chromosome. Southern blot analysis using DNA probes spanning the *m-dy* complex indicates that the m^{MR} mutation is a 19-25kb chromosomal deletion in the *m-dy* interval. Our unpublished molecular analysis of this region indicates the existence of separable *m* and *dy* transcription units, and we postulate that m^{MR} removes part or all of both transcription units.

Mutation Notes - Other Species

Report of E. Solé. Dept. Genètica, Facultat de Biologia, Universitat de Barcelona, Spain.
Spontaneous *yellow* mutation in the *ch cu* strain of *Drosophila subobscura.*

Two *yellow* male flies spontaneously arose in a homokaryotypic stock of *D. subobscura* kept in the laboratory for a long time. This stock bears the recessive mutations ch (*cherry*, bright red eyes) and *cu* (*curled*, wings curled concave upwards), both located on chromosome O. Another *yellow* male fly arose after some generations in a cross between a wild male and five *ch cu* females. Only the right half of this mutant individual was *yellow*; his half left was wild type. It was fertile and no mutant flies appeared either in the F1 or F2 of a cross with *ch cu* females.

The *yellow* mutation is recessive, located in the A (sexual) chromosome and has been previously described in *D. subobscura* (Krimbas, 1993; Mestres, 1996).

References: Krimbas, C.B., 1993, Drosophila subobscura: *Biology, Genetics, and Inversion Polymorphism.* Verlag Dr. Kovac, Hamburg; Mestres, F., 1996, Dros. Inf. Serv. 77: 148.

Report of Albert Kamping and Wilke van Delden. Department of Genetics, University of Groningen, P.O. Box 14, 9750 AA Haren, The Netherlands.
A rare *α-Gpdh* allele in *Drosophila simulans.*

In contrast to many other allozyme loci, *α-Gpdh* is remarkably invariable in *Drosophila.* The *α-Gpdh* locus is monomorphic for electrophoretic variation in almost all *Drosophila* species. Some species show alleles at very low frequencies and in only two out of almost 200 species that have been analyzed, the *α-Gpdh* locus is classified as polymorphic (*D. melanogaster* and *D. subarctica*). The low level of variation is ascribed to the important functions in energy metabolism of the enzyme. New mutants at this locus are assumed to be deleterious, and only under conditions without biochemical or physiological constraints new mutants may be maintained. Allele substitutions have taken place in the evolution of *α-Gpdh* in *Drosophila*, because different species carry different alleles. Alleles with identical electrophoretic mobility are restricted to certain species or species groups. The distribution and uniformity in alleles within and between species cannot be explained without the action of natural selection, where metabolic function of the enzyme and ecological niches of the species are assumed to be main factors in the evolutionary process of *α-Gpdh.*

Table 1. Changes of the rare $\alpha Gpdh^{S}$ allele frequency in laboratory *D. simulans* populations, started with different initial frequencies at 20°C and 29.5°C and raised under uncrowded conditions.

Temperature	Initial frequency	Generations 1	5	10	15
20°C	.25	.24	.25	.16	.14
	.50	.53	.52	.37	.31
	.75	.74	.69	.59	.59
29.5°C	.25	.28	.29	.21	.19
	.50	.51	.49	.41	.36
	.75	.72	.69	.65	.68

D. melanogaster is one of the exceptions concerning the level of variation at the *α-Gpdh* locus. Almost every wild population of *D. melanogaster* is polymorphic for two common alleles, Slow (*S*) and Fast (*F*). The sibling species *D. simulans* is monomorphic and carries an allele with identical electrophoretic mobility as the *D. melanogaster F*-allele. In consecutive years we observed an additional *α-Gpdh* variant in a wild population of *D. simulans* in The Netherlands. Electrophoretic mobility of this allele is comparable with the *S* allele of *D. melanogaster*, and its frequency reaches the level of polymorphism. Four out of 21 captured *D. simulans* females produced progeny (no hybrids) carrying the *S* allele in a frequency not significantly different from .25. We derived homozygous *S* and *F* strains, and laboratory populations with different

initial frequencies were started at 20°C and at 29.5°C under uncrowded conditions, to test whether the rare *S* allele would persist in the populations. The F2 generations were analyzed, and the observed numbers of genotypes were tested for deviations from the expected ratio and allele frequencies were followed for 15 generations, with 100 individuals per sample. We also tested the F2 generation under more stressful developmental conditions.

In Table 1 the frequencies of the rare α-*Gpdh*^{S} allele in *D. simulans* populations started with different frequencies at 20°C and 29.5°C under optimal developmental conditions are presented. In generation one (= F2) no deviations from the expected 1:1 or 1:2:1 genotypic ratios were observed. A slight decrease in α-*Gpdh*^{S} frequency is observed after 10 and 15 generations at both temperatures. At 29.5°C a tendency for heterozygote advantage was observed at all three starting frequencies in generation one, and the combined data showed a significant excess of heterozygotes ($P < 0.05$). The tendency of heterozygote advantage was also observed in later generations at 29.5°C. This is possibly the reason for the lower decrease in α-*Gpdh*^{S} frequency at that temperature. Under stressful developmental conditions, *i.e.,* high larval crowding, a highly significant deviation from the expected numbers of genotypes was observed ($X^2_2 = 20.27$, $P < 0.001$). Under these conditions, individuals homozygous for the rare *S* allele have a significant fitness disadvantage compared to homozygous *FF* and heterozygote individuals. Fitness values derived from F2 ratios of the three genotypes were .65, 1.00, and .86 for *SS* homozygotes, heterozygotes, and *FF* homozygotes, respectively.

We observed clear differences in α-*Gpdh* genotype frequencies between optimal and stressful conditions. The fitness differences among α-*Gpdh* genotypes under stressful conditions may be caused by functional restrictions of the enzyme product of the rare allele. Only under conditions without these restrictions, the rare allele may be maintained in the population or species.

References: Coyne, J.A., W.F. Eanes, J.A.M. Ramshaw, and R.K. Koehn 1979, Syst. Zool. 28: 164-175; Johnson, F.M., and H.E. Schaffer 1973, Biochem. Genet. 10: 149-163; Lakovaara, S., A. Saura, and P. Lankinen 1977, Evolution 31: 319-330; Lakovaara, S., and L. Keränen 1980, Hereditas 92: 251-258; O'Brien, S.J., and R.J. MacIntyre 1972a, Genetics 71: 127-138; O'Brien, S.J., and R.J. MacIntyre 1972b, Biochem. Genet. 7: 141-161.

Report of F. Mestres and M. Pascual. Dept. Genètica. Universitat de Barcelona. Barcelona (Spain).
bm (*bombolles*) a wing mutation of *D. subobscura.*

When analyzing a sample of *D. subobscura* flies from Observatori Fabra (Barcelona) many flies with abnormal wings were detected. Young flies presented big bubbles in the wings due to lymph accumulations between the two cell layers of this body structure. Usually bubbles tear after a few days releasing the lymph. As a consequence the wings get crumpled in the places where lymph bubbles were present and the wing tips curl upwards. Many individuals also showed bubbles inside their abdomens, being bulky and presenting alterations in the tegument of their abdominal segments.

The trait is autosomal and recessive. Its penetrance is incomplete and its expressivity is variable.

All these characteristics are similar to other mutations described in *D. subobscura* as *bubble*, *bladder* and *blister-curly* (Krimbas, 1993).

References: Krimbas, C.B., 1993, *Drosophlia subobscura*: Biology, Genetics and Inversion polymorphism. Verlag Dr. Kovac.