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BMS

Judging Dept.

Venkata Chalamcharla

Student

BMS

4

Marlene Belfort

Dept or Program Years in program

Mentor

A bacterial Group II intron as a substrate for nonsense-mediated mRNA decay in yeast

Author (s)

Venkata Chalamcharla, Marlene Belfort

Group II introns are self-splicing ribozymes capable of invading DNA, as mobile elements. They have been invoked as the progenitors of nuclear spliceosomal introns, and in nucleus-cytosol compartmentalization. However, there is no evidence for the functioning of group II introns outside of mitochondria and chloroplasts, and in the nucleus. Here, we show that the *Lactococcus lactis* Ll.LtrB group II intron transcribed within the eukaryotic nucleus in *Saccharomyces cerevisiae* is able to splice. RNA analysis showed that the splicing of Ll.LtrB is not only dependent on its encoded protein, but is also accurate and efficient in *S. cerevisiae*. We next developed a CUP1-based phenotypic assay that measures Ll.LtrB expression based on the level of copper-resistance conferred. Surprisingly, the CUP1-based splicing-phenotype was undetectable. In an attempt to restore the splicing-phenotype, we tested the role of nonsense-mediated mRNA-decay (NMD) as a quality-control mechanism in determining the fate of Ll.LtrB-transcript in yeast. The absence of UPF1p, a RNA-dependant ATPase and 5'-to-3' helicase essential for NMD, partially restored the group II intron CUP1 phenotype in yeast. These findings suggest that the absence of group II introns from nuclear genomes resulted from the eukaryotic surveillance systems such as NMD. Further, we suggest that such mRNA-decay mechanisms might have fragmented the group II introns, forcing their establishment as genes-in-pieces, the eukaryotic spliceosomal introns.