Bruno is a host co-factor that establishes natural variation in P-element transposition

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Abstract

Transposable elements recurrently invade new host genomes through horizontal transfer, often between distantly related species. These horizontal transfer events are characterized by a burst of transposition in the naive host genome, followed by the evolution of piRNA mediated silencing that brings the invader under host control. However, lost in this story of evolving host defense is the initial requirement for positive regulation of the TE by the host genome, in the form of host-encoded factors that promote the transcription, splicing and translation of TE derived mRNA. Variation in these host co-factors both within and between species is predicted to be highly influential of TE invasions following horizontal transfer, yet this variation remains almost entirely uncharacterized.
We recently discovered that Bruno likely represents a host-cofactor that promotes the transposition of P-elements in *Drosophila melanogaster*. We uncovered *bruno* through QTL mapping of natural variation in hybrid dysgenesis: a sterility syndrome resulting from *P*-element transposition. *bruno* encodes an RNA binding protein expressed in the female germline, with known functions in regulation of mRNA translation and alternative splicing. Consistent with a model in which Bruno regulates *P*-element mRNA, Bruno binding sites occur within *P*-element transcripts, and P-element mRNA abundance and splicing are altered in *bruno* heterozygotes. Furthermore, reduced bruno dosage suppresses both *P*-element excision and dysgenic sterility. Our work represents the first demonstration of natural variation in a host-cofactor of transposition. It further reveals how such variation can have a major effect on host fitness when new TEs invade: with permissive genotypes suffering increased transposition rates and reduced fitness.

Keywords: P, element, hybrid dysgenesis, RNA binding proteins, alternative splicing

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