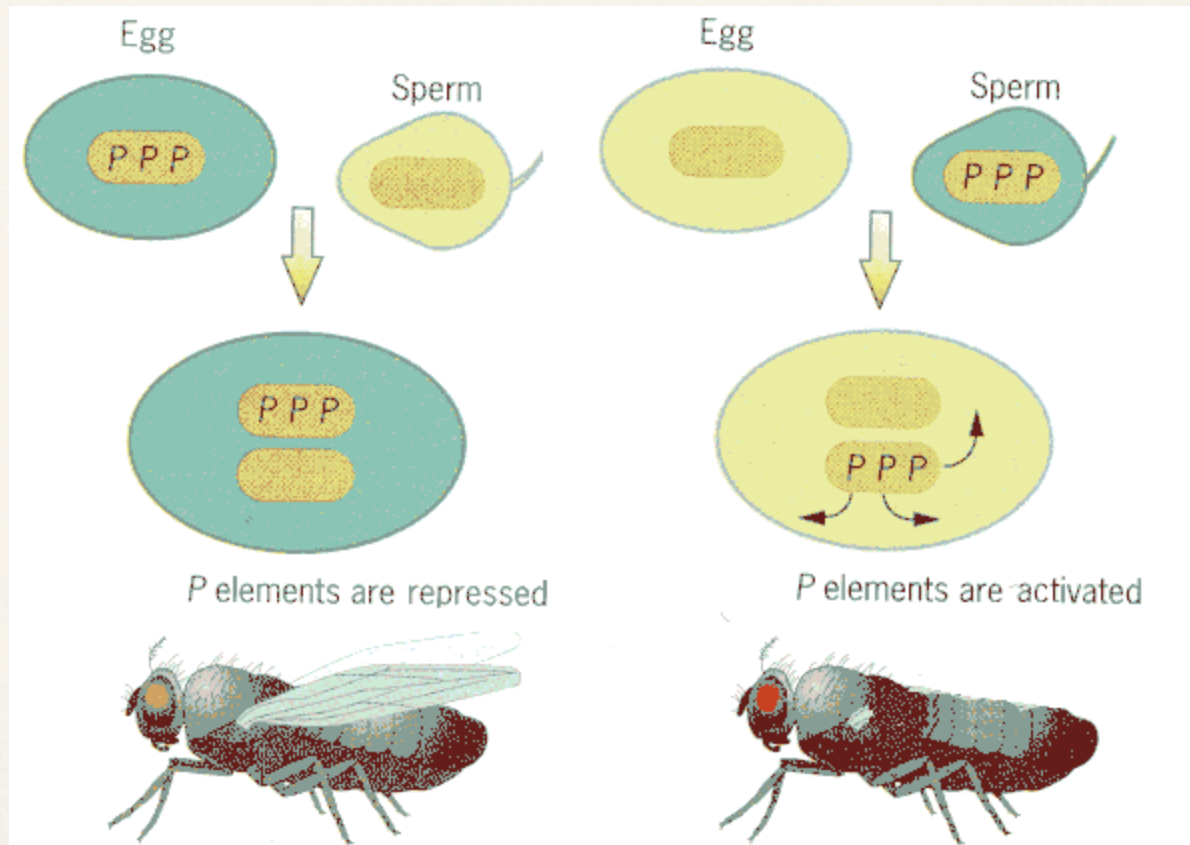


Adaptation to transposon invasion in *Drosophila melanogaster*

Zhiping Weng
UMass Medical School

P-element-mediated hybrid dysgenesis in *Drosophila*

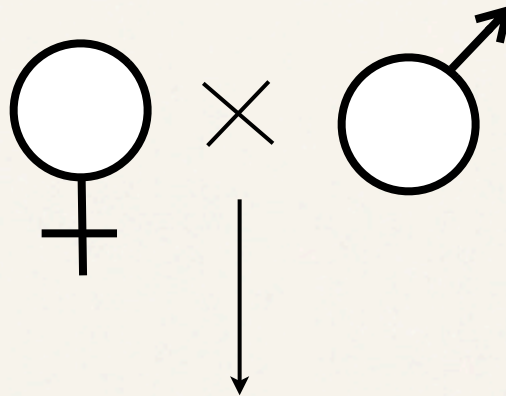


non-dysgenesis
fertile

dysgenesis
sterile

Transposon invasion causes “hybrid dysgenesis”

Mom deposits
piRNAs in the egg

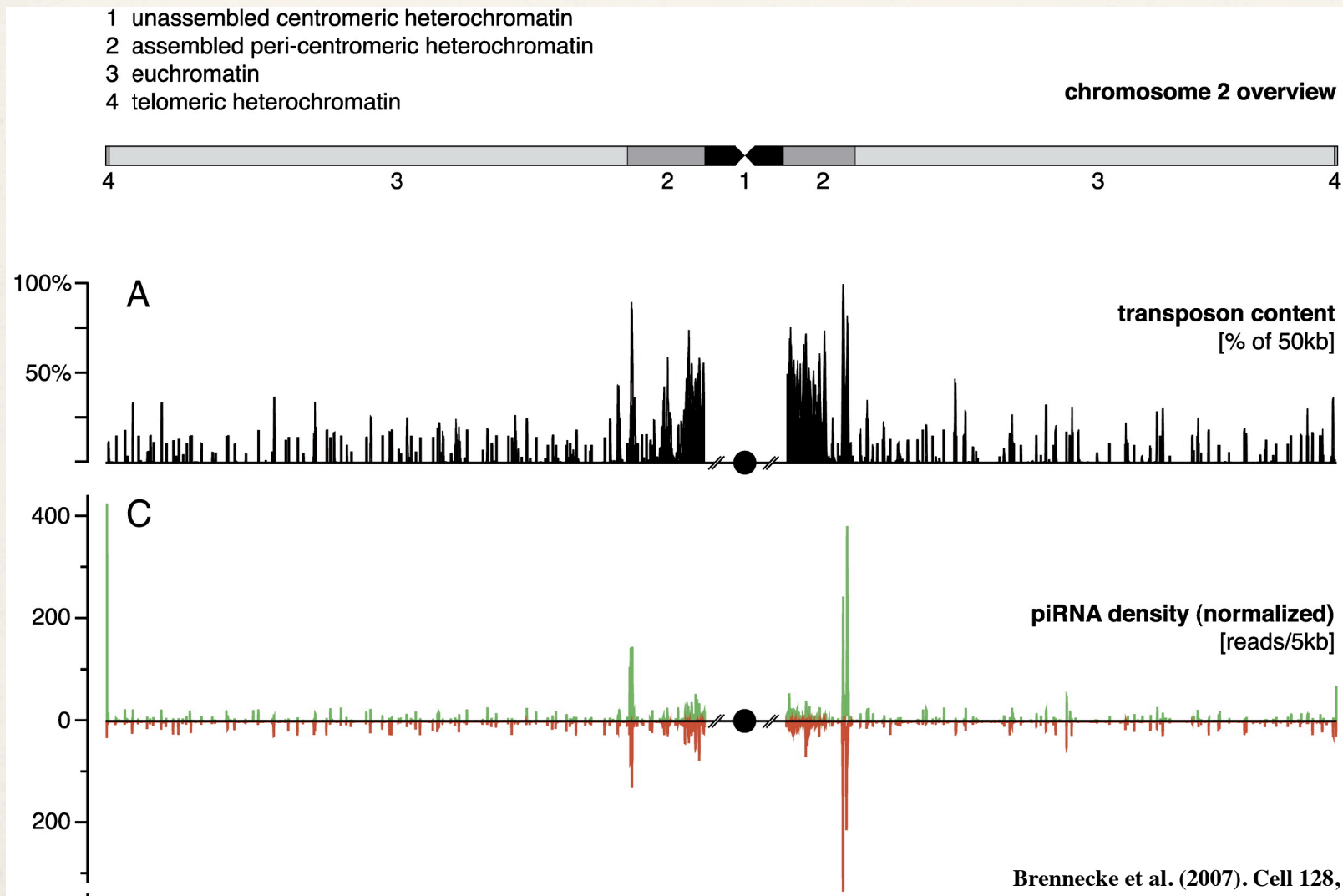


Dad carries a “new”
transposon (P-element)

F1 hybrid

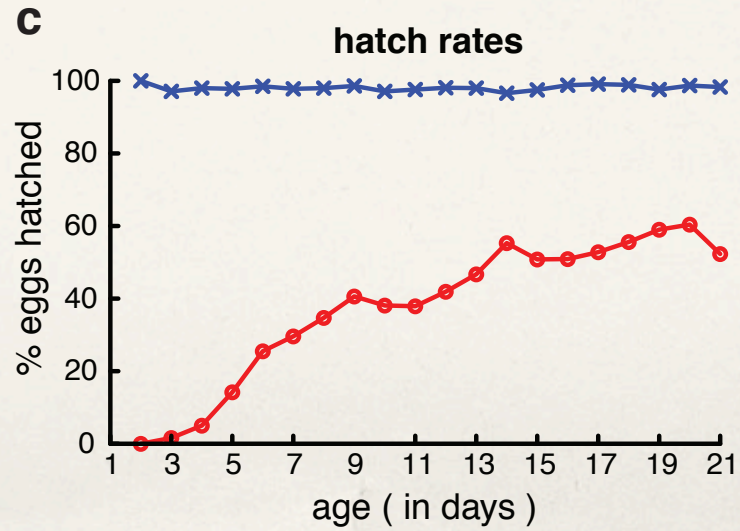
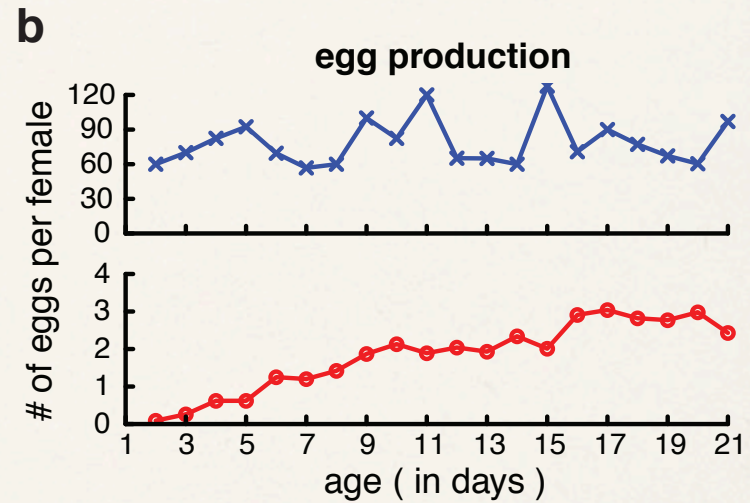
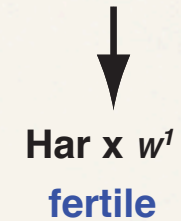
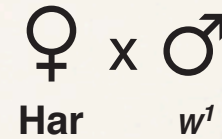
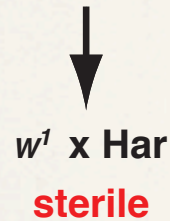
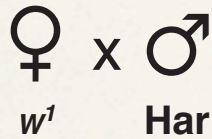
**Invading elements escape silencing
and lead to genomic instability
and hybrid sterility**

piRNAs map to transposon-rich heterochromatic clusters

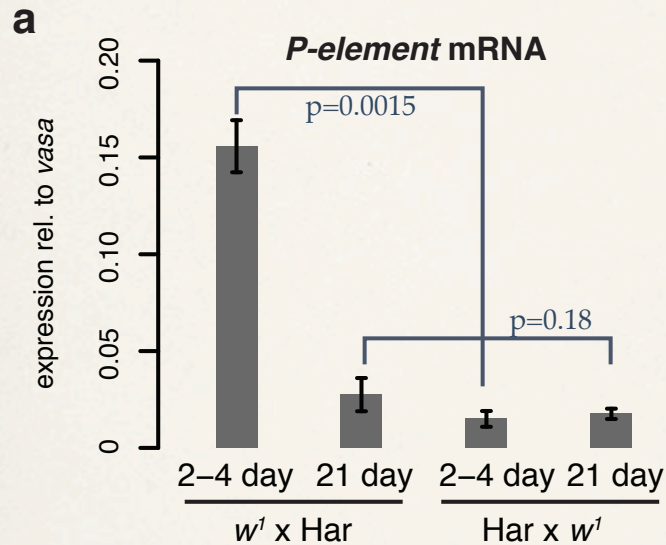


Phenotypic adaptation to P-element invasion

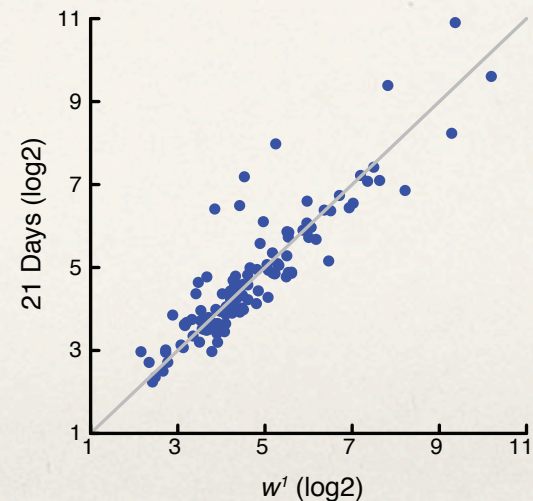
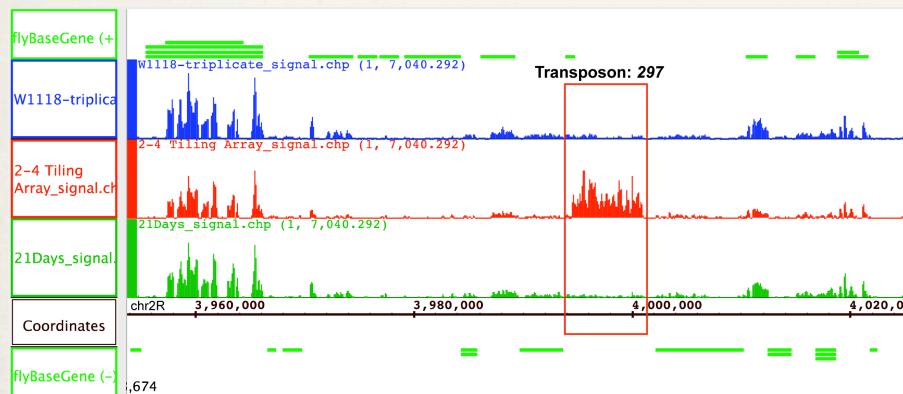
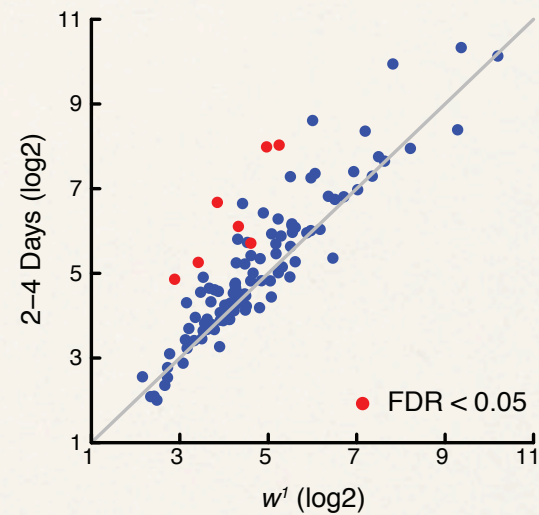
a **dysgenic hybrids** **reciprocal hybrids**



P-element and resident transposon expression

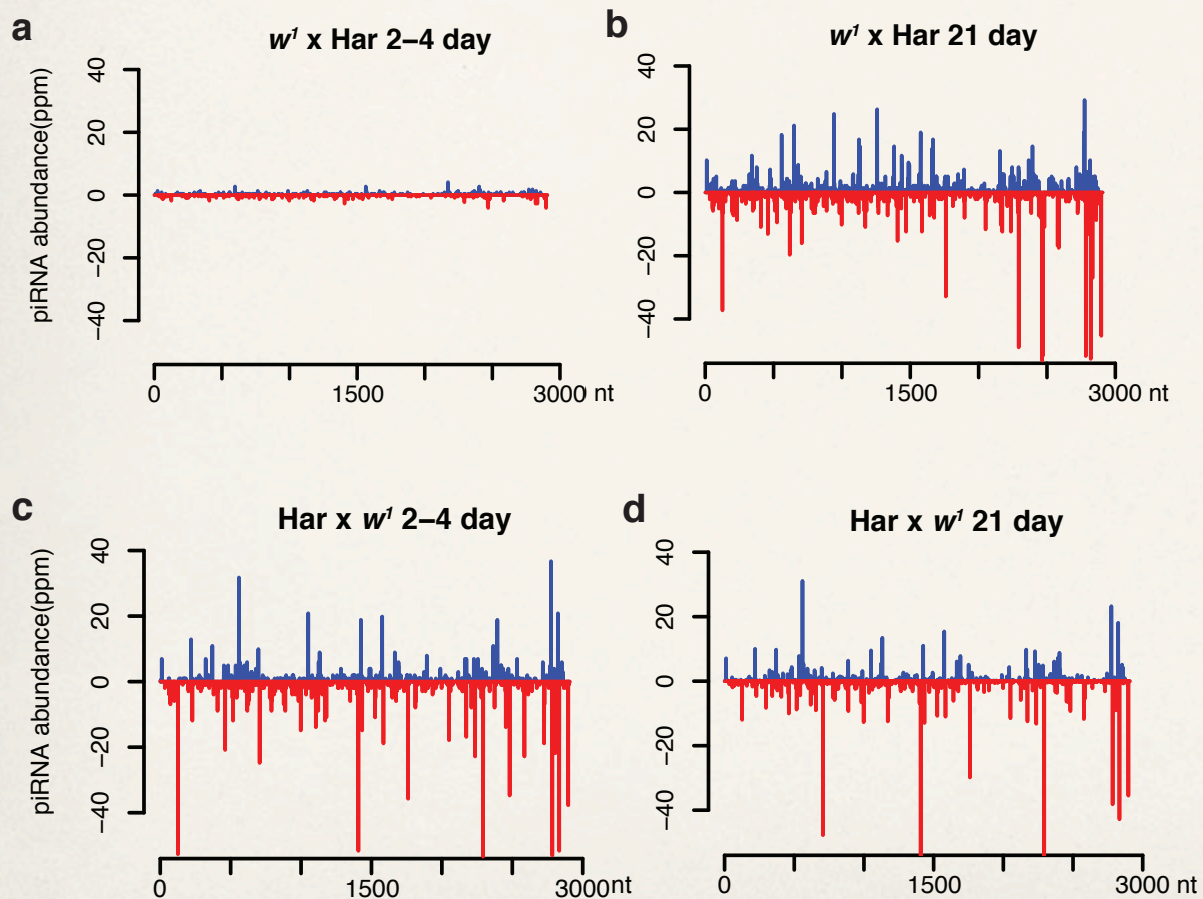


Transposon expression

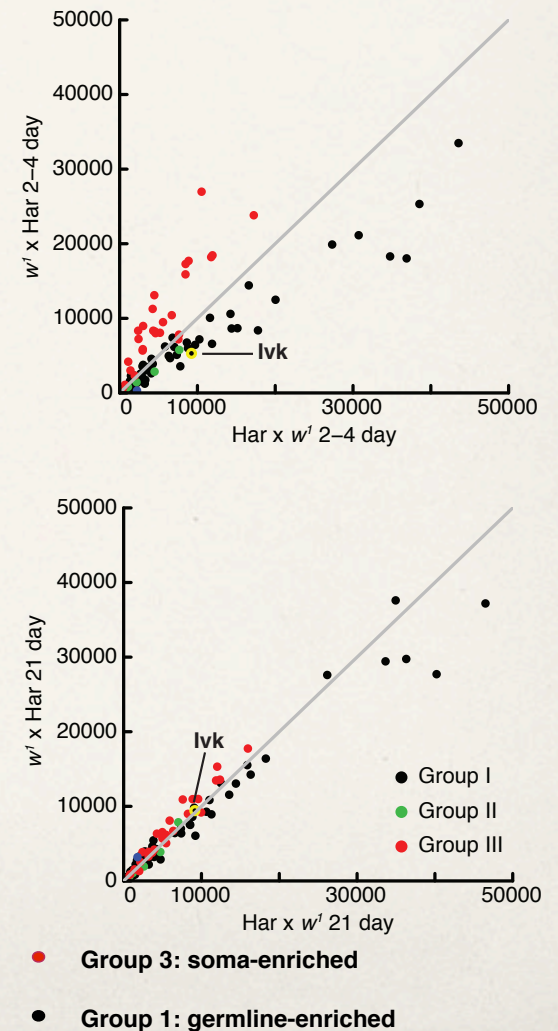


piRNA expression of P-element and resident transposon

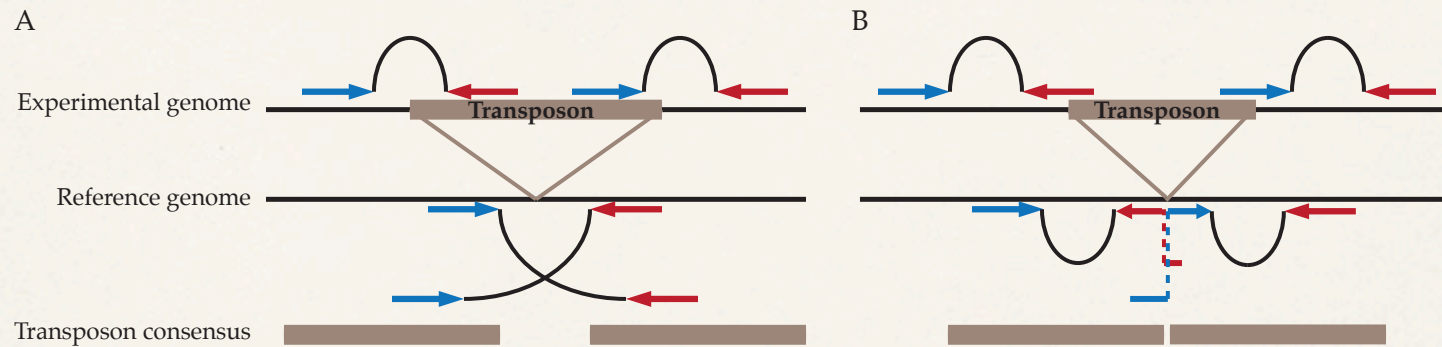
P-element



resident transposon



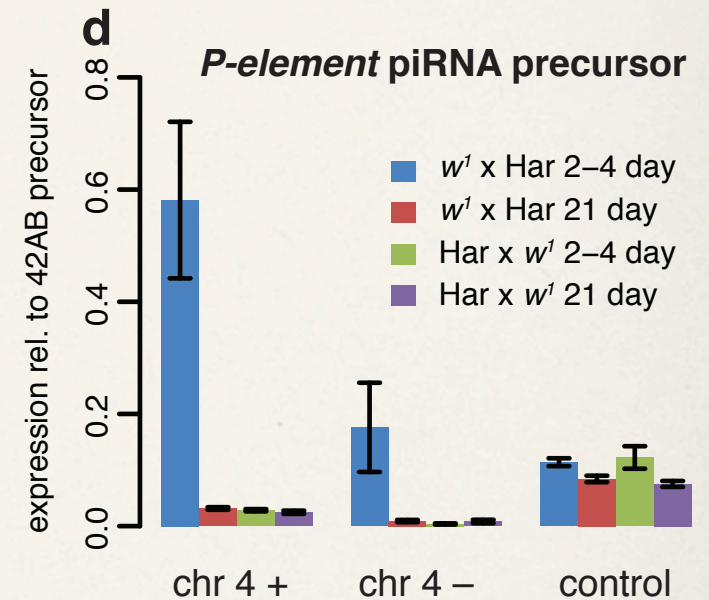
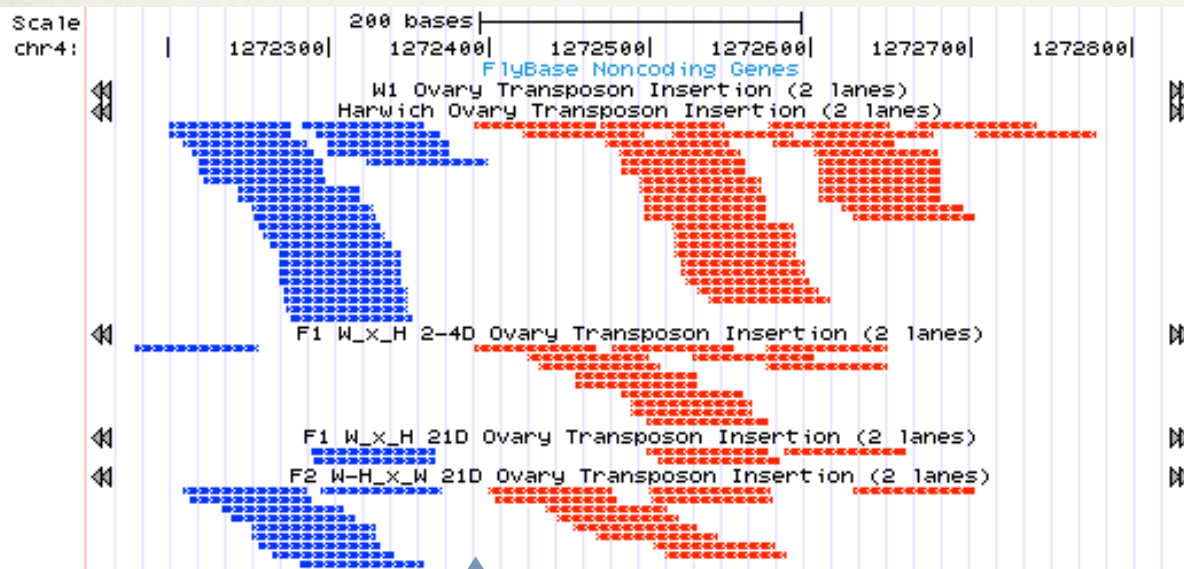
Detection of transposon insertion by Paired-end Genomic Sequencing



P-element transposition into cluster does not significantly contribute to de novo piRNA production in dysgenic females

- ❖ Genomic sequencing
 - ❖ F1 2-4 Day, 21 Day $w \times Har$ dysgenic females
 - ❖ F2 21 Days, $(w \times Har) \times w$ females
- ❖ 814 sites carrying new P-element insertions in at least one of the three progeny populations.
- ❖ 14 of these sites mapped to piRNA clusters, but 12 were defined by only single reads and thus represent rare polymorphisms.
- ❖ The other 2 sites were identified by multiple reads in 21-day-old dysgenic females, but were not detected in 2-4 day old dysgenic females or in backcrossed F2 progeny.

P-element piRNA precursor abundance at paternal clusters



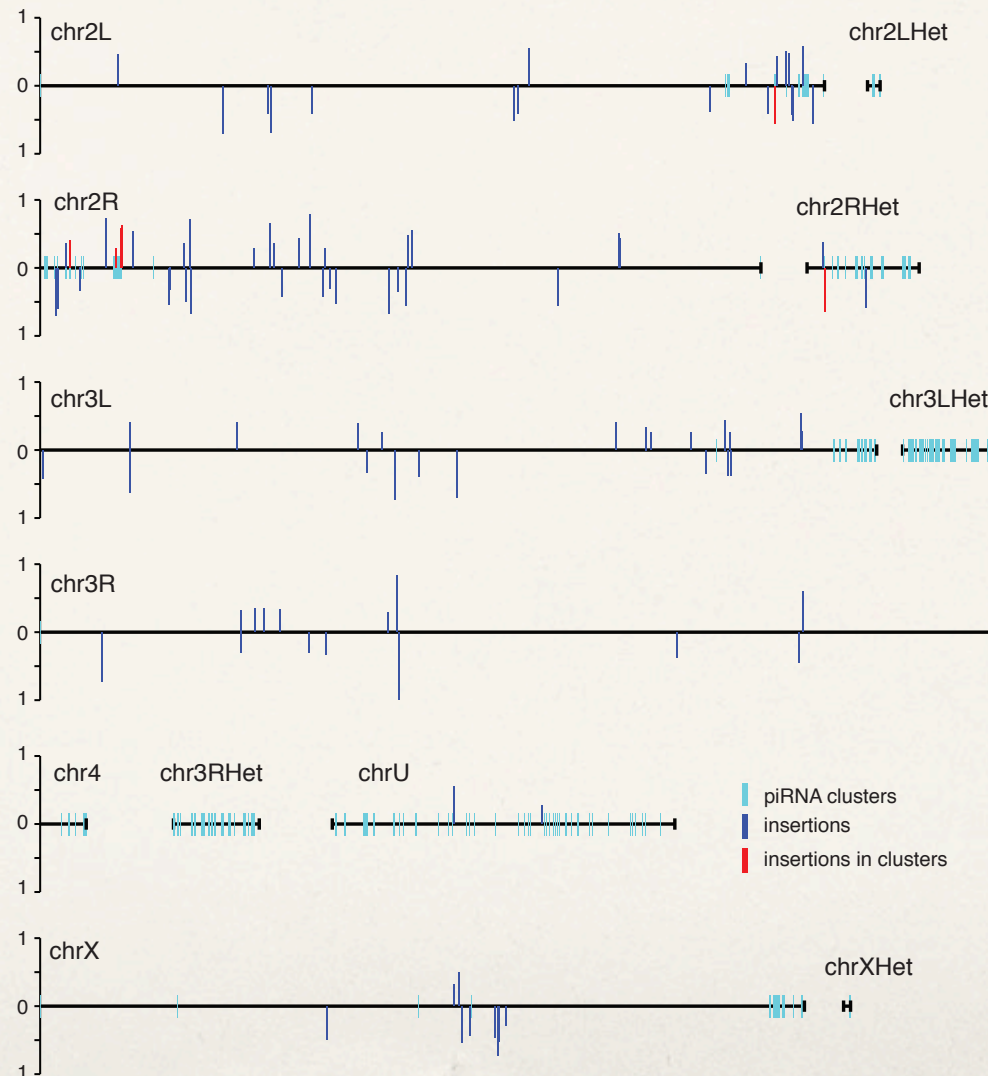
Thus:

P-element silencing is coupled with de novo production of P-element piRNAs encoded by clusters inherited from fathers.

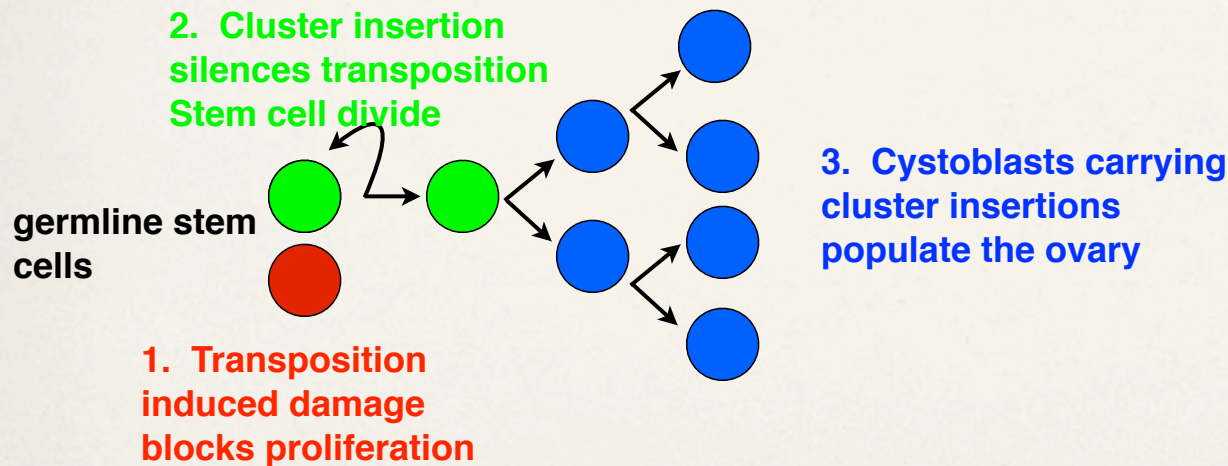
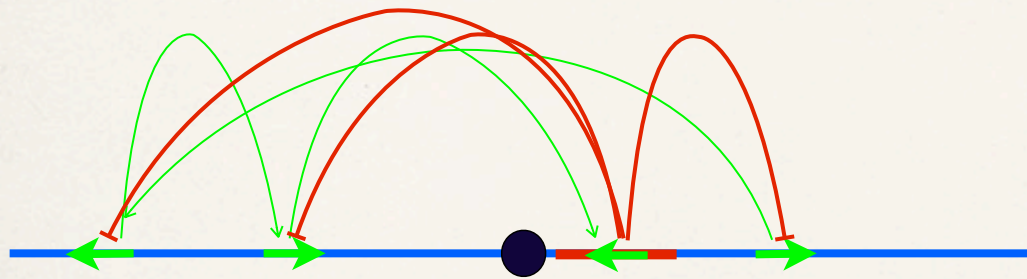
Resident transposon families mobilized as well



Genome distribution of inherited transposon insertions



Model



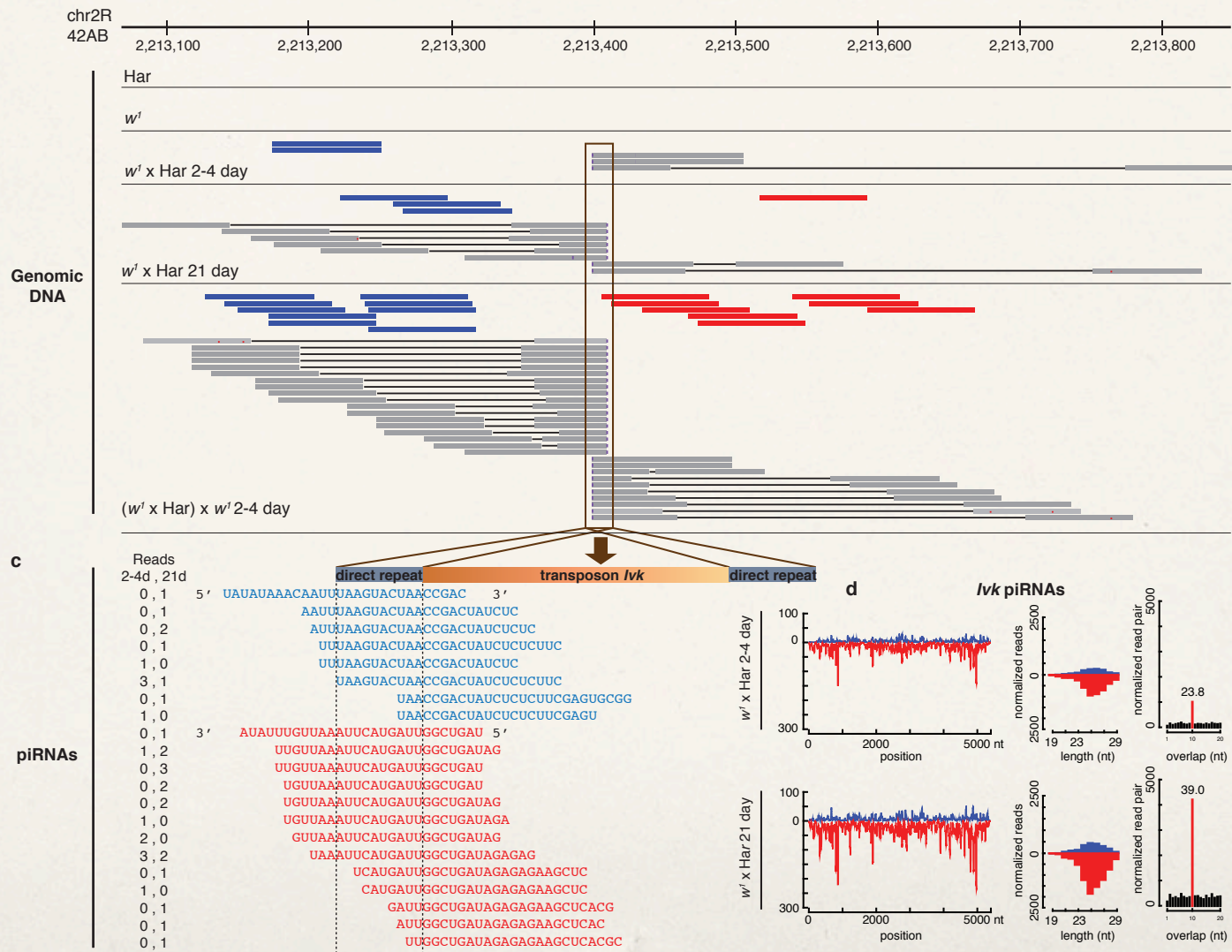
- * Transposon insertion into piRNA clusters could provide selective advantage, possibly by enhancing piRNA production and transposon silencing.
- * Inherently biased transposition should lead to accumulation of new cluster insertions independent of selective pressure, while selective pressure is required for cluster insertion enrichment through clonal expansion.

Crunch the numbers

Transposon Family	$w^1 \times \text{Har}$						$(w^1 \times \text{Har}) \times w^1$		
	2-4 day			21 day			21 day		
	Total reads	Reads in piRNA clusters	% clusters	Total reads	Reads in piRNA clusters	% clusters	Total reads	Reads in piRNA clusters	% clusters
All	2653±31	264±11	9.94±0.40%	4290±18	335±8	7.81±0.18%	867±27	64±6	7.40±0.63%
Inherited cluster insertions									
Bari1	21±2	11±1	53.47±6.05%	37±0	18±0	48.64±0%	1±1	0±0	0±0%
Blood	59±4	6±2	9.99±2.83%	96±2	9±2	9.17±2.23%	10±3	1±1	5.93±7.10%
copia	42±4	9±2	20.44±4.91%	95±3	18±1	18.48±1.25%	9±3	1±1	10.79±11.08%
Ivk	9±2	3±1	32.58±8.85%	18±1	4±0	22.55±2.30%	5±2	1±1	10.47±15.32%
Tabor	25±3	4±2	16.87±7.41%	24±2	4±2	18.25±5.07%	4±2	0±0	0±0%
1731	21±3	11±2	51.72±6.21%	25±3	17±0	68.75±7.09%	2±1	0±1	5.46±17.91%
Total	178±8	44±5	24.72±2.17%	295±6	70±3	23.74±0.88%	31±5	2±1	6.90±4.41%

- ❖ The inherited cluster insertions are linked to enhanced transposon silencing.
- ❖ Accumulation of piRNA cluster insertions is specific to dysgenic hybrids, where the pressure to increase silencing is strong.

Transposition into a piRNA cluster can lead to de novo piRNA production.



Summary

- ❖ The fertility of hybrid females progressively increases as age, indicating the adaptation to P-element invasion takes place in a single generation, within the female germline.
- ❖ This is associated with P-element silencing and de novo production of P-element piRNAs that encoded by paternal piRNA clusters.
- ❖ Silencing of resident elements correlates with site specific transposon insertions in piRNA clusters.
- ❖ Adaptation to transposon invasion is mediated by a combination of epigenetic and genetic mechanisms that enhance silencing and drive changes in chromosome architecture.

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