transition at the unstable sn. There are preferential sites of integrations for each mobile genetic element (Engels & Preston 1981). As for the elements like "copia" or "mobile dispersed genes" the average number of integration sites in D. melanogaster genome is about 50 or approximately 10-15 for the X-chromosome. Taking it into account we may suppose that the increase of X-chromosome mutability by 0.5% for one generation needs the rising of mutation rate for each of these 10-15 sites by hundreds of times. The X-chromosome non-disjunction frequency in females sn^*/Basc is also higher than in the control (Table 3). The hybrid dysgenesis systems induce the transposition of TE inherent to the genome and as a result the level of genic and chromosomal mutations is also increasing. From this point of view it is interesting that high levels of X non-disjunction had been shown for the I-R system of hybrid dysgenesis in the germ line of dysgenic F_1 females (Picard et al. 1978). Two possible explanations may be suggested: (1) the increased level of chromosome breakage, and (2) the sn locus may take part in the genetic control of chromosomal disjunction as it was established for w-z region (Robins 1981).

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Recessive sex-linked female-specific lethals at deltex locus discovered in natural populations of D. melanogaster.

Sex-specific lethals provide a remarkable means for analysis of sex determination and genetically controlled developmental processes (Baker & Ridge 1980). The recent extensive studies have shown (Belote & Lucchesi 1980): (1) such lethals are associated with a small number of loci, and (2) at the moment the majority of these mutants are male-specific; many of them are clustered at the centromere region of chromosome 2.

It is to be mentioned that such mutations are the component of sex-limited genetic load in wild populations (Dresher 1964). For example, the recessive mutation killing the males (mak-male killer, 2-54±) was first discovered in a natural population of Crimea, USSR (Golubovsky & Ivanov 1972), then the other allele (named mle) was found in the wild population from Japan (Fukunaga et al. 1975) and only the third allele was recently isolated during special extensive screening by chemical mutagenesis (Belote & Lucchesi 1980). The first male-killing lethal in chromosome 3 was also originated from a natural population (Uchida et al. 1981).

Taking into account the unequal deficit of female limited lethals, I report here the description of the mutation. It was isolated from nature in 1972 and studied briefly in 1973. In autumn 1972 together with Dr. R.L. Berg we investigated the phenotype of wild flies in several remote populations of USSR. We also searched the visible sex-linked mutations crossing all aberrant males with X-attached females. Among about 1300 males captured in Kashira population (about 100 km to the south from Moscow), we found two sex-linked mutations, yellow and "deltex vein," the latter first discovered by Dr.R.L. Berg as male having slight terminal deltas on veins. Unfortunately (but it turned out that it was lucky) at that time the common deltex mutation (dx) was absent in our laboratory. So I carried out the typical crosses for localisation of the founded vein mutation (let's designate it as dx^*). I combined dx^* with v and f markers and analyzed the progeny of crosses:

♀♀ dx^* v f /+ + + on ♂♂ dx^* v f. Results are shown at the left.

phenotypes	male progeny	female progeny
1. dx v f	57	0
2. + + +	55	51
3. dx + +	12	0
4. + v f	15	12
5. dx v +	20	0
6. + + f	23	13
7. dx + f	4	0
8. + + +	7	12
	193	88

The two facts are evident from these recombinational data: the lethality of the homozygous dx^*/dx^* females and localisation of dx^* in the region of 17 m.u. where deltex locus is placed. Then I received the "ec dx" line from Oregon stock

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 $\bar{X}\bar{X}$/0 females. In the progeny the 64 ♀♀ and 63 ♂♂ appeared; it meant the normal viability of dx^{fl}/0 males. In the progeny of other similar cross: 1♀ +/dx^{fl}/ x $\bar{X}\bar{Y}$/0 ♂♂ 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 supressors can normalize both phenotypic and lethal expression. At last, dx^{fl} is very convenient mutant for balancing the stocks like C1B or FM3. In our laboratory we are steadily keeping the C1B stocks by crosses C1B/dx^{fl} x dx^{fl} from 1973.

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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 $Ubx^{130}e^s$/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 $Ubx^{130}e^s$/CSb females.

All third chromosomes were maintained, as lines, at 19º balanced with TM2(Ubx^{130}) 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.

Population	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.

Population	Total	% lethals	D:L
Cellar	155	23.85	2.58
Vineyard	145	28.27	2.40