McCrady, W.B. University of Texas at Arlington. Search for variation in response to CO_2 in wild-caught Drosophila melanogaster.

Collections of D. melanogaster were completed during the latter part of June and the first part of July, 1973, at three sites in Dallas and one in Arlington, Texas. A total of 564 strains were developed from isolated wild-caught females. Progenies were reared at 19-20°C to prevent temperature curing of CO_2 sensitivity symptoms and were first screened for any abnormal response to CO_2 by exposure to pure carbon dioxide at 19-20°C. A total of eighty (14.2%) of the strains tested showed departures from control (Oregon) resistant flies in proportion and/or time required for recovery.

For further screening, 72 of the above strains were treated with CO_2 for fifteen minutes at 9°C and their recovery behavior was recorded. Twenty of these strains showed departures in response to CO_2 from that observed in resistant flies. In each of the seven strains shown on the left in Table 1 over 30% of the flies were killed by CO_2 treatment; no recovery occurred later than fifteen minutes following exposure as is typical for classical sensitivity caused by virus sigma. Two strains (80 and 210) seem to exhibit stabilized CO_2 sensitivity and will require no selection. Selection will be carried out in the other five strains in an attempt to establish new stabilized lines.

Table 1. % recovery following CO_2 treatment at 9°C for 15 minutes.

Strain	n	15 min	30 min	1 hr	2 hrs
7	150	50%	50%	-	-
80	52	0%	0%	0%	0%
101	39	18%	18%	18%	18%
210	16	0%	0%	0%	0%
375	42	14%	14%	14%	14%
379	18	56%	56%	-	-
381	18	67%	67%	-	-

Strain	n	15 min	30 min
18	100	70%	85%
19	100	80%	90%
20	150	73%	83%
37	150	83%	87%
41	125	84%	88%
71	70	93%	86%
72	90	83%	94%
74	140	86%	93%
84	70	94%	94%
106	90	94%	96%
257	33	88%	94%
469	21	76%	90%
481	20	80%	85%
Oregon	75	100%	100%

If it is found that these seven strains are the only ones with classical CO_2 sensitivity, the proportion (1.24%) is slightly lower than that determined by Williamson (1961) for wild-caught D. melanogaster in Nebraska. He found 1.6% sensitives in 6,300 flies tested. However, interesting departures from resistant type behavior were observed in thirteen other strains as shown on the right side of the table. In two of these (strains 71 and 84) less than 100% recovery occurred during the first 15 minutes following CO_2 exposure and no recovery occurred later; in fact, fewer flies were able to stand after an additional 15 minutes in strain 71. These may be non-stabilized strains. Recovery behavior in the other strains suggests delayed-recovery as described by McCrady and Sulerud (1964). In all cases recovery was slower than for control flies and less than 100% had recovered within 15 minutes. Furthermore, additional recovery was observed during the next 15 minutes. Some of these flies may be homozygous or heterozygous for gene Dly, the determiner of delayed-recovery. Testing of this hypothesis is now being attempted.

At the present time only two delayed-recovery stocks exist, TDR (Texas Delayed Recovery) and TDR-B. The latter stock has only recently been developed by selective breeding and testing for Dly, although the strain was started with a female collected at the same time and site (Pittsburg, Texas in 1959) as the originator of TDR. Delayed-recovery in TDR-B has been shown to be determined by a gene apparently identical in function and location to that responsible for the delayed-recovery phenomenon in TDR. It appears evident that the two females were a part of the same gene pool. Therefore, the establishment of delayed-recovery strains from other areas seems desirable and will be attempted by selection within the aberrant stocks now available. Investigation of the causative mechanism of delayed-recovery in different strains could be very informative in illumination of the broad question of the relationship

between gene Dly and virus sigma.

References: McCrady, W.B. and R.L. Sulerud 1964, Genetics 50:509-526; Williamson, D.L. 1961, Genetics 46:1053-1060.

Gold, J.R. and M.M. Green. University of California, Davis, California. mu - a mutator gene in Drosophila melanogaster.

In two previous reports, an apparently new mutator gene, mu, in D. melanogaster was identified and genetically characterized (Green, 1970; Green and Lefevre, 1972). In these reports, it was shown that mu significantly increases the reversions of the sex-linked mutants y^2 and f^{3N} to their respective wildtype alleles, and the frequency of sex-linked lethal mutations in homozygous mu females.

In addition to the frequent reversions of y^2 and f^{3N}, several other visible mutations have been recovered from experiments using single P_1 homozygous mu females. Some of these "forward" mutations are listed in Table 1, and are presented to demonstrate the influence on spontaneous mutability of the mutator gene. Most of the newly recovered mutations were progeny tested to determine the origin, i.e. somatic or germinal. Multiple events or clusters

Table 1

Forward visible mutations recovered from experiments using homozygous mu females.

No	Phenotype of the mutation	Number of occurences	Somatic or germinal**
1	achaete	2	?
2	bithorax-like	1	S
3	Beadex	1	S
4	Blistery wing	1	S
5	bulgeing eye (extreme)	1	G
6	cut wing	2*	G
7	Delta wing	4*	G
8	Dicheate-like	1	S
9	Hairless	5*	?
10	hairy eye (extreme)	10	S
11	held-out wing	1	G
12	Lobe or reduced eye	many	S
13	lozenge spectacle	1	sterile
14	Minute	12*	S,G
15	Notch	16*	G
16	roughened eye	6*	G
17	scute	2	S
18	zeste eye color	1	sterile
19	Ultrabithorax-like	2	S
20	several bristle irregularities	many	-
21	several eye shape mutations	many	-
22	several synanders (mitotic loss)	many	-

* Recovered as clustered events

** Somatic - S (not recovered in F_1 progeny tests)
Germinal - G (recovered in F_1 progeny tests)

from single P_1 females were found in several instances and are noted in the Table. Three conclusions can be drawn from the results: 1) mu induced mutability is not gene or allele specific; 2) mu induced mutations occur in both somatic and germinal cells; 3) at least some of the mu induced mutations occur premeiotically as evidenced by the clustered mutations. All three observations were made previously and are extended by the observations reported here.

References: Green, M.M. 1970, Mutation Res. 10:353-363; Green, M.M. and G. Lefevre, Jr. 1972, Mutation Res. 16:59-64.