

center and performed complementation test. The flies $dx^*/ex\ dx$ were alive but had the weak mutant phenotype similar to dx^* . It became clear that dx^* was a new allele of *deltex* locus (1-17.0). I named it as dx^{fl} or *deltex*^{female lethal}. The new dx^{fl} allele shows only faint terminal deltas (especially on L4 and L5) without any vein thickening as in dx or dx^{st} . The dx^{fl}/dx compounds are viable and have weak mutant expression (as dx^{fl}) or intermediate one. Males dx^{fl}/Y have normal viability.

To exclude opportunity that the cause of female lethality is the absence of Y chromosome (the Y-suppressed lethals are well known) I crossed dx^{fl}/Y males to $\bar{X}\bar{X}/O$ females. In the progeny the 64 ♀♀ and 63 ♂♂ appeared; it meant the normal viability of dx^{fl}/O males. In the progeny of other similar cross: $1♀ +/dx^{fl}/x\ \bar{X}\bar{Y}/O$ ♂♂ 27 females and 44 males (as dx^{fl} and +) appeared. So the reason of female lethality of dx^{fl} is not the absence of Y chromosome. The analogous results were reported for interaction Y with other sex-specific lethals (Baker & Ridge 1980; Belote & Lucchesi 1980).

From a genetic point of view, it is of importance that *deltex* locus includes alleles both female and male influence on sex differentiation: dx^{st} discovered by Bridges in 1931 is male sterile and dx^{fl} is female-lethal. The similar situation was found in adjacent X-chromosome locus *Sxl* (1-19.1) where allele Sxl^{Fl} is semidominant female lethal and Sxl^{Ml} is male-lethal allele (Cline 1978). This fact makes the study of *deltex* alleles quite intriguing. The other convenient trait of dx^{fl} is visible phenotypic effect in males for at the moment almost all sex-specific lethals are killing one sex without any visible effect on the other one. It is very interesting also to analyze the interaction of dx^{fl} with the known recessive and dominant suppressors of dx . The question arises whether the suppressors can normalize both phenotypic and lethal expression. At last, dx^{fl} is very convenient mutant for balancing the stocks like *ClB* or *FM3*. In our laboratory we are steadily keeping the *ClB* stocks by crosses $ClB/dx^{fl} \times dx^{fl}$ from 1973.

References: Baker, B.S. & K.A. Ridge 1980, *Genetics* 94:383-423; Belote, J.M. and J.C. Lucchesi 1980, *Genetics* 96:165-186; Dresher, W. 1964, *Am. Naturalist* 98:167-171; Golubovsky, M.D. and Yu.N. Ivanov 1972, *DIS* 49:117; Fukunaga, A., A. Tanaka & K. Oishi 1975, *Genetics* 81:135-141; Uchida, S., T. Uenoyama & K. Oishi 1981, *Jpn. J. Genet.* 56:523-527; Cline, T.W. 1978, *Genetics* 90:683-696.

Gonzalez, A. & J.L. Mensua. University of Valencia, Spain. High detrimental load in two populations of *Drosophila melanogaster*.

Late in October of 1979 a capture of *Drosophila melanogaster* was carried out simultaneously in two sites: one cellar and one vineyard both located in Requena (Valencia) in the east of Spain. The distance between the two sites was 4 km.

Three hundred chromosomes were extracted, 155 chromosomes from the cellar and 145 chromosomes from the vineyard, in the following way: males were individually mated to females $Ubx^{130}es/CSb(Ubx^{130})$ Ultrabithorax, which is included in *IN(3LR)TM2*). This chromosome suppresses virtually all crossing over in the third chromosome. A single Ubx^{130} male fly from each F_1 was mated again with $Ubx^{130}es/CSb$ females.

All third chromosomes were maintained, as lines, at 19^2 balanced with *TM2(Ubx¹³⁰)* chromosomes, which help maintain less viable or lethal chromosome types until the moment when the crosses are made to estimate viabilities.

Homozygote and heterozygote relative viabilities in both cellar and vineyard populations were estimated as in Wallace (1956), following a mating scheme similar to that of Watanabe et

Table 1. Average homozygote and heterozygote viabilities from cellar and vineyard populations.

Popu- lation	Total	Homozygotes including lethals	Homozygotes excluding lethals	Heterozygotes
Cellar	155	0.4055±0.0255	0.5219±0.0252	1.0027±0.0075
Vineyard	145	0.3612±0.0254	0.4879±0.0266	1.0053±0.0073

Table 2. Detrimental: lethal load ratio and percentage of lethals from cellar and vineyard populations.

Popu- lation	Total	% lethals	D:L
Cellar	155	23.85	2.58
Vineyard	145	28.27	2.40

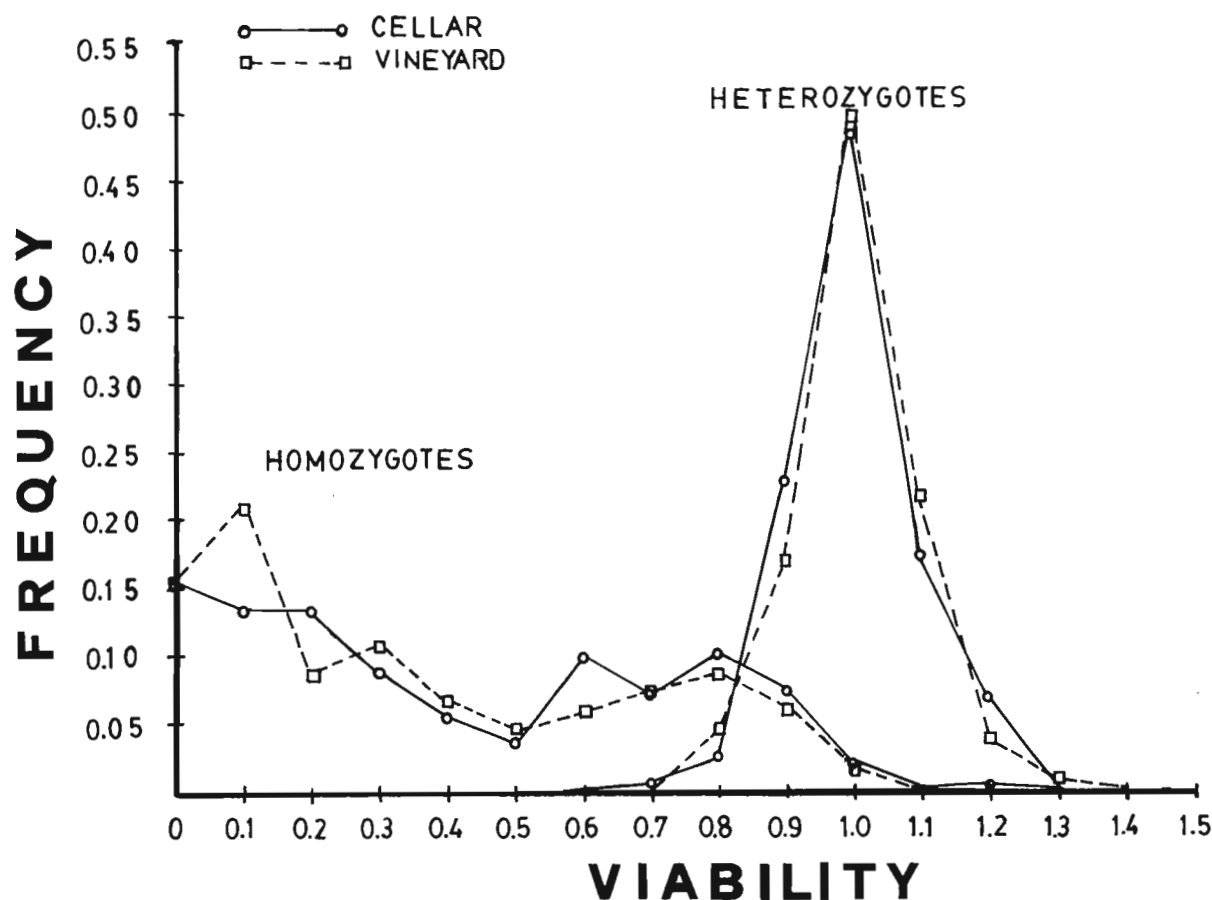


FIGURE 1. — Frequency distributions of homozygote and heterozygote viabilities

al. (1976) for the 3rd chromosome. Each relative viability was expressed as the ratio of the number of wild-types to the number of Ubx flies plus one (Haldane 1956).

The relative viabilities of all third chromosomes from cellar and vineyard lines were examined. No significant difference was detected between the patterns of frequency distributions of homozygote and heterozygote viabilities using the Kolmogorov-Smirnov non parametric test, as can be seen in figure 1.

The average homozygote viabilities, computed on the basis of average heterozygote viabilities, including lethal lines and excluding those from the cellar and vineyard populations, are given in table 1. No significant differences between means have been found.

The frequency of lethal-carrying chromosomes and the values of the D:L ratio (the detrimental load to lethal load ratio) in both populations are given in table 2. The lines showing a viability index lower than 0.1 of the average viability of random heterozygotes are classified as lethals according to Greenberg and Crow (1960).

There are no significant differences between the percentages of lethals of the two populations, which are in agreement with those of the literature.

The values of D:L relation in the two populations studied, are similar. However they are higher than those cited in the literature (D:L around 0.5-1) on *Drosophila melanogaster*. The results given in table 2 are close to the values of other *Drosophila* species, for instance in *Drosophila pseudoobscura* D:L=2.181 (Dobzhansky and Spassky 1953) and indicate that an unusually big detrimental load exists in these populations.

The allelism crosses, intra and inter populations are being made at this moment.

References: Dobzhansky, Th. and B. Spassky 1953, *Genetics* 38:471-485; Greenberg, R. & J.F. Crow 1960, *Genetics* 45:1154-1168; Haldane, J.B.S. 1956, *J.Genetics* 54:294-296; Wallace, B. 1956, *J.Genetics* 54:280-293; Watanabe, T.K., O. Yamaguchi & T. Mukai 1976, *Genetics* 82:63-82.