Timothy Pouland, Biochemistry

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Faculty Mentor: Dr. Elizabeth Rogers, Nutritional Sciences
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Screening for suppressors of frd3-3 mutation in *Arabidopsis*

Iron is an essential nutrient for plants. It is required for a number of reactions in cells including chlorophyll biosynthesis. Plants that are iron deficient tend to show a chlorotic phenotype. Most iron in soils is iron(III), which is highly insoluble at neutral or basic pH. Since only water-soluble atoms and molecules are available for uptake by plants, plant need to reduce iron(III) in soils to the more water-soluble form, iron(II). Ferric chelate reductase, which is localized in the roots, catalyzes the reduction of iron(III) to iron(II) when iron is needed by the plant. frd3-3 mutants are slightly chlorotic, but have been found to have high amounts of iron in the leaves, indicating possible mislocalization of iron. These mutants also show constitutive ferric chelate reductase activity. This is most likely due to the FRD3 protein being involved in iron homeostasis. However, the exact role of FRD3 in iron homeostasis is still under investigation. In this study, plants containing a frd3-3 mutation have been mutagenized with EMS in hopes of recovering a wild-type phenotype by mutation of a separate gene coding for a protein also involved in iron transport. Studies involve growing M2 plants on B5 media, transferring the least chlorotic plants to iron rich media, and performing ferric chelate reductase assays. Plants that appear to have normal reductase activity are then grown in soil for further analysis of the M3 generation. To date I have screened over 6,000 M2 generation plants and identified 20 putative mutants. Currently, these putative mutants are being rescreened in the M3 generation, along with continued M2 screening. The result of this study will be the identification of additional proteins that work with FRD3 in iron localization, which will lead to a better understanding iron homeostasis in plants.