Transposable element deregulation in Drosophila buzzatii-Drosophila koepferae hybrids

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Résumé

Transposable elements (TEs) can be activated under different stressful conditions, such as interspecific hybridization. This leads to higher transposition rates of some TEs, which have been reported both in animals and plants. Previous studies in our group showed mobilization of 28 different TEs in D. buzzatii-D. koepferae hybrids, as well as an increased transcription rate of the retrotransposon Osvaldo in male germline.
The mechanisms allowing TE deregulation, however, remain so far unknown. Two explanatory hypotheses are advanced: the hypothesis of piRNA gene pathway failure, by which TE activation is due to the divergence of the effector proteins between parental species; and the maternal cytotype failure hypothesis, due to differences between parental species' piRNA pools. Under the first hypothesis, a generalized derepression of TEs is expected; whereas only some particular TEs are expected to be deregulated under the second hypothesis. The aim of this study is to evaluate the effects of hybridization on the expression of whole genome TEs, and identify their possible causes. We have performed mRNAs sequencing in ovaries, notorious for playing a major role in the piRNA pathway, of both parental species as well as of F1 and backcrossed hybrids. We have also sequenced the transcriptome of D. buzzatii (male parental species) and F1 hybrid testes. Moreover, in order to detect changes in TE expression regulation, we have performed piRNA sequencing of the same samples.
We find modification of TE expression patterns in hybrids compared to parental species, which is not the case for piRNA expression patterns. Up to the 10% of the studied TEs are significantly deregulated compared to parental species, but this is not always associated with a decrease of piRNA levels. Furthermore, we also find unexpected cases in which TE expression is lower in hybrids than in both parental species. Thus, our results point to a complex TE deregulation pattern, involving not only factors mentioned in the above described hypotheses, but also the host genome defences to counteract the TE deregulation.

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