

Frye, Sara H. P.O. Box 267, Irvine, Kentucky. Open letter to Drosophila geneticists pertaining to the simultaneous occurrence of a double marker mutant in *Drosophila melanogaster*.

On April 24, 1965, a single exceptional male, $Y/y \text{ ac In49 B}^{M1*}$, was recovered among progeny whose origin were parents consisting of relatively young males of genotype, $Y/y \text{ ac In49 B}^{M1}$, which had been exposed to 2 kr (i.e., 2000 r delivered at 400 r/minute, 210 kvp., 15 ma., 1mm A1 + 1/2 mm

Cu filter) and mated to a series of virgin females of different genotypes, one of which was $Y/y \text{ f} =$ virgin females (i.e., phenotypically yellow wings, yellow body color, yellow-two (?) bristles, and forked bristles). Simultaneous and identical controls did not yield any exceptional F_1 fertile yellow and/or achaete males even though the same precautions were used to avoid environmental selection against any exceptional phenotype of spontaneous origin occurring among the expected phenotypes.

In49 B^{M1} chromosomes are representative of chromosomes of normal structure for the yellow region, i.e., there is no chromocentral heterochromatin adjacent to the yellow region (as in scute-8 chromosomes) and B^{M1} chromosomes were used by Belgovsky for this reason. In my opinion, yellow mutants induced in B^{M1} chromosomes are a better test in regards to the problem as to whether two or more closely linked markers are ever involved in mutational events induced by X-irradiation than yellow mutants induced in scute-19i chromosomes. Scute-19i chromosomes contain an insertion from the distal tip of the X-chromosome bearing the normal alleles of the mutant markers yellow, achaete, scute, and scute-lethal into the second chromosome between the normal alleles of the mutant markers, dumpy (2, 11.0) and clot (2, 16.5). Consequently, the yellow region is now free of chromocentral heterochromatin. As to the presence of intercalary heterochromatin in the neighborhood of the yellow region in scute-19i chromosomes or In49 B^{M1} chromosomes I am not qualified to give an opinion (see Prokofyeva-Belgovskaya papers between 1930-1939). I sincerely hope that several (not just one) investigators will examine this stock cytologically (see Frye, materials available, this issue, DIS 41) and report their findings to the other *Drosophila* workers at the 8th annual *Drosophila* Conference which meets in Chicago, May 27-29, 1966.

* $y \text{ ac In49 B}^{M1}$ chromosome is hereafter designated as $y \text{ ac In49 B}^{M1}$ to indicate the simultaneous occurrence of the double marker mutant phenotype* (see Frye, new mutants, this issue DIS).

I proceeded with a reverse mutant phenotype experiment by irradiating young males of genotype, $sc \cdot Y/y \text{ ac In49 B}^{M1}$ at 2 kr or 4 kr and mating them to $Y/y \text{ f} =$ virgin females and scoring for non-yellow (symbolized y^-) and/or non-achaete (symbolized ac^+) male-viable phenotypes. Between August 31, 1965 and September 8, 1965 I recovered some non-achaete (ac^+) phenotypes, but I did not recover any non-yellow (y^-) phenotypes or any non-yellow, non-achaete ($y^- \text{ ac}^+$) phenotypes. Later observations made in the author's office in Irvine, Kentucky showed that some of the non-achaete phenotypes (ac^+) were fertile on progeny-testing and that several forward mutant phenotypes* had been superimposed in the irradiated $y \text{ ac In49 B}^{M1}$ chromosomes.

The pattern of investigation (multiple genetic perspectives) employed in approaching the problem of structure and function of the yellow-achaete region within the framework of a replicating, functioning chromosome is considered to be of utmost experimental and interpretative value.

I acknowledge with great pleasure the assistance in much of the routine work by my son, Mark Evan Frye. I want to thank Dr. Burke Judd for space in his laboratory in the Genetics Foundation at the University of Texas, Austin, Texas. I would like to thank Drs. Fabergé (1962) and Schultz (1963) for emphasizing in written communication to the author the importance of the genetic scheme and the structure of the chromosome used in the recovery of yellow marker mutants.

* These mutants will be printed in the next issue of DIS.