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# Protein Mistranslation In Endosymbiotic Organelles Of Plant Cells

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Accurate protein translation is a hallmark for cell function. It guarantees an efficient proteome while minimizing detrimental unfolded proteins and concomitant energy loss. Surprisingly, protein mistranslation happens quite frequently in vivo (error rates 10-2 to 10-4) mainly due to transfer RNA (tRNA) mis-decoding and tRNA mis-acylation. Bacteria, mitochondria and plastids synthesize glutaminyl-tRNAs (Gln-tRNAGln) via an indirect pathway. Initially, an unspecific aminoacyl-tRNA synthetase charges tRNAGln with glutamate. Subsequently, glutamine is produced through trans-amidation by the aminoacyl-tRNA amido-transferase complex GatCAB. Bacteria can vary GatCAB activity. The resulting mistranslation increases proteome plasticity helping them to withstand adverse conditions. Conversely, fungi and animal mitochondria are highly sensitive to amino acid misincorporation. The role of GatCAB in plant organelles and the plant response to mistranslation is unknown. Our study of the Arabidopsis gatb-1 mutant provides global insights into GatCAB function in plants. Proteomics revealed varying degrees of Gln-to-Glu misincorporation in mutant plastid- and mitochondrially-expressed protein complexes of up to >90%. It appears that there are differences in nuclear, plastid, and mitochondrial control of mistranslation. Through transcriptomics and biochemical assays, we identified efficient compensatory mechanisms that yield only modest abundance changes of the steady-state plastid proteome and explain the surprisingly subtle phenotypes of gatb-1 plants. Hence, the gatb-1 mutant represents a premiere tool to dissect mistranslation effects in plant organelles, study their global effects, and pinpoint critical players in proteostasis and protein quality control events.

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