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EVOLUTION

Transposons drive sex chromosome evolution

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Two new studies from the same group show how transposons can drive the evolution of sex chromosomes and how this affects dosage compensation.

Drosophila miranda contains three different X chromosomes that were formed at different times. Males of this species contain one copy of each X chromosome, whereas females contain two; thus, to equalize expression of X-chromosome genes between the sexes, the transcription of genes on each single X chromosome in males is doubled. The different evolutionary timing of the origin of the three X chromosomes provides an opportunity to explore how this dosage compensation evolved.

Upregulation of X-chromosome genes in males is achieved by the binding of the male-specific lethal (MSL) complex to high-affinity chromatin entry sites, which contain MSL recognition elements (MREs). The MSL complex spreads along the chromosome and catalyses the deposition of chromatin marks, which leads to gene upregulation.

The two most recently evolved *D. miranda* X chromosomes, XR and neo-X, show full and partial dosage compensation, respectively. By comparison with an ancestor of *D. miranda* — *Drosophila pseudoobscura* — Ellison *et al.* found that about half of the MRE sites on the neo-X contain species-specific insertions that are derived from the transposable element ISX. This element is homologous to the ISY element that is found in both *D. miranda* and *D. pseudoobscura*. However, the ISX element is specific to *D. miranda*. Using transgenic assays in which either ISX or ISY elements were inserted into autosomes in *Drosophila melanogaster*, the authors showed that MSL was recruited to ISX but not to ISY and confirmed that a 10-bp sequence deletion between the two explains the difference in MSL recruitment.

Ellison *et al.* then showed that a similar evolutionary event occurred on XR. In this case the transposable element involved is called ISXR, which belongs to the same family as ISX but binds to MSL more strongly than ISX and has evolved separately. By comparing the evolutionary events on neo-X and XR, the authors suggest a three-step model for acquiring the ability to recruit the dosage compensation machinery. The steps include: domestication, in which the MRE sequence is acquired by a transposable element; amplification, in which the transposable element amplifies and inserts across the genome but is selected against on autosomes; and refinement, in which secondary 'fine-tuning' mutations create an MRE that binds to MSL more strongly. The last step also includes erosion, in which non-functional parts of the transposon are lost.

Both neo-X and neo-Y are thought to have descended from a pair of homologous autosomes and thus share some genes. Unlike neo-X, neo-Y is undergoing heterochromatin formation, which has silenced several of these shared genes. Zhou *et al.* focused on how this heterochromatin structure has evolved so far to determine whether the resultant silencing of homologous genes has played a part in dosage compensation on neo-X. The authors found that heterochromatin formation is triggered by the presence of repetitive DNA that is derived from transposable elements. However, the heterochromatic genomic regions on neo-Y and the homologous regions that are dosage compensated on neo-X do not overlap. Thus, heterochromatin formation on neo-Y does not necessarily drive dosage compensation on neo-X.

These studies not only provide insights into the evolution of mechanisms of dosage compensation in *D. miranda* but may also inform about potential roles of transposons in the evolution of other processes.

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ORIGINAL RESEARCH PAPERS Ellison, C. E. & Bachtrog, D. Dosage compensation via transposable element mediated rewiring of a regulatory network. *Science* **342**, 846–850 (2013) | Zhou, Q. *et al.* The epigenome of evolving *Drosophila* neo-sex chromosomes: dosage compensation and heterochromatin formation. *PLoS Biol.* **11**, e1001711 (2013)

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