Transposons Up the Dosage

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This combination stabilized and catalyzed the interface for oxygen evolution. An unexpected finding is that lithium ions from the electrolyte further improved the stability.

Formally, this device could be viewed as a buried junction, where the charge carriers form at the junction buried underneath the 2-nm nickel layer (the MIS structure). However, thicker 5- and 10-nm nickel films did not display the same high voltage, so the aqueous solution must play a role in the operation of this device. The authors attribute this effect to incomplete screening of the solution by the Ni/NiO_x layer.

Recent technoeconomic analysis (*5*) shows that to produce cost-competitive hydrogen via a PEC process, the solar-to-hydrogen efficiency should be at least 15% and perhaps greater than 20% (*6*). Other studies have shown that to achieve this efficiency, not only must the semiconductor electrode have the same solid-state properties as current photovoltaic devices, it also must have a tandem configuration (*7*–*9*). In a tandem configuration, two semiconductors with different band gaps are illuminated in series, so that the top semiconductor with the higher band gap absorbs the visible light and transmits the rest through to the bottom cell with a lower band gap. Thus far, the only PEC system that shows greater than 10% water-splitting efficiency is a tandem device composed of high-efficiency III-V semiconducting materials (*10*), such as gallium arsenide. This tandem configuration limits the semiconductors that can be used to pairs of highly crystalline materials that have matching crystal lattices.

An alternative tandem scheme presented by Nozik (*11*, *12*) relaxes these requirements by making use of separated p-type and n-type photoelectrodes with two different band gaps. This separated p-n arrangement eliminates the need to either match lattices or create stacks of dissimilar materials, and further allows the use of polycrystalline materials. Nozik showed that this configuration could perform unassisted water splitting, but the efficiency was limited by the photoanode. There are a number of excellent p-type photoelectrodes, including silicon, that can produce hydrogen with high efficiency, but no known n-type photoelectrodes (photoanodes) can produce oxygen with high efficiency.

The reason why n-type silicon was thought to be unsuitable for oxygen evolution was its instability in basic conditions and the formation of a thick oxide film that blocks the reaction in acidic conditions. However, the results of Kenney *et al*. show that a thin Ni film can protect the n-Si surface for oxygen evolution, as well as afford a good photovoltage. Thus, their result opens up the possibility of using this electrode in a p-n tandem configuration by coupling it with a photocathode that has a wider band gap, such as p-type copper gallium diselenide (p-CGS).

A tandem configuration of these two materials has a maximum theoretical efficiency greater than 25% (*9*). As shown in the figure, sunlight first illuminates p-CGS, which has a band gap of 1.68 V. The light that is not adsorbed illuminates the Ni-coated Si photoanode; when CGS is deposited on transparent conducting glass substrates, it shows good transparency for the longer-wavelength light below its band gap (*13*). Such a configuration illustrates the ability of a PEC system to integrate polycrystalline thin films with single-crystal photoelectrodes into a viable tandem device; this would be more difficult to accomplish with a solid-state device.

The results of Kenney *et al*. are a long way from being integrated into a viable water-splitting device. However, they do point the way toward reconsideration of a long-held belief about n-type silicon as a photoanode for oxygen evolution. The results open up some additional possibilities for a solar water-splitting system with efficiencies of 15% or greater.

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EVOLUTION

Transposons Up the Dosage

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A family of transposable elements have played an important role in the evolution of dosage compensation in fruit flies.

It took more than three decades—and a Nobel Prize in 1983—for Barbara McClintock's discovery of transposition and mobile genetic elements to become widely accepted. However, her vision of transposons as "controlling elements" and architects of genome organization has remained controversial. On page 846 of this issue, a report by Ellison and Bachtrog brings McClintock's prescient ideas back to center stage (*1*). They show that transposition has shaped the regulatory landscape of an entire chromosome at least twice in the evolutionary history of *Drosophila*, facilitating the emergence of novel sex chromosomes.

In species such as humans and fruit flies, where sex is established by an XX/XY chromosome system, the X chromosome is present in two copies in females (XX) and one copy in males (XY). A dosage compensation mechanism is necessary to ensure that genes located on the X chromosome are expressed at equivalent levels in both sexes. Curiously, humans and flies achieve this feat in opposite ways: in humans, one of the X chromosomes is inactivated in females, whereas in flies the transcription of X-linked genes is up-regulated by about twofold in males (*2*). Mechanisms of dosage compensation are remarkable in that they must be established rapidly during evolution for a new sex chromosome system to be able to emerge, and they must act at the level of an entire chromosome to regulate hundreds of genes. How do such mechanisms evolve?

Several lineages of *Drosophila*, which have recently evolved sex chromosomes, provide a unique system to address this question. *Drosophila miranda*, in addition to its ancestral >60 million year old "XL" chromosome, which is homologous to the X of *D. melanogaster*, has a younger

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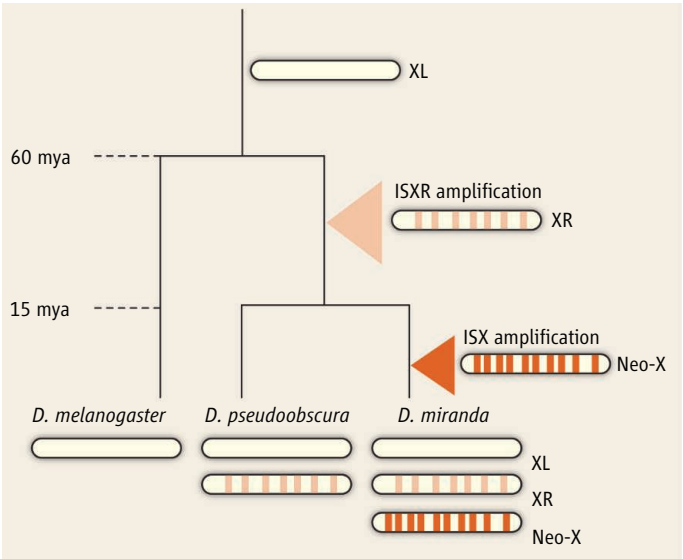
15-million-year-old "XR" chromosome, and a "neo-X" that emerged just 1 million years ago (*1*) and that is unique to *D. miranda* (see the figure). Whereas XL and XR exhibit full dosage compensation, the dosage compensation on the neo-X is still incomplete—a sign of its relative youth.

In *D. melanogaster,* dosage compensation requires the male-specific lethal (MSL) complex, which binds to MSL recognition elements (MREs) along the male X chromosome, from which it spreads to create a localized chromatin state that promotes a two-fold increase in gene transcription levels (*3*). Previous experiments in *D. miranda*, including mapping of MSL binding sites using chromatin immunoprecipitation followed by deep sequencing (ChIP-seq), revealed that the MSL complex is also responsible for establishing dosage compensation on the ancestral XL chromosome and on the newly evolved XR and neo-X chromosomes (*4*).

Ellison and Bachtrog delved more deeply into the evolutionary origins of the MRE on the neo-X of *D. miranda*. Unexpectedly, they uncovered that about half of 41 sites most robustly bound by MSL on the neo-X map to repetitive elements derived from a single transposon family named ISX. They found that ISX elements were amplified shortly after the formation of the neo-X, about 1 million years ago, and are almost entirely absent from the rest of the genome. But where did ISX come from? The ISX family is nearly identical, except for a 10–base pair (bp) internal deletion, to another family of *Helitron*-type transposons called ISY, which unlike ISX is dispersed throughout the *D. miranda* genome. By creating transgenic lines of *D. melanogaster* where various ISX or ISY elements are individually inserted into an autosomal chromosome, the authors demonstrate that ISX—but not ISY—successfully recruited the MSL complex at the ectopic insertion site and that the 10-bp deletion was necessary to create a functional MRE in ISX.

Ellison and Bachtrog go on to show that the recruitment of ISX for dosage compensation on the neo-X mirrors a previous episode of transposon recruitment on the 15-million-year-old XR chromosome. They found that the XR chromosome is highly enriched for yet another related family of *Helitrons*, named ISXR, which also recruits the MSL complex. The distribution of ISXR on the XR chromosome parallels that of ISX in the neo-X. Individual copies of ISXR show many more changes in their sequence compared with ISX (and ISY) but, paradoxically, exhibit stronger MSL binding. Thus, the same lineage of transposons has been co-opted or "domesticated" at two different time points to facilitate the evolution of dosage compensation in *Drosophila*. As such, ISX and ISXR provide evolutionary snapshots capturing different phases of the transposon domestication process.



Jumping genes drive evolution of dosage compensation. A schematic depicting the phylogenetic relationship between *D. melanogaster*, *D. pseudoobscura*, and *D. miranda*. Triangles represent waves of transposon amplification that distributed MSL binding sites across the XR and neo-X, facilitating the evolution of dosage compensation in novel sex chromosomes. Million years ago (mya).

Based on these results, the authors propose a three-step model for transposon-mediated evolution of dosage compensation: (i) A mutation introduces a functional MRE binding site in a progenitor transposon; (ii) the newly "functional" transposon replicates throughout the genome, and adaptive insertions on the nascent X chromosome are selectively retained; and (iii) binding sites are gradually fine-tuned while nonfunctional regions of the transposon are eroded by point mutations. The authors speculate that ISX has entered the third stage of domestication, whereas ISXR has essentially completed the process, as attested by its stronger MSL recruitment activity and the more profound sequence divergence of its nonfunctional parts. Importantly, the model predicts that the sequence signatures of domesticated transposons in *Drosophila* may disappear completely within a few million years, leaving only the functional binding sites as vestiges of the domestication process.

Several outstanding questions remain. For example, is there a special feature of *Helitrons* or the specific progenitor of the ISX/ISXR families that predisposed these elements to evolve a MRE? Is MSL binding to ISX and ISXR indispensable for proper dosage compensation in *D. miranda*? This question might be tested using newly developed genome-editing technologies to delete the MRE located within these elements (*5*).

Intriguingly, this is not the first time that transposons have been implicated in the process of dosage compensation. In mammals, LINE-1 retroelements are enriched on the X chromosome and have long been thought to play a role in the process of X-chromosome inactivation (*6*, *7*). Although the putative mechanism by which LINE-1 appears to contribute to mammalian X inactivation is distinct from ISX-mediated MSL recruitment in *D. miranda*, it shares a common theme of a chromosome-wide regulatory activity spread by transposon amplification.

This work adds another example of transposition acting as a rapid evolutionary mechanism to wire up a genomic regulatory network (*8*–*13*). It also suggests that many transposon domestications are likely to go undetected due to the rapid erasure of their sequence signatures. Although we now understand that controlling gene expression is not the raison d'être of transposons, we are gaining a better appreciation of their propensities to promote regulatory evolution, which led McClintock to originally call them "controlling elements."

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