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.