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Iron deprivation induces a rapid increase of the sulfate uptake and accumulation in tomato (*Solanum lycopersicum* L.) seedlings

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The mechanism of Fe uptake in tomato and all other plants except grasses, involves the mobilization of Fe^{3+} ions from soil particles by rhizosphere acidification, likely driven by an increase in plasma membrane H^+ATPase activity, the induction of the Fe^{3+}-chelate reductase activity, which allows higher reduction rate of Fe^{3+} to Fe^{2+}, and the uptake of the resulting Fe^{2+} via an Fe^{2+} transporter (Strategy I). Previous work has focused on characterising the relationship between S nutrition and Fe acquisition, since methionine is required for the synthesis of S-adenosylmethionine (SAM), which is a common precursor of molecules as nicotianamine (NA) and ethylene. NA is a key molecule for long-distance transport of Fe in plants and ethylene is likely involved in the regulation of the Fe deficiency responses in Strategy I plants. Although it is well known that an adequate sulfur availability, allowing an adequate level of methionine and its derivatives, could alleviate Fe-deficiency effects in Strategy I plants, it remains unclear how S homeostasis is regulated in response to Fe deficiency in the same plants.

In the present study we investigated the time course of the activation of physiological responses to Fe deficiency in tomato roots. Measurements included plant growth, leaf chlorosis status, leaf Fe concentration and root ferric chelate reducing activity. Furthermore, we followed the development of Fe-deficiency stress response through the analysis of expression of *SlFRO1*, *SlIRT1* and *SlFER* genes. Finally, we investigated the dynamics of total S and thiols accumulation and allocation between roots and shoots, and the effect of Fe deprivation on the root sulfate uptake and mobilization capacity, by analysing the expression of genes encoding sulfate transporters (STs) of Groups 1, 2 and 4 (*SlST1.1*, *SlST1.2*, *SlST2.1*, *SlST2.2*, *SlST4.1*).

When tomato plants were grown in a iron-free solution, the activation of iron deficiency stress response occurred in a progressive manner via increased root ferric chelate reductase activity and the induction of several iron-responsive genes as *SlFRO1*, *SlIRT1* and *SlFER*. Most of the tomato ST genes belonging to Groups 1, 2 and 4 were significantly up-regulated in iron-deficient roots, as it commonly occurs under S-deficient conditions. The up-regulation of the two high affinity ST genes, Sl*ST1.1* and *SlST1.2*, by iron deprivation clearly suggests an increased root capability to take up sulfate. Furthermore, the up-regulation of the two low affinity ST genes *SlST2.1* and *SlST4.1* in iron-deficient roots, accompanied by a substantial accumulation of total sulfur and thiols in shoots of iron-starved plants, likely supports an increased root-to-shoot translocation of sulfate.

In conclusion, it appears that tomato plants exposed to Fe deficiency are able to change sulfur metabolic balance mimicking sulfur starvation responses through an increased sulfate uptake and root-to-shoot translocation to meet the increased demand for ethylene and NA, allowing them to cope with this stress. Furthermore, transcriptional analysis of iron-responsive genes, *SlFRO1*, Sl*IRT1* and Sl*FER*, carried out at regular intervals during the induction of Fe deficiency shows that, although they were strongly and rapidly up-regulated by Fe deprivation, their transcription levels did not follow exactly the same trend over time, suggesting that *LeFER* likely regulates *LeFRO1* more directly than *LeIRT1*.

Parole chiave: ferro, pomodoro, Strategia I, trasportatori del solfato