Milkman, Roger D. Syracuse University, New York. cve polygenes in laboratory mutant stocks.

Combinations of genes producing the cve phenotype have arisen in the progeny of crosses among the following laboratory strains: Ore R, y ct ras f, od sy, dp cn bw, and ey. Inbreeding and intrachromo.

somal recombination increase the numbers of cve flies, as is shown in the table. All matings were 4 x 4 except that inbred lines began with pair matings. In each inbred sample, 100 flies from each of 10 replicate lines were examined. The table shows the number of samples with at least one cve fly per thousand (at 18° C) and the percentage of cve flies. Comparison results are given for flies collected in nature (previously reported). Data for the inbred F_6 are not quite complete. Here the increased percentage of cve flies was found in spite of a decrease in incidence in the progeny of the major contributing line, od sy X y ct ras f, which contained 58 cve flies in the mass F_6 and 46 in the inbred F_6 . Rapid response to selection for cve in the od sy X y ct ras f mass progeny is seen from the penetrance after 4 generations: males, 23%; females, 70%. Penetrance is much lower at 25° .

	No. of Samples		% cve
	Total	With cve	
Parental stocks	6	3	0.07
Parental inbred F ₂	6	3	0.07
F. 2	1 5	2	0.01
Mass F	1 5	4	0.06
Inbred ² F ₂	1 5	6	0.19
Mass F ₆	1 5	7	0.49
Mass F Inbred F ₂ Mass F ₆ Inbred F ₆	1 5	1 2	0.65 (est.)
F ₂ 's from wild inseminated females			
inseminated females	70	54	0.70

Traut, H. Universität Münster, Germany. Pre-existing Y-suppressed lethals in Drosophila melanogaster.

Individual In49 v sn $^{\rm X2}$ B males were crossed with X•Y females (genotype Y $^{\rm S}$ •X InEN y v•Y $^{\rm L}$). F₁ virgins were mated singly to Y $^{\rm S}$ •X InEN y v•Y males (no free Y). The F₂ In49 v sn $^{\rm X}$ 2 B males carry no

Y. If this class of males is missing this may be due either tố an "orthodox" lethal or to a lethal, the expression of which is suppressed by the Y-chromosome. In order to distinguish between these two possibilities, F₂ virgins carrying the In49 v sn B chromosome were mated to males with a free Y. If in the progeny of these females the In49 v sn B male class was missing again, an orthodox lethal was present, if not, a Y-suppressed lethal. The progeny of each P male has been scored separately. The Y-suppressed lethals found by us were not distributed randomly over the total offspring but could be traced back - even through several broods - to only a few P males. Therefore, these Y-suppressed lethals pre-existed already in the P males, from which they were derived; i.e. they did not arise as new mutations in the germ cells of these males. From 78 P males tested, 6 carried a pre-existing Y-suppressed lethal (7.7%). This high accumulation of Y-suppressed lethals seems to have been enabled by the fact that the In49 v sn B males were kept in the stock cultures with X·X Y females. Thus spontaneously arising Y-suppressed sex-linked lethals are conserved instead of being eliminated through homozygosity of the X-chromosome. Since these experiments were not especially designed to detect Y-suppressed lethals but for another purpose, the nature of these lethals has so far not been analyzed. (Work supported by USAEC grant A T 11-1-195 to Dr. H. J. Muller and co-workers.)