**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 occurs 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 reversion 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.

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).

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.


Two males of yellow phenotype were detected in a homokaryotypic stock (O1+4+22/O3+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.