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**A conjugative plasmid promotes retrotransposition of a group II intron and increases dissemination during cell stress and DNA damage**

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Group II introns are mobile genetic elements that invade their cognate intron-minus gene via an RNA intermediate in a process known as retrohoming. They can also retrotranspose to ectopic sites at low frequency. Retrotransposition of the Ll.LtrB group II intron of *Lactococcus lactis* primarily targets the template for lagging-strand DNA synthesis, which suggests the utilization of the replication fork as a source of single-stranded DNA (ssDNA). To date, ~35% of bacterial group II introns have been shown to reside on other mobile genetic elements, which may aid in dissemination. The Ll.LtrB group II intron was originally discovered in the pRS01 conjugative plasmid of *L. lactis* in the *ltrB* gene, which encodes a conjugative relaxase. LtrB initiates conjugation by nicking the DNA at the origin of transfer (*oriT*) sequence. Splicing of the intron is necessary to produce an intact, functional LtrB relaxase, thus allowing conjugative strand transfer. First, it was shown that retrotransposition of Ll.LtrB was elevated in the presence of an autonomously replicating pRS01. Second, retrotransposition was examined following treatment with the DNA damage agent mitomycin C. Interestingly, retrotransposition is inhibited in the absence of a chromosomally-encoded *ltrB* when cells are treated by mitomycin C. These results indicate that *ltrB* enhances retrotransposition during times of cell stress and DNA damage, and together with elevation of mobility by pRS01, implicates conjugation as a possible facilitator of group II intron mobility.