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FRATAXIN NULL MUTANTS OF ARABIDOPSIS ARE EMBRYO LETHALS

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Frataxin is a nuclear encoded protein targeted to the mitochondrial matrix. In humans, frataxin deficiency is associated with Friedreich's ataxia, a neurodegenerative and cardiac disorder characterized by accumulation of iron in the mitochondria and a diminished activity of various mitochondrial proteins, including aconitase. Yeast cells lacking frataxin show a complex respiratory deficient phenotype, defective in the maturation of mitochondrial Fe/S enzymes, hypersensitivity to oxidative stress, instability of mtDNA and defects in heme biosynthesis. It has been proposed that frataxin has ferroxidase activity and iron storage properties which may protect the mitochondria from iron toxicity, and that it also acts as a chaperone to donate iron to the proteins involved in the two major pathways of iron utilization, Fe/S cluster assembly and heme synthesis. Recently, an Arabidopsis gene (AtFH) highly similar to the human frataxin gene and possessing a mitochondrial targeting sequence has been described (Busi et al. 2004): AtFH is able to complement a yeast frataxin null mutant and in the plant is mainly expressed in flowers. We identified in the Salk collection two T-DNA insertions in the AtFH gene and characterized genetically the two mutants. Upon selfing heterozygous plants, we cannot recover in the progeny homozygote null seeds while homozygous wt and heterozygous seeds were in a ratio 1: 2 (as observed after PCR analysis). The ratio was consistent with lethality of the homozygous null genotypes during embryogenesis. Accordingly we analyze the pattern of embryo development in siliques segregating homozygous null embryos: an early arrest at the 8-16 cells stage was consistently observed.

Busi M. et al. FEBS Letters 576, 141-144, 2004.