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Costas, J.[1], **E. Valade**[1], **and H. Naveira**[2]. [1] Dpto. Bioloxia Fundamental, Facultade de Bioloxia, Universidade de Santiago de Compostela, E-15703 A Coruna, Spain. [2] Dpto. Bioloxia Celular e Molecular, Facultade de Ciencias, Universidade de A Coruna, E-15071 A Coruna, Spain. A preliminary study on the relationship between the blood transposable element and the *Drosophila* genome.

Genetic and molecular studies of the coevolution between transposable elements and the host genome can provide important clues for the elucidation of gene regulation, identifying new loci involved in different aspects of gene expression, from chromatin insulation (*Su(Hw)*, Gerasimova *et al.*, 1995) to RNA stability (*su(f)*, Mitchelson *et al.*, 1993).

In this context, we have initiated an analysis of the relationship between the blood transposable element and the *Drosophila* genome. blood was first described as an insertion into the *white* gene of *Drosophila melanogaster* (Bingham and Chapman, 1986). This mutant, called *white-blood* (w^{bl}), has been included in several studies of modifier genes of the white locus: w^{bl} is partially suppressed by *Inr-a* (Rabinow *et al.*, 1991) and *Mow* (Bhadra and Birchler, 1996), partially enhanced by *Wow* (Birchler *et al.*, 1994) and *Lip* (Csink *et al.*, 1994b) and not affected by *Doa* (Rabinow and Birchler, 1989), $E(w)^a$ (Birchler and Hiebert, 1989), *Msu* (Csink *et al.*, 1994a) and *mw* (Birchler *et al.*, 1989). Nevertheless, a direct interaction between the modifier gene and the blood transposable element has only been demonstrated in the case of *Lip*, which modifies the total transcript abundance of blood.

We tried to search for another modifier of w^{bl} in order to find genes that interact with the blood element. We chose our candidate genes among that than meet these two requirements: i) they modify spontaneous mutations at other loci, but ii) have not been characterized at the molecular level. The selected genes were: $su(t)^1$, $e(dp^v)$, $Su(ss)^2$ and $su(pr)^B$ (Lindsley and Zimm, 1992). We also included $Su(Hw)^3$ because of the proposal of the existence of two potential *Su(Hw)* binding sites in the blood element (Wilanowski *et al.*, 1995).

All of these genes are on the third chromosome. So, males bearing the putative modifier gene were crossed with w^{bl} females over the TM3 *Sb Sr e* balancer chromosome. The F1 males (heterozygous for the tested gene) were screening for modification of the w^{bl} phenotype. The F2 generation allowed us to test for modification of the w^{bl} phenotype in homozygous condition of the tested gene.

w^{bl} is a temperature sensitive allele and the critical period for the w^{bl} eye phenotype is during the first half of pupal development (Ephrussi and Herold, 1945). So, pupae were subjected to two different developmental temperatures during these tests, 24° and 28°C.

No evidence of modification was found in any of these cases, suggesting that these genes do not interact with blood.

When you begin to search for coevolution between a transposable element and the host genome, you must take into account the possibility of a recent horizontal transfer, thus reducing the coevolution period (Kidwell, 1993). The importance of coevolution is clearly shown in the case of the copia transposable element. A transgenic copia element shows 100-700 fold increased expression in cell lines derived from *D. hydei*, which lack copia elements, relative to *D. melanogaster* cells (Cavarec *et al.*, 1994). These data have been interpreted as a result of coevolution between copia and the *D. melanogaster* genome to limit the mutagenic potential of copia.

Using the blood sequence submitted to the Genbank data base by the Berkeley Genome Group (AC: L49394) we designed a PCR to search for the presence of the blood element within the genome of the species of the *D. simulans* complex (*D. simulans*, *D. mauritiana* and *D. sechellia*). We amplified around 800bp comprising the integrase domain of blood in each of these species, using primers:

CAAAAGCCGGAATGCATAAAA and TCTGGGTAGTCTGCCAAATACT.

The comparison of the sequences we obtained revealed eight variable sites. In each site, only one of the species was different from the others. In four cases, *D. melanogaster* was the different one, in two cases, *D. mauritiana*, and in one case each, *D. simulans* and *D. sechellia*.

These data confirm the presence of blood within the genome of the four species of the *D. melanogaster* complex. Besides, they suggest vertical transfer of blood between these species. blood was present in the genome of the common ancestor of these species, thus allowing enough time to expect coevolution between blood and the *D. melanogaster* genome.

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Kozlova, A., and L. Omelyanchuk. Institute of Cytology and Genetics, Novosibirsk 630090. Y-chromosome factor controls transcription of fertility genes in *Drosophila melanogaster*.

Three transposants of P[lArB] showing sterility of homozygous males were induced and mapped by *in situ* hybridization (ms (3) P50 - 67A4-B13, ms (3) P122 - 92A2-14 and ms(3) P115 - 75D). The lacZ reporter gene in P[lArB]construction is under the control of a weak P-element promoter. In chromosome, nearby enhancer can activate lacZ, and β-galactosidase encoded by this gene is registered by X-gal staining of tissues. We use this approach to reveal an influence of Y aneuploidy on the activity of male fertility genes.

The presence of β-galactosidase in testes was detected after fixation in 0.1M PIPES pH6.9, 2mM EDTA, 1mM $MgSO_4*H_2O$, 9.2% formaldehyde for 20 min and incubation in staining solution (10mM $NaH_2PO_4*2H_2O$/ $Na_2HPO_4*2H_2O$ pH 7.2, 150 mM NaCl, 1mM $MgCl_2*6H_2O$, 0.3% Triton X-100, 0.2% X-gal) overnight.

The results of X-gal staining in male testes with different sets of sex chromosomes (Table 1) show that the presence of Y-chromosome is neccessary for the expression of male fertility genes. Our next step was to test the effect of Y-chromosome arm aneuploidy on the LacZ expression (Table 2).

Table1.

Genotype	Staining
X / Y; ms(3) P50 / TM3	+
X / Y; ms(3) P122 / TM3	+
X / Y; ms(3) P115 / TM3	+
X / O; ms(3) P50 / +	_
X / O; ms(3) P122 / +	_
X / O; ms(3) P115 / +	_
X / O; TM3 / +	_

Table 2.

Genotype	Staining
X/Y; ms(3) P50/TM3	+
X/Y; ms(3) P122/TM3	+
X/Y; ms(3) P115/TM3	+
X/O; ms(3) P50/+	_
X/O; ms(3) P122/+	_
X/O; ms(3) P115/+	_
YSX., y cv v f /O; ms(3) P50/+	+/-
YSX., y cv v f /O; ms(3) P122/+	+/-
YSX., y cv v f /O; ms(3) P115/+	+/-

Table 3.

Genotype	Staining
X/Y; ms(3) P50/TM3	+
X/Y; ms(3) P122/TM3	+
X/Y; ms(3) P115/TM3	+
X/R(Y)L; ms(3) P50/+	_
X/R(Y)L; ms(3) P122/+	_
X/R(Y)L; ms(3) P115/+	_

Table 4.

Genotype	Heat-shock 37°C 30 min.	Staining
X/Y ; +/+	_	_
X/Y; Bg 9.61/+	+	+
X/Y; Bg 9.61	_	_
y w/O; P103/+	_	+
y w/Y; P103/CyO	_	+
X/O; Bg9.61/+	+	+