Mukai, T., L.E. Mettler, and S.I. Chigusa. North Carolina State University, Raleigh, North Carolina. On the linkage equilibrium of isozyme genes in a Raleigh, N.C. population of D. melanogaster.

Three hundred and four second chromosomes were examined for alcohol dehydrogenase (ADH), α-glycerophosphate dehydrogenase-1(α-GPDH-1), and malic dehydrogenase-1(MDH-1). The frequencies of the fast alleles (F) are 0.237± 0.024 for ADH, 0.819±0.022 for α-GPDH-1, and 0.033±0.010 for MDH-1; hence, these three genes are located in the left arm. Using all chromosomes, no linkage disequilibrium was discovered between any two loci.

Two polymorphic inversions were discovered: Inversion A (breakage points are approximately 51-D and 57-A, and the frequency is 30/304) and Inversion C (breakage points are approximately 22-D and 33F - probably the same as In(2L)Cy but not associated with Cy with a frequency of 24/304). Although Inversion A is located in the right-arm of the chromosome, the associations between the inversion and the genes in question were examined. In ADH locus, F genes seem more associated with Inversion A than the chance, but not significantly so ($\chi^2_{d.f.=1}=3.45$, $0.05<P<0.10$). In the remaining two loci, no close association was detected.

With respect to Inversion C, a significant association was discovered between the inversion and S genes of the ADH locus ($\chi^2_{d.f.=1}=8.12$, $P<0.005$), although it is located outside the inversion (the distance is very small). This is linkage disequilibrium due either to some interaction between the inversion and the gene in question or to the lack of recombination between them. (The random genetic drift might not be significant because the effective population size has been estimated to be of the order of 10^4.) On the contrary, it was not possible to detect association between the α-GPDH-1 alleles and Inversion C although this locus is most probably located in this inversion. The MDH locus cannot be examined because of the low frequency of F genes.

Linkage disequilibrium was not detected using only the 241 completely inversion-free chromosomes.

Schalet, A. and V. Finnerty. University of Connecticut, Storrs, Connecticut. Is a deficiency for maroonlike lethal?

Recently, Lifschytz and Falk have presented 3 versions of a complementation map of the proximal region of the X chromosome of D. melanogaster in which the maroonlike locus as the only visible unit is a prominent feature. (DIS 1968; Mutation Research 1968, 1969). All three maps describe combinations of overlapping deletions yielding viable females that showed a "mal" phenotype. It was concluded that mal deficiencies were not lethal.

Because of our interest in the mal locus, Lifschytz and Falk were kind enough to send us four of their "mal" deletion stocks. We can report that none of the deletions have proven to involve the mal locus when tested against our extensive collection of viable and lethal mal mutants. We can confirm that females heterozygous for two of the deletions, A118/Q539 do survive and manifest a mutant phenotype. The eyes of these females sometimes display the mutant coloration as a large, irregular area. (The uneven distribution of pigment is clearly seen as a splotch in the eyes of pupae.) Adult females often have "material" protruding from the vagina and abnormal wings. Additional tests with non-mal mutants have located the locus in question to the right of mal and immediately proximal to lf. Salivary analysis by Lefevre shows that A118 and Q539 chromosomes carry deletions that overlap for at least band 19E7. The real maroonlike is located distal to lf, proximal to mel, and has been positioned cytologically at 19C4-19D3. (See note of Schalet, Lefevre and Singer in this issue.)

The question posed by the title of this note remains to be answered. Chovnick, Finnerty, Schalet and Duck (1969) have examined genetically 18 lethal mal mutants and all tested combinations have proved lethal. However, all lethal mal mutants behave like deficiencies in that each is lethal with at least one non-mal lethal locus adjacent to mal. Furthermore, all 7 deficiencies thus far examined by Lefevre show cytological deletions. Yet, the possibility that complete loss of mal alone may be lethal cannot be ruled out. Mal mutants lose the activity of three enzymes, xanthine dehydrogenase, aldehyde oxidase and pyridoxal oxidase. The loss of xanthine dehydrogenase activity alone is insufficient to produce lethality. At the rosy locus eye color mutants lacking only XDH activity are viable. Complete loss of the rosy locus is probably not lethal. This inference is drawn from the observation that the heterozygote, ry^{54}/ry^{74}, two probable overlapping deficiencies, is viable.