D. paulistorum.

1. The *white* mutant (Figure 1f). In the offspring of two laboratory populations of *D. paulistorum* we observed six males with the characteristic *white* phenotype, inherited as a X-linked recessive allele (see Lindsley and Zimm, 1992).

2. The *Bar* mutant. Figure 1g shows one female descendent of the F1 of the mass cross between three mutant males and seven wild females found in a population of *D. paulistorum*. This mutant strain presented extremely variable phenotypic expression, being the fly in Figure 1h representative of the more extreme expression of the gene. As it occurrs in *D. melanogaster*, this gene is X-linked in *D. paulistorum*. This phenotype disappeared from the strain after a bottleneck induced by temperature accidental elevation of the culture chamber. The lack of the mutant phenotype may be also a consequence of a reversion of the phenotype to the wild one, or both phenomena. The first known *Bar* mutation was isolated by Tice as a single male in 1914. Homozygous or hemizygous *Bar* flies have narrow eyes in which the facet number has been reduced from the wild-type number. The mutations are all associated with chromosomal rearrangements as *tandem* duplications or inversions and translocations sharing a common breakpoint within the 16A1-2 region of the X chromosome (Tsubota *et al.*, 1989). Zeleny (1919, 1921) reported the instability of the mutation and its revertion to the wild type at the frequency of 1 in 1000 to 2000. Sturtevant (1925) suggested that this mutation was restricted to females and associated with the recombination, unequal crossing-over being the phenomenon responsible for *Bar* instability.

3. The *lozenge*-like mutant (Figure 1h). Seven eye-mutant males emerged in a same strain after six generations of rearing in laboratory. Both phenotype and genetic pattern are similar to those described in *D. melanogaster* (see Lindsley and Zimm, 1992) and in the same hypermutable strain of *D. simulans* already mentioned (Loreto *et al.*, 1998). The sterility of the homozygous female is a consequence of the detected absence of spermathecae.

4. The *yellow* mutant. Six yellow-pigmented body males were found in a recently established strain at the fourth generation (photos not shown). The gene is X-linked, similar to the same gene reported for other species of the genus (see Lindsley and Zimm, 1992). This strain is stable, being easily kept in our laboratory.

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Recovery and mapping of an *Antennapedia* mutation in *Drosophila simulans.*

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Hox genes are critical players in determining the basic body patterns of all animals and have attracted much attention from both developmental and evolutionary biologists (Carrol, 1995). While performing an X-ray mutagenesis screen (~ 4000 rad) for X chromosome deletions in *Drosophila simulans*, we recovered an apparent *Hox* gene mutation — a dominant *Antennapedia*-like allele (*Antp*). Most mutations in *D. simulans* are homologous to known mutations in *Drosophila melanogaster*. For recessive mutations, homology is easily established by complementation tests in species hybrids. However, because the *D. simulans Antp*-like mutation is dominant and likely homozygous lethal (*i.e.*, we were unable to construct homozygous lines), complementation tests were not possible. We thus attempted to infer homology by mapping *Antp* using visible markers available within *D. simulans*.

We crossed virgin *Antp* females to a multiply-marked *D. simulans* stock carrying the recessive visible mutations *forked* (*f*: 1-56), *net* (*nt*: 2-0), *plum* (*pm*: 2-100), *scarlet* (*st*: 3-49), *ebony* (*e*: 3-63). (Third chromosome map positions from Jones and Orr, 1998). F1 *Antp* males were then backcrossed to virgin *f; nt pm; st e* females. Their male and female progeny were scored for *Antp* and each of the five markers. The results showed that *Antp* is not on the X chromosome as both male and female progeny showed the mutant phenotype. We further found that while 17.4% (n = 218) of *nt pm* progeny also showed *Antp*, no *st e* progeny (n = 168) showed *Antp*. Therefore, like *D. melanogaster*, *Antp* in *D. simulans* is on the third chromosome.

Table 1. Backcross progeny from F1 *Antp* females x *f, nt pm; st e* males.

Genotype	Progeny
st + e	750
st Antp e	1
st Antp +	63
st + +	93
+ + e	149
+ Antp e	8
+ Antp +	355
+ + +	514

We determined *Antp*'s map position on the *D. simulans* third by backcrossing F1 *Antp* females to *f; nt pm; st e* males. Nearly 2000 progeny were then scored for the presence of *Antp* and the two third chromosome markers, *st* and *e* (Table 1). It should be noted that gene orders in *D. melanogaster* and *D. simulans* are essentially the same with the exception of loci included in a known paracentric inversion on the right arm of the third chromosome (Ashburner, 1989). In fact we found that the order of the three genes in *D. simulans* (*st e Antp*) differs from that in *D. melanogaster* (*st Ant e*) indicating that *Antp*, along with *e*, is included in the 3R inversion of *D. simulans*. Distances between markers are presented in Table 2. These map distances place *Antp* at 3-78.7. After accounting for the inversion difference between species, the map distances between the three loci in *D. simulans* are 27% higher than those in *D. melanogaster*. This is consistent with the known average 30% genome-wide increase in recombination rate in *D. simulans* relative to *D. melanogaster* (Ashburner, 1989).

Table 2. Map distances (cM).

Intervals	*D. simulans*	*D. melanogaster* †
st e	16.2	26.7
e Antp	31.9	23.2
st Antp	48.1*	3.5

**st Antp* distance is the sum of the smaller distances.

*† Data are from Lindsley and Zimm (1992)

These findings indicate that the *Antp* mutation reported here is likely a lesion at the *D. simulans* homologue of the *Antennapedia* locus in *D. melanogaster*.

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Recurrence of *yellow* mutation in *Drosophila subobscura*.

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Two males of *yellow* phenotype were detected in a homokaryotypic stock (O_{3+4+22}/O_{3+4+22}) of *D. subobscura*. These males were crossed with virgin females of the *yellow* stock. All offspring individuals were *yellow*, confirming that the original males presented the *yellow* mutation. This mutation has appeared many times in our laboratory stocks of *D. subobscura* (Mestres, 1996; Solé, 1997). In all cases the *yellow* mutation has arisen in the *cherry curled* strain or in stocks obtained by means of genetic crosses with this strain. This suggests that the *yellow* mutation originates in the *ch cu* strain, and that some transposable genetic element could be involved.

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