

Mutation Notes — *Drosophila melanogaster*

A genetical mapping and characterization of a new mutation affecting the *rotund* and *roughened* eye genes.

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A new recessive mutation of *Drosophila melanogaster* under conditions of mobilization of *hobo* elements has been obtained. This mutation modifies different organs of the fly. Legs of homozygotes are shortened due to the fusion of 2-4 tarsal segments. In males sex combs are reduced.

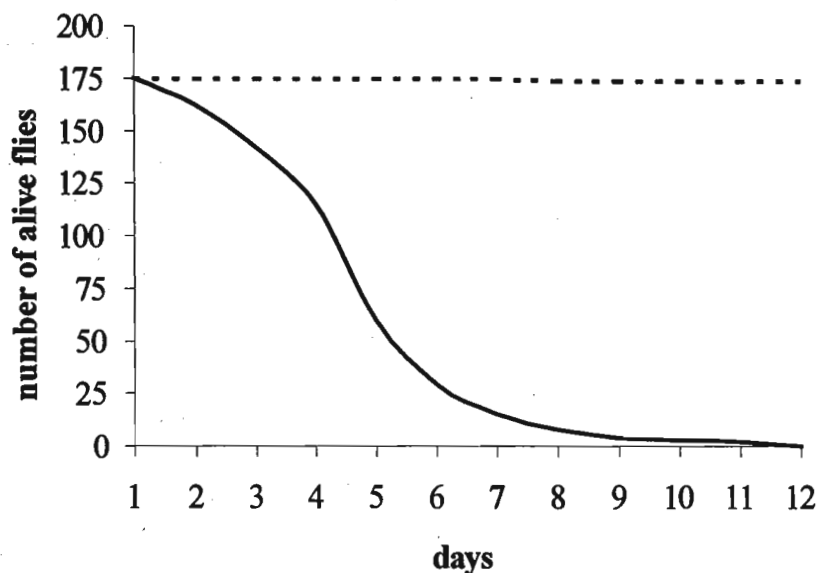


Figure 1. Lifetime of flies under investigation. (----- – $rn^{13-1}/TM2$; ——— – rn^{13-1}/rn^{13-1}).

The wing blade is shortened. The wing width is almost normal and the wing is oval. L2 is interrupted and L5 is shortened. The wings are slightly raised. Eyes are stripy and strongly reduced in size. The eye is rough owing to irregular faceting. Lifetime of the imago is sharply shortened. All of them died during 12 days after pupation (Figure 1). Homozygous males and females are totally sterile. In females, ovaries are reduced in size. Mobility is reduced. Polytene chromosomes are normal.

This mutation was demonstrated to be allelic

with an allele rn^3 of gene *rotund* which causes respective changes in wings and legs. *rn* is located in 84D3-4 (Agnel *et al.*, 1989). We carried out a deletion mapping with deficiencies Df84C1-3;84E1 (Df(3R)dsx2M/TM2), Df84D3;84F1-2 (Df(3R)dsx10M/TM2), Df84D8-9;85A1-3 (Df(3R)dsx11/TM2) and Df84B2;84D3 (Df(3R)Antp2, Antp² e¹/TM3, Sb¹) from Bloomington *Drosophila* Stock Center and the mutation was mapped in cytological region 84D3;84D8-9. We refer to this new *rn* allele as rn^{13-1} . rn^{13-1} is allelic with allele roe^1 of gene *roughened* eye, too. Such heterozygotes demonstrate the same phenotype as roe^1 homozygotes. roe^1 previously was mapped in cytological region 84D1+ (Agnel *et al.*, 1989; Lewis *et al.*, 1980). We mapped roe^1 in cytological location 84D3 using deficiencies mentioned above. Using fluorescent *in situ* hybridization we revealed the *hobo* element insertion to the cytological region 84D-E – region of location of genes *rotund* and *roughened* eye.

In a large number of cases, mutations in the *rn* locus also affect gene *roughened eye* (Agnel *et al.*, 1989). C. Ma and colleagues set up a hypothesis that *roe* and *rn* are two classes of mutation of the same gene (Ma *et al.*, 1996). Our data on a new location of *roe* give indirect evidence in favour of this hypothesis. It's also possible that *roughened eye* and *rotund* are two closely linked genes. There are two major transcripts from the *rn* region: 1.7 kb and 5.3 kb (Agnel *et al.*, 1989). We suppose that *rn*¹³⁻¹ affects the 5.3 kb transcript which accounts for development of subdistal parts of appendages (Agnel *et al.*, 1992).

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References: Agnel, M., S. Kerridge, C. Vola, and R. Griffin-Shea 1989, *Genes Dev.* 3:85-95; Agnel, M., L. Roder, R. Griffin-Shea, and C. Vola 1992, *Roux's Arch. Dev. Biol.* 201:284-295; Lewis, R.A., T.C. Kaufman, R.E. Denell, and P. Talerico 1980, *Genetics* 95:367-381; Ma, C., H. Liu, Y. Zhou, and K. Moses 1996, *Genetics* 142:1199-121.



An auditory-deficient double mutant in *Drosophila melanogaster*: *aristaless*¹; *thread*¹ (*al*¹; *th*¹).

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The aristae in *Drosophila melanogaster* contain the Johnson's organ, which is the "hearing center" of the fly (Eberl *et al.*, 1997, and references contained within). Here we report the construction of a mutant stock which has disrupted aristae morphology and function. This stock consists of mutations in the genes *aristaless* (*al*: 2-0.4) and *thread* (*th*: 3-43.2). *al* mutants have reduced-length branched aristae, and *th* mutants possess normal-length aristae with reduced branching. *al*; *th* individuals have both shorter and less branched aristae and are auditory-deficient, as demonstrated by Burnet *et al.* (1971).

To our knowledge, an *al*; *th* stock is not currently available in any public nor private stock center. We created an *al*; *th* stock by the following crossing scheme: First, *al*¹ (2-0.4) *b*¹ (2-48.5) *c*¹ (2-75.5) *sp*¹ (2-107.0) males were crossed to *th*¹ (3-43.2) females. F₁ males and females were then intercrossed. F₂ individuals displaying an *al*; *th* phenotype (but not the *b*, *c*, or *sp* phenotypes) were recovered and intercrossed, thereby combining in the F₃ *al* + + + 2nd chromosomes in a *th* background.

Auditory mutants such as *al*; *th* have a number of potential uses. Here we briefly note two of them. First, because hearing is one of the poorest genetically characterized sensory modalities, auditory-deficient mutants may help us understand the genetic mechanisms underlying the auditory sense (e.g., Eberl *et al.*, 1997). Second, since insect senses are utilized during courtship bouts and hence influence sex-specific behaviors, sensory-deficient males and females may shed light on the basis of male courtship behaviors and female choice of mates. The *al*; *th* double-mutant has already been studied from this standpoint: it is known to affect courtship duration (Burnet *et al.*, 1971),