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IMPORT OF MACROMOLECULES IN PLANT MITOCHONDRIA

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The plant cell has a particular feature to contain three instead of two compartments where translation events take place: the cytosol, mitochondria and chloroplasts. To be active, each of these translational machineries must possess complete sets of aminoacyl-tRNA synthetases (aaRS) and tRNAs. During evolution, chloroplast and mitochondrial genomes of plants have lost a number of corresponding genes. All aaRS are now encoded in the nucleus and imported into the organelles. Using in silico, analyses and in vivo and in vitro experiments, we have shown that dual targeting is the rule for organellar aaRS. This situation represents an unprecedented degree of cross-compartment sharing of aaRS that has occured during evolution.

All chloroplastic tRNAs are encoded in plastid DNA. In contrast, the mitochondrial population of tRNA is much more complex. MtDNA-encoded tRNAs characterized by typically mitochondrial features (called "native") are not sufficient to decode all 61 codons of the genetic code and mitochondria uses two other sources to complete the tRNA set. One portion of missing tRNAs is replaced by expressed tRNA genes carried by chloroplast DNA fragment inserted into the mitochondrial genome during evolution. The second one is provided by import of nuclear DNA encoded tRNAs from the cytosol. At least one third of the mitochondrial tRNAs of higher plants are nuclear-encoded and imported into mitochondria. This phenomenon is not plant specific and the import of tRNAs encoded in the nucleus is essential for proper protein translation within mitochondria of a variety of evolutionary distant organisms. However, in spite of a broad occurence of this pathway among organisms, mechanisms governing RNA targeting into mitochondria remain poorly understood. By developing in vivo approaches, biochemical studies and an *in vitro* tRNA import system, we are trying to elucidate the mechanism involved in plants. Moreover, in an attempt to study tRNA stability once internalized into the organelles, we have developed a strategy based on direct DNA uptake into the mitochondria, this strategy will be presented.

As a whole, our recent data will be compared to those obtained in other systems. Besides the fundamental importance of a novel mechanism of intracellular targeting, the results expected might be exploited in the future to develop an original system of complementation of pathogenic mutations in human mitochondrial DNA.