric chelate reductase from Arabidopsis thaliana

Scheibe, 2004
R. Scheibe
Malate valves to balance cellular energy supply
Physiol Plant, 120 (2004), pp. 21-26

Schmidt and Schuck, 1996
W. Schmidt, C. Schuck
Pyridine nucleotide pool size changes in iron-deficient Plantago lanceolata roots during reduction of external oxidants
Physiol Plant, 98 (1996), pp. 215-221

Schmidt, 1999
W. Schmidt
Mechanism and regulation of reduction-based iron uptake in plants
New Phytol, 141 (1999), pp. 1-26
View Record in Scopus
Shapiro, 2005
A.D. Shapiro
Nitric oxide signaling in plants
Vitam Horm, 72 (2005), pp. 339-398
ArticlePDF (520KB)View Record in Scopus
Shen et al., 2006
Involvement of a glycerol-3-phosphate dehydrogenase in modulating the NADH/NAD1 ratio provides evidence of a mitochondrial glycerol-3-phosphate shuttle in Arabidopsis
Plant Cell, 18 (2006), pp. 422-441
CrossRefView Record in Scopus
Shen et al., 2003
W. Shen, Y. Wei, M. Dauk, Z. Zheng, J. Zou
Identification of a mitochondrial glycerol-3 phosphate dehydrogenase from Arabidopsis thaliana: evidence for a mitochondrial glycerol-3 phosphate shuttle in plants
ArticlePDF (311KB)CrossRefView Record in Scopus
Sijmons and Bienfait, 1983
P.C. Sijmons, H.F. Bienfait
Source of electrons for extracellular Fe(III) reduction in iron-deficient bean plants
Physiol Plant, 59 (1983), pp. 409-415
CrossRefView Record in Scopus
Sijmons et al., 1984
P.C. Sijmons, W. Van Den Briel, H.F. Bienfait
Cytosolic NADPH is the electron donor for extracellular Felll reduction in iron-deficient bean roots
Plant Physiol, 75 (1984), pp. 219-221
CrossRefView Record in Scopus
Slatni et al., 2011
Metabolic changes of iron uptake in N2-fixing common bean nodules during iron deficiency
ArticlePDF (689KB)View Record in Scopus
Steuer et al., 2007
From structure to dynamics of metabolic pathways: application to the plant mitochondrial TCA cycle
Bioinformatics, 23 (2007), pp. 1378-1385
CrossRefView Record in Scopus
Subbaiah et al., 1994
C.C. Subbaiah, D.S. Bush, M.M. Sachs
Elevation of cytosolic calcium precedes anoxic gene expression in maize suspension-cultured cells
Plant Cell, 6 (1994), pp. 1747-1762
CrossRefView Record in Scopus
Sweetlove et al., 2010
Not just a circle: flux modes in the plant TCA cycle
ArticlePDF (639KB) View Record in Scopus

Sweetlove et al., 2007
L.J. Sweetlove, A. Fait, A. Nunes-Nesi, T. Williams, A.R. Fernie
The mitochondrion: an integration point of cellular metabolism and signalling
CrossRef View Record in Scopus

Tan et al., 2010
Y.F. Tan, N. O’Toole, N.L. Taylor, A.H. Millar
Divalent metal ions in plant mitochondria and their role in interactions with proteins and oxidative stress-induced damage to respiratory function
CrossRef View Record in Scopus

Tarantino et al., 2010a
D. Tarantino, F. Casagrande, C. Soave, I. Murgia
Knocking out of the mitochondrial AtFer4 ferritin does not alter response of Arabidopsis plants to abiotic stresses
ArticlePDF (488KB) View Record in Scopus

Tarantino et al., 2010b
AtFer4 ferritin is a determinant of iron homeostasis in Arabidopsis thaliana heterotrophic cells
ArticlePDF (1MB) View Record in Scopus

Thimm et al., 2001
O. Thimm, B. Essigmann, S. Kloska, T. Altmann, T.J. Buckhout
Response of Arabidopsis to iron deficiency stress as revealed by microarray analysis
CrossRef View Record in Scopus

Tiffin, 1966
L.O. Tiffin
Iron translocation. I. Plant culture, exudate sampling, iron–citrate analysis
CrossRef View Record in Scopus

Tomasi et al., 2008
Flavonoids of white lupin roots participate in phosphorus mobilization from soil
ArticlePDF (320KB) View Record in Scopus

Viganì et al., in press

Viganì et al., 2009
G. Viganì, D. Maffi, G. Zocchi

---

iris-AperTO
University of Turin’s Institutional Research Information System and Open Access Institutional Repository
Iron availability affects the function of mitochondria in cucumber root
New Phytol, 182 (2009), pp. 127-136
CrossRef View Record in Scopus
Vigani and Zocchi, 2010
G. Vigani, G. Zocchi

Effect of Fe deficiency on mitochondrial alternative NAD(P)H dehydrogenases in cucumber roots
Article PDF (565KB) View Record in Scopus
Vigani and Zocchi, 2009
G. Vigani, G. Zocchi

The fate and the role of mitochondria in Fe-deficient roots of Strategy I plants
Plant Signal Behav, 5 (2009), pp. 375-379
CrossRef View Record in Scopus
von Wiren et al., 1999

Nicotianamine chelates both FeIII and FeII. Implications for metal transport in plants
CrossRef View Record in Scopus
Wang et al., 2010

Nitric oxide is involved in phosphorus deficiency induced cluster-root development and citrate exudation in white lupin
CrossRef View Record in Scopus
Waters et al., 2002
B.M. Waters, D.G. Blevins, D.J. Eide
Characterization of FRO1, a pea ferricchelate reductase involved in root iron acquisition
CrossRef View Record in Scopus
Waters et al., 2007

Ethylene involvement in the regulation of H+-ATPase CsHA1 gene and of the new isolated ferric reductase CsFRO1 and iron transporter CsIRT1 genes in cucumber plants
Plant Physiol Biochem, 45 (2007), pp. 293-301
Article PDF (1MB) View Record in Scopus
Welkie et al., 1990
G.W. Welkie, H. Heckmat-Shoar, G.W. Miller

Responses of pepper (Capsicum annum L.) plants to iron deficiency: solution pH and riboflavin
M. van Beusichem (Ed.), Plant nutrition—physiology and applications, Kluwer, Dordrecht (1990), pp. 207-211
CrossRef
Welkie and Miller, 1993
G.W. Welkie, G.W. Miller

Plant iron uptake physiology by nonsiderophore systems
Wendehenne et al., 2004
D. Wendehenne, J. Durner, D.F. Klessig
Nitric oxide: a new player in plant signalling and defence responses
ArticlePDF (211KB)View Record in Scopus

Zancani et al., 2004
Evidence for the presence of ferritin in plant mitochondria
CrossRefView Record in Scopus

Zocchi, 2006
G. Zocchi
Metabolic changes in iron-stressed dicotyledonous plants
CrossRefView Record in Scopus

Zocchi et al., 2007
G. Zocchi, P. De Nisi, M. Dell’Orto, L. Espen, P. Marino Gallina
Iron deficiency differently affects metabolic responses in soybean roots