UNDERSTANDING ARABIDOPSIS ION HOMEOSTASIS IN THE POST-GENOMIC ERA: ASSIGNING FUNCTION TO TWO PROTEINS INVOLVED IN IRO METABOLISM

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ABSTRACT

Two projects studying different aspects of iron deficiency in the model plant *Arabidopsis thaliana* are detailed here. The first project describes the isolation and characterization of the Arabidopsis *frd4-1* and *frd4-2* mutants that do not induce Fe(III) chelate reductase activity in their roots in response to iron deficiency. Map-based cloning revealed that the *frd4* mutations reside in *cpFtsY*, which encodes a component of one of the pathways responsible for the insertion of proteins into the thylakoid membranes of the chloroplast. A number of different hypotheses were tested in an attempt to explain how defects in *cpFtsY* could affect the expression of root Fe(III) chelate reductase activity. The second project involves the further characterization of the protein FRD3, which was previously shown to be important for the efficient translocation of iron from roots to the shoots. Xylem exudate from *frd3* plants contains significantly less citrate and iron than the exudate from wild type plants. Additionally, supplementation of growth media with citrate rescues the *frd3* phenotypes. The ectopic expression of FRD3-GFP results in enhanced exudation of citrate from roots. Finally, heterologous studies in *Xenopus laevis* oocytes reveal that FRD3 mediates the transport of citrate. These results strongly support the hypotheses that FRD3 effluxes citrate into the root vasculature, a process important for the translocation of iron to the leaves, and that iron moves through the xylem as a ferric-citrate complex.