

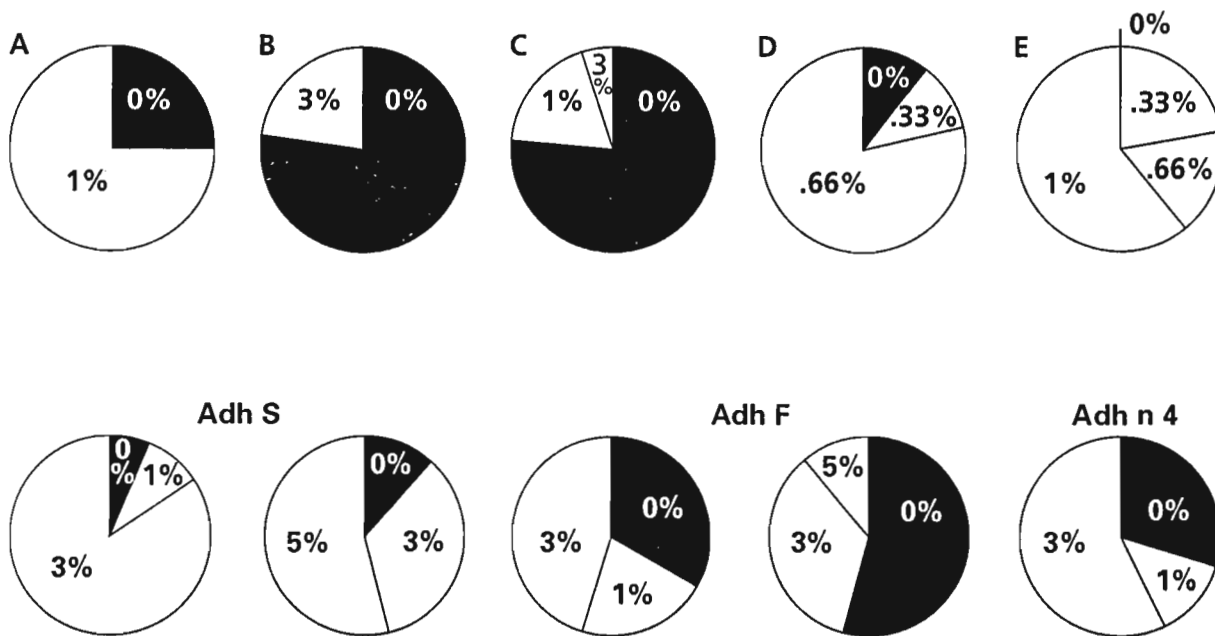
D. simulans

Figure 2. Oviposition site preferences of *D. simulans* and *D. melanogaster* strains, homozygous for *Adh^F* (FFN), *Adh^S* (SSN) and *b Adhⁿ⁴*, in two-way and three-way choice experiments with various combinations of concentrations of acetic acid, supplemented to standard medium.

patches with 3% (v/v) acetic acid negatively influenced the choice of patches containing 1% (v/v) acetic acid (compare Figure 2C with Figure 2A). In a concentration range below 1% (v/v), 78% of the eggs were laid on medium patches supplemented with 0.66% (v/v) acetic acid (Figure 2D).

Our *D. melanogaster* strains, homozygous for different *Adh* alleles, and *D. simulans* showed different adult tolerances toward acetic acid (Eisses and Den Boer, 1995), as has been shown before with other strains (McKenzie and McKechnie, 1979; Parsons, 1982). The same pattern has been repeated in the oviposition site preferences toward various concentrations of acetic acid. This phenomenon could indicate that flies search for oviposition sites they are pre-adapted for. Although flies prefer to lay eggs in medium patches consisting of acetic acid, newly-hatched larvae tried to escape these patches in a multiple choice situation and crawled into ethanol supplemented patches or into patches with standard medium (Eisses and Bets, 1992). The multiple choice situation probably mimics the situation in decaying fruit with inverse gradients of acetic acid and ethanol, respectively, perpendicular to the surface of the pile of rotting grapes (McKenzie and McKechnie, 1979).

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Weisman, Natalya Ya, and Ilya K. Zakharov.

Institute of Cytology and Genetics, Russian Academy of Science, Siberian Division, Novosibirsk, 630090, Russia. Penetration of a new minus allele isolated from a wild population of *Drosophila melanogaster*.

The presented research is part of a scientific program on localization and identification of mutants isolated from wild populations (Weisman *et al.*, 1995; Weisman and Zakharov, 1995).

Isolation of mutation. The visible mutation #89381 was found and isolated from the offspring of a *Drosophila melanogaster* female fertilized in nature,

from Uman in 1989. Mutants often have reduced body. Bristles vary in number and size. The hairs are also truncated and thinned in comparison with the norm. Some individuals have reduced number of hairs and their disposition is

disturbed. Besides, the order of eye facet disposition is broken; the breakage is better expressed in males rather than in females. In addition to the mentioned above visible characters, the homozygous #89381 females are sterile as a rule, i.e., in crosses with the fertile males, they produce no eggs. The external genitals of the females do not differ from the norm. The internal organs are represented by under-developed spermatheca and parovarium, which are two-fold diminished in comparison with the genitals of normal females of the same age. The ovaries of #89381 females are filled with eggs in the early stages of development, but mature eggs are absent. The homozygous males are fertile.

The #89381 is a recessive mutation localized on chromosome 2. The chromosome #89381 is maintained in a balance with the chromosome *Cy In(2LR)*. In culture, the viability of #89381 homozygotes is reduced and the average share of #89381 homozygotes equals to 40% of the theoretically expected. However, life duration of adult homozygotes does not differ significantly from that of heterozygotes *Cy/#89381*.

Location of #89381 mutation. The location of #89381 mutation on the chromosome was made by using recessive mutations of the chromosome 2; such as dumpy (*dp*; 2-13.0); cinnabar (*cn*; 2-57.5); vestigial (*vg*; 2-67.0) and brown (*bw*; 2-104.0). The flies from the stock *Cy/#89381* were crossed to those from corresponding marker stocks in order to set the compounds of #89381 with the marked chromosomes. The resulted heterozygous females *dp/#89381*, *cn/#89381* and *vg/#89381* were crossed to corresponding dihomozygous males *dp #89381*, *cn #89381* and *vg #89381*.

The offspring of all three types of crossings were characterized by a decrease in homozygous #89381 mutation classes as opposed to heterozygous classes (see Table 1). On this basis, to calculate recombination frequency, the reciprocal homozygous class was considered equal in number to the corresponding heterozygous #89381 mutation class. This principle was applied both to recombinant and non-recombinant classes. The frequency of recombination between #89381 mutation and three marker genes, *dp*, *cn* and *vg*, was 49.1; 41.2; and 52.4, respectively.

In analogous crosses with the marker mutation brown, amongst the offspring of females *bw/#89381* out of more than 2500 flies, we did not observe any crossovers. This fact points out that #89381 mutation is located in the vicinity of *bw* locus at position 100.

Allelism test. The hybrid F1 from crosses of the flies from the stock *Cy/#89381* with the stocks carrying mutations with the similar phenotypical expression and localization at the region of 100 units, in particular, abbreviated (*abb*; 2-105.5), morula (*mr*; 2-106.7) and minus (*mi*; 2-104.7), were analyzed. The heterozygous #89381/*shr bw-2b abb sp* and #89381/*px bw mr sp* were phenotypically normal. The #89381 mutation was found to be allelic to the *mi* mutation. Heterozygotes #89381/*mi* have the phenotype of the minus mutation, characterized by reduced body size and bristles and by truncated and reduced number of hairs. Homozygous *mi* have low variable viability, and the hatching of flies occurs later than in normals. The females *mi/mi* are completely sterile, whereas the males are fertile (Lindsley and Zimm, 1992).

The expression of the *mi*-#89381 allele from the Uman population differs from the previously known allele mainly by the influence on the eye structure and by varying phenotypic expression. It is not inconceivable that there are genetic modifiers which influence expression of the *mi*-#89381 allele. We have noticed an interesting peculiarity of recessive *mi*-#89381 mutation. In heterozygous condition with the chromosome from laboratory strain marked as Lobe 2 (*L*; 2-72.0) (Catalog number 2-23 in the fund of the laboratory of genetics of populations of the Institute of Cytology and Genetics, Russian Academy of Sciences), the *mi*-#89381 behaves as the dominant mutation. The heterozygous phenotype *L-2/mi*-#89381 is similar to homozygous phenotype *mi*-#89381/*mi*-#89381. The minus mutation from the fund of Umea *Drosophila* Stock Center is exhibited in heterozygote with *L-2* (2-23) analogously to *mi*-#89381 mutation. In compounds of *mi*-#89381 chromosome with the chromosomes carrying the other Lobe alleles, i.e., *L-4*, *L-81* and *L-2* (from the other laboratory stocks), the *mi*-#89381 is recessive. The phenomenon observed may be caused either by deletion or by interaction of *mi*-#89381 mutation with the gene *L-2* or some other loci from this chromosome. An attempt to isolate by recombination the mutation with phenotypical expression similar to *mi*-#89381 from the chromosome Lobe-2 (2-23) of the laboratory strain gave no positive effect. To test the presence/absence of rearrangements at the chromosome Lobe-2 (2-23) in the region of minus location, the chromosomes from salivary glands of heterozygotes *L-*

Table 1. Segregation in crosses for #89381 localization (R.f. = recombination frequencies).

Female genotype	Progeny genotype	Number of progeny	
<i>vg/#89381</i>	<i>vg/vg #89381</i>	298	R.f. = 49.1%
	<i>#89381/vg #89381</i>	210	
	<i>vg #89381/vg #89381</i>	77	
	<i>+vg #89381</i>	288	
<i>cn/#89381</i>	<i>cn/cn #89381</i>	385	R.f. = 41.2%
	<i>#89381/cn #89381</i>	178	
	<i>cn #89381/cn #89381</i>	63	
	<i>+cn #89381</i>	271	
<i>dp/#89381</i>	<i>dp/dp #89381</i>	266	R.f. = 52.4%
	<i>#89381/dp #89381</i>	239	
	<i>dp #89381/dp #89381</i>	158	
	<i>+dp #89381</i>	293	

2/+ were analyzed. The gene minus has a cytological location at the region 59 D9-C1 of the chromosome 2. The analysis by light microscope did not reveal any chromosomal rearrangements in this and close to this region. However, we can not exclude the microdeletion which is detectable only by molecular genetics methods. Mutations *abb*, *mi*, *mr* and *slite* (*slt*; 2-106,7) constitute a group of genes with the similar phenotypical expression, which are located within the limits of a small chromosomal region. Possibly, the genes responsible for the normal development of imaginal disks are located in this chromosomal fragment. The abnormalities of these genes may cause the arising of phenotypically similar mutations. On the other hand, the *mi*-#89381 allele described above is related by phenotypical expression to the gene *morula*. The minus and *morula* genes are probably the duplications of one and the same gene, which diverged in evolution. The analysis of functioning of such genes, the knowledge of their pleiotropic effect, peculiarities of expression of different genetic variants are necessary for solving the problems of genome evolution, chromosome structure and regulation of gene expression.

Acknowledgments: The authors are indebted to Dr. V.F. Semeshin for cytological analysis of salivary gland chromosomes. We are also grateful to Karin Edstrom for providing *Drosophila melanogaster* stocks from Umea *Drosophila* Stock Center. The work was supported by the Russian Fund of Fundamental Research (No. 96-04-50009) and State Programme Frontiers in Genetics (No. 5.742).

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Ryerse, J., J. Swarthout, and B. Nagel. Department of Pathology, St. Louis University Health Sciences Center, 1402 South Grand Avenue, St. Louis, MO 63104. tel 314-577-8480; fax 314-577-8489; e-mail ryersejs@sluvcu.slu.edu Cloning and molecular characterization of a partial ATP citrate lyase cDNA from *Drosophila melanogaster*.

ATP citrate lyase (ATPCL) catalyzes the conversion of citrate and CoA to oxaloacetate and acetyl-CoA. Acetyl CoA is an essential intermediary metabolite in the biosynthesis of cellular fatty acids and cholesterol (Kornacker and Lowenstein, 1965; Sullivan *et al.*, 1973; Singh *et al.*, 1976). ATPCLs have been cloned from the rat (Elshourbagy, *et al.*, 1990; Kim *et al.*, 1994) and the human (Elshourbagy *et al.*, 1992). We report here a partial ATP citrate lyase cDNA from

Drosophila melanogaster, denoted DmATPCL.

While screening a *D. melanogaster* Canton S 2-14 hour embryonic cDNA library (Stratagene) with antibodies to candidate gap junction proteins (Ryerse, 1993, 1995), a clone was isolated with sequence homology to ATPCLs from the human and rat. The nucleotide sequence of the *D. melanogaster* ATPCL cDNA is 1623 bps in length (including the polyA tail) and contains an open reading frame which codes for a protein of 391 amino acids (Figure 1).

GCG PileUp alignment of the human, rat and fly ATPCL amino acid sequences is shown in Figure 2. The fly sequence is proximally incomplete, beginning at amino acid 710 of the human sequence. In the region of overlap, DmATPCL has 75.5% and 75.7% identity with human and rat ATPCLs, respectively (GCG BESTFIT analysis).

Human and rat ATPCLs contain domains which are considered essential for enzyme function (Elshourbagy *et al.*, 1992), including a catalytic phosphorylation site at His759, two ATP binding domains (aa 700-720 and 751-777) and a potential acetyl-CoA binding site (aa 778-788). His759 is phosphorylated by nucleoside diphosphate kinase and ATP (Wagner and Vu, 1995). His759 is conserved in the DmATPCL sequence. DmATPCL has 64% identity with the first ATP binding domain, 74% identity with the second ATP binding domain and 50% identity with the putative acetyl-CoA binding region in the human sequence (Figure 2). ATPCL is thought to associate with the ATP conducting voltage dependent anion channel (VDAC) in the outer mitochondrial membrane (Brdiczka, 1990), presumably for ready access to newly synthesized ATP. DmATPCL has been assigned Genebank accession number U87317.

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Figures 1 and 2 on following two pages.