tion contained 2% agar to set it in the ring. Six amino acids which can stimulate sugar receptor of larger flies were used (Shiraishi and Kuwabara 1970). About 100 D. melanogaster, 24-48 hours old, allowed to take only water for 24 hours before experiments, were introduced into the petri dish and the distribution of the flies on the rings was examined by photographing them 4 times at 30-minute intervals (for details see Isono and Kikuchi 1974b). Isogenic strain AA75-3 (Isono and Kikuchi 1974b) and wild-type laboratory stock derived from natural populations were used. The results are shown in Table 1.

Table 1. Response of D. melanogaster to six L-type amino acids presented at the concentration of t x 10^{-2}M.

Amino acid	Strain	Sex	Number of flies observed on the amino acid	Number of flies observed on the phosphate buffer	χ^2	P
L-Leucine	AA75-3	female	103	82	2.38	>0.1
		male	30	38	0.94	>0.7
L-Valine	AA75-3	female	139	45	48.02	<0.001
		male	93	61	6.65	<0.01
L-Methionine	AA75-3	female	276	138	46.00	<0.001
		male	116	79	7.02	<0.01
L-Phenylalanine	Wild	female	219	76	69.32	<0.001
		male	323	103	113.62	<0.001
L-Isoleucine	AA75-3	female	230	92	59.14	<0.001
		male	119	59	20.22	<0.001
L-Tryptophan	AA75-3	female	98	74	3.35	>0.05
		male	37	38	0.01	>0.9

D. melanogaster could discriminate L-valine, L-methionine, L-phenylalanine, and L-isoleucine from the buffer control but they did not seem to taste L-leucine and L-tryptophan at the concentration tested. Because 5 x 10^{-2}M is the concentration at which the magnitude of the electrophysiological responses of larger flies reached maximum and usually behavioral assay is more sensitive than electrophysiological assay, there must be a large difference in stimulating ability between the former four and the latter two amino acids. However, further study is necessary to conclude that D. melanogaster is not able to taste L-leucine and L-tryptophan at all. Shimada and Isono (1978) reported two of these six amino acids, L-phenylalanine and L-tryptophan, differed from the other four in such a manner that these two reacted with furanose site of the fleshfly. This classification, however, did not agree with our data here. It would be interesting to know the relationship of these six amino acids and the furanose site in D. melanogaster by electrophysiological technique.

Temin, R.G. and R. Kreber. University of Wisconsin, Madison, Wisconsin. A look at SD (Segregation Distorter) in the wild population in Madison, Wisconsin, more than 20 years after its initial discovery there.

Flies were collected in the fall, 1979, for a study of how SD behaves in nature and to understand the factors determining its frequency. With the recent work on hybrid dysgenesis in our laboratory (Engels 1979-1980) and with the more detailed understanding of the substructure of the SD region (Hartl and Hiraizumi 1976; Ganetsky 1977), it became of interest to see what bearing these might have on the course of SD in wild populations.

There were two trapping sites in Madison, in woods near the homes of R.G. Temin and J.F. Crow. Since the observations, listed below, were similar in the two subpopulations, they have been combined:

1. SD is still present in Madison, in 20 chromosomes among 741 screened. This frequency of 3% is, in fact, the same as it was in 1956 (6/183) and must represent the equilibrium frequency. The average "k" value, representing the degree of distortion, was 0.95, from the

ratio of 1603 + : 86 cn bw progeny from individual test crosses of +/cn bw ♂♂, where + is derived from a wild-caught male.

2. Cytological analysis revealed that 19 of the 20 SD chromosomes contained the small pericentric inversion In(2LR) 39E1.4-5.8; 42A-B, as well as the large distal Nova Scotia inversion in the right arm. This set of inversions is of the SD-72 type, as designated for the original SD's. The remaining one of the 20 was of the SD-5 type, namely with In(2R)NS and a small proximal inversion also in the right arm, In(2R)45D2-E1; 49 A2-B1, but lacking the pericentric inversion. As controls, 23 SD^+ lines had no second chromosome inversions and two SD^+ lines had In(2R)NS only. (Two of the SD lines from Crow's collection, in addition to being SD-72, had a highly complex set of second chromosome inversions; the more complex of these also showed a reduced k value of 0.66 on further testing.)

Thus, nearly all the SD chromosomes are now SD-72, in contrast to 1956, when five of the six were SD-5 and one was SD-72. This difference in relative frequencies of SD-5 and SD-72 in the two collections was significant at p = 0.0005, by Fisher's Exact Test. The chromosome with the pericentric inversion has become predominant, maintaining the tight linkage between the SD and Rsp alleles in that region.

Even before the pericentric inversion was discovered by Lewis in 1962, Hiraizumi, Sandler and Crow (1960) predicted, from population cage studies, that SD-72 might replace SD-5. Finding that SD-72 (which was lethal-free) maintained a higher equilibrium frequency than SD-5 (lethal bearing) in cages, they suggested that in nature SD-72 had just arisen as a derivative of SD-5 but had not yet in 1956 had time to replace it. Studies are underway to determine the homozygous viabilities of the newly collected SD's.

3. Male recombination was also detected in the SD screening test crosses of the F_1 +/cn bw dysgenic male, indicating that components of hybrid dysgenesis are present in these populations. Among 27,216 progeny of such heterozygous males, 82 were either cn or bw, a frequency of 0.3% recombinants. These occurred in SD lines as well as in SD^+ lines, in the same approximate frequency. Recombinant progeny from heterozygous fathers continued to appear even in the F_4. In the SD lines the ratio of bw to cn recombinants (67:10 among 14,536 progeny) was significantly greater than in the non-SD lines (44:33, among 25,523), with p = 0.00003. This supports the notion that segregation distortion and hybrid dysgenesis can occur simultaneously.

It is interesting to speculate whether male recombination was occurring in the SD screening of the Madison population in 1956. The original isolation of SD by Hiraizumi during a study of lethal heterozygotes (1960) was from +/cn males which would not have revealed this. However, other tests in our laboratory in that era (Mange 1961; Greenberg 1962) to screen wild populations for SD did use +/cn bw dysgenic F_1 males, but recombinants were not reported. We do not know if they were too infrequent or were overlooked, or actually did not occur. However, this raises the question of whether the situation has changed since that time with regard to components of hybrid dysgenesis, either in the cn bw lab stock or in the wild flies themselves.

4. Previous studies have demonstrated suppressors of SD activity, on the X and on other autosomes, as well as on the second chromosome (non-allelic and at the Rsp locus itself). To estimate directly how active SD is in a wild genome, k values were measured for the 20 wild caught males revealed to be harboring SD. This was done by testing a number of F_1 sons (14-45) of each such line. If SD was fully operative in the P_1 male (k = 1) then all of the F_1 sons would have inherited SD and themselves have a high k value. If SD was completely suppressed in the P_1 male (k = 1/2) then about half of the F_1 sons would have a high k.

SD appeared to be fully active in only about 1/4 of the cases; in fact, there was a wide range of distortion in the wild males. Of the 20 lines, 8 showed no distortion in the P_1 male (k less than 0.57), 5 showed high distortion (k = 0.87 or greater), 6 showed reduced distortion (k between 0.63 and 0.77), and one gave too few sons to test.

5. Another approach to the question of how much distortion occurs in nature was to screen SD^+ chromosomes from Madison for sensitivity vs. insensitivity. A strongly distorting SD-5 bw recombinant, provided by Dr. Barry Ganetsky, was used to test 122 non-SD chromosomes, as +/R(SD-5 bw), in the fourth generation after crossing to laboratory stocks, providing an opportunity for some of the unlinked modifiers to be crossed out. Of the 122, approximately 82 were sensitive to SD-5 action (k = 0.83 or greater), 24 had reduced sensitivity (k between 0.61 and 0.80), and 16 appeared to be in the range of insensitivity (k less than 0.60). Thus, the maximum frequency of insensitive chromosomes is about 13%; to establish these as having true Rsp^{ins} alleles would require further special tests. This figure is substantially lower than the 45% found by Hartl and Hartung (1971) in more specific and thorough testing. This variability among populations with regard to the non-SD chromosome bears further investigation.

References: Hartl, D.L. and Y. Hiraizumi 1976, in: The Genetics of Drosophila, 1, Novitski and Ashburner, eds.; Ganetsky, B. 1977, Genetics 86:321; Engels, W.R. and C.R. Preston 1980, Genetics 95:1 (in press); Hartl, D.L. and N. Hartung 1975, Evolution 29:512; Hartl, D.L. 1970, Can. J. Genet. Cytol. 12:594; Hiraizumi, Y. 1971, PNAS 68:268; Hiraizumi, Sandler and Crow 1960, Evolution 14:433.

Tobari, I. and M. Murata. National Institute of Radiological Sciences, Chiba-shi, Japan. Fertility load and frequency of lethal second chromosome in Drosophila populations with radiation histories.

It has in general been considered that most of the radiation-induced mutations are sooner or later eliminated from a population by the acts of natural selection of the irradiation is suspended. The purpose of this study is to see a recovery of genetic damages caused by the radiation-induced mutations by estimating the amounts of fertility load and the frequency of lethal second chromosomes in the populations with radiation histories. Experimental populations of D. melanogaster used in this study were identical with those reported by Murata and Tobari (1973). Three experimental populations, B, C and D, were derived from the irradiated population which had been successively exposed to 5,000r of X-rays in every generation (Tobari and Murata 1970). The populations B, C and D have been subjected to the cumulative radiation exposures of 25,000r, 50,000r and 75,000r, respectively. These populations were maintained for 75-77 generations without X-irradiation before the present experiment was carried out. The frequency of lethal second chromosomes was estimated by the Cy/Pm technique, using about 200 males taken from each of the experimental populations. To estimate the fertility load the homozygous and heterozygous flies for wild-type second chromosomes were reconstituted. For each of approximately 100 chromosomes, in homozygous as well as heterozygous condition, 10 males and 10 females were tested. Each wild-type male (or female) was mated individually to three cn bw virgin females (or males). After one week all cultures were examined for evidence of fertility. A vial was classified as sterile (S) if there were no larvae or pupae present and the parents were alive. Cultures bearing progeny were classified as fertile (F). In some of the cultures which contained no progeny, the parent of interest was dead; this type was recorded as D.

The frequency of lethal second chromosomes in a non-irradiated (control) population was estimated to be 17.8 ± 1.9%, while it was 30.7 ± 3.7%, 32.7 ± 3.8%, and 32.5 ± 3.4%, respectively, in populations B, C and D. The difference in frequency between the control populations, A, and the experimental one is statistically significant.

The proportion of fertile cultures among the total fertile and sterile cultures was computed and these fertility ratios, F/(F+S), for males and females are given in Table 1. In all

Table 1. The mean fertility ratios, F/(F+S), in the irradiated and control populations.

Population		A: Control	B: 25KR-77G	C: 50KR-75G	D: 75KR-75G
Males:					
Heterozygotes	n	99	72	100	117
	F/(F+S)	0.934±0.016	0.957±0.012	0.868±0.021	0.881±0.023
Homozygotes	n	101	99	102	105
	F/(F+S)	0.866±0.020	0.892±0.025	0.789±0.032	0.748±0.031
Homozygotes excluding complete steriles	n	99	97	94	95
	F/(F+S)	0.884±0.016	0.916±0.019	0.856±0.023	0.856±0.024
Females:					
Heterozygotes	n	105	97	101	120
	F/(F+S)	0.969±0.008	0.969±0.008	0.941±0.011	0.961±0.007
Homozygotes	n	104	99	100	100
	F/(F+S)	0.909±0.019	0.871±0.028	0.842±0.026	0.817±0.030
Homozygotes excluding complete steriles	n	102	95	94	93
	F/(F+S)	0.926±0.014	0.907±0.022	0.896±0.015	0.896±0.021

F = fertile, S = sterile, n = number of chromosomes tested