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Mutation analysis in *Arabidopsis thaliana* reveals a new gene locus regulating the uptake of uracil

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A fluoroorotic acid (FOA)-resistant mutant was isolated in a screen of a mutagenized population of *Arabidopsis thaliana.* Genetic analysis revealed that FOA resistance was due to a nuclear recessive gene, *for1-1*. Assay of key regulatory enzymes of the *de novo* synthesis and salvage of pyrimidines as well as uptake studies using radiolabelled purines and pyrimidines showed that *for1-1/for1-1* plants were specifically defective in the uptake of uracil or uracil-like bases. Genetically-engineering FOA-resistant *for1-1/for1-1* plants with the uracil transporter gene *uraA* of *E. coli* restored FOA sensitivity to the transformed plants confirming the specific role of FOR1 in regulating uracil uptake. Genetic mapping of *for1-1* placed the new locus in chromosome five. Molecular mapping using CAPS (co-dominant amplified polymorphic site) markers placed *FOR1* in a region of chromosome 5 that does not correspond to loci belonging to any of the six known nucleobase transporter families identified in the *Arabidopsis* genome. This suggests that *FOR1* may be encoding a regulatory protein (transcription factor) that is involved in regulating the uptake of uracil. Chromosome walking experiments, using PCR polymorphic deletion markers, resulted in fine tune mapping of *FOR1* to an area of ~500 kb which includes several putative transcription factors. Testing *Arabidopsis* mutants, with insertion/knock out mutations in such putative genes, for FOA-resistance should identify the nature of the *FOR1* gene product and reveal the precise mechanism behind regulating the uptake and recycling of uracil within cell compartments and between cells.