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Genetic analysis of mutation causing disruption of veining in *Drosophila simulans.*

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We performed a genetic analysis of the *Drosophila simulans* population in Tashkent in 2001. Among the flies caught in the wild, a single female (T91) had an aberration disrupting wing veining, namely, one wing had an additional vein segment at the end of the 1st posterior cell (between L3 and L4 veins), whereas the other wing had a long branching from the posterior crossvein extending to the 2nd posterior cell. The aberration T91 was inherited and resembled the aberrated phen A discovered by A.V. Ivannikov (2000). According to his data, an additional vein of varying length was found either on a single wing or on both wings.

The *D. simulans* females inseminated in the wild were used to establish isofemale cultures and a total of 73 families were obtained. Additionally, aberrations resembling the T91 mutation were found in nine families of *D. simulans* females from Tashkent population, which corresponds to a 3% concentration of this aberration in the original population.

We used the T91 mutation for genetic analysis. In homozygotes, we observed the formation of the networks of additional veins, dilatation of the distal part of the first posterior cell, strong swellings at the ends of longitudinal veins, additional vein segments in the 1st, 2nd, and 3rd posterior cells, and vesicles close to the posterior crossvein (Figure 1 B).

The intraspecific hybrid progeny of the crosses between T91 mutant homozygous males and the wild type females from the other isofemale lines show a strong prevalence of phen A phenotype, which is similar to the phenotype of the original T91 female derived from the wild population (Figure 1C). This provides the evidence to a semi-dominant inheritance of T91 mutation with the varying expression in homo- and heterozygous states.

The close genetic similarity between *D. simulans* and *D. melanogaster* and their ability to mate and produce viable F1 hybrids (Sturtevant, 1921; Ashburner, 1989) was used for characterization of the T91 mutation. Likewise in intraspecific hybrids, the T91 mutation in interspecific hybrids has a semi-dominant pattern of inheritance with varying expression. In the wings of the hybrid progeny females from the crosses between T91 males of *D. simulans* and the wild type *Oregon* and *Canton-S* females of *D. melanogaster*, we observed ectopic vein segments varying in size in different combinations of the wing cells. Most frequently, these veins were found in the third posterior and submarginal cells. Sometimes, we observed the bifurcation of L4 vein terminus. Unlike the intraspecific *D. simulans* +/T91 heterozygotes, the interspecific + (*D. melano-gaster*)/T91 (*D. simulans)* hybrids do not carry additional vein fragments in the first posterior cell.

The phenotypes similar to that of T91 in *D.simulans* are known to be produced by several mutations in *D. melanogaster* (Fristrom *et al.*, 1994; Gotwals and Fristrom, 1991; Thompson, 1974). One of those is the *net* mutation, which usually produces the network of extra veins in the wing blade, additional longitudinal fragments of veins in the wing blade, and enlargement of distal part of the first posterior cell (Lindsley and Zimm, 1992). Different *net* alleles are known, which are characterized by

incomplete dominance over the norm and which may cause formation of the small additional vein fragments in the wing blade in *net* / + heterozygotes (Biehs *et al*., 1998).

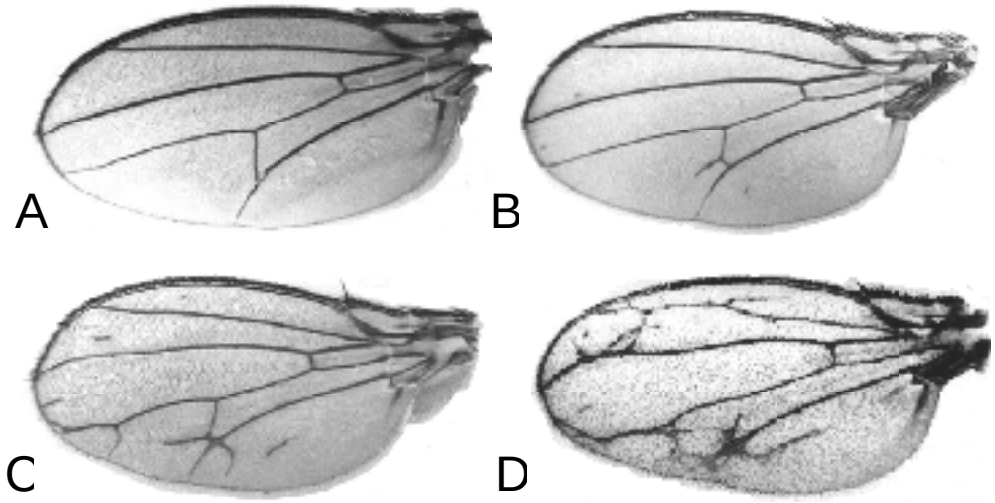

Figure 1. Wing vein patterns in *Drosophila simulans*: wild-type wing (A), phenotypic expression of the net^{ST91} allele (B), the net^{ST91} / + (C), and $Df(2)net^{62}$ / net^{ST91} (D) heterozygotes.

Taking into account the strong phenotypic similarity between T91 and *net* homozygotes, we obtained the interspecific compound of T91 mutation from *D. simulans* with $Df(2)net^{62}$ deficiency from *D. melanogaster*. The phenotype of such interspecific hybrid females with T91 / $Df(2)net^{62}$ genotype (Figure 1 D) is similar to the phenotype of the mutant _91 or *net* homozygotes. The aberration T91 in *D. simulans* is therefore identical to the *net* mutation in *D. melanogaster*. We denominated this mutation as net^{ST91}.

Following our data, the concentration of net^{ST91} mutation in the *D. simulans* population from Tashkent in 2001 was about 3%. Meanwhile, different alleles of the *net* locus are known to be permanently present in considerable concentrations in wild populations of *D. melanogaster* (Korochkina and Golubovsky, 1978; Weisman et al., 2001). Presumably, the high concentration of net^{ST91} in heterozygous state corresponding to the phen A indicates at the permanent presence of this mutation at the high concentrations in the natural population of *D. simulans*.

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