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Allelism of second chromosome melanotic tumor genes.

To test for suspected allelism of second chromosome mutants of diverse origins which produce melanotic tumors, reciprocal pair matings were made in all combinations of wild type (Caribbean, a virtually tumor-free strain), tu bw, tu bw;st su-tu, tu 55g (from Jacobs), tu B_3, tu A_2, tu^K, and Frd. In crosses of the other strains with Frd, a dominant lethal which is phenotypically distinct from the others, half the offspring showed Frd as expected; there was no evidence of allelism with the other loci, and the Frd data are not included in the table below. The remainder of the tumor-initiating genes are essentially recessives, and the suppressor of tu bw (su-tu) behaves as a semi-dominant in most matings.

The larvae resulting from these matings were reared on standard maizemeal-molasses medium seeded with live yeast, and incubated at 25°C. It is well known that penetrance and expressivity of the trait in melanotic tumor strains vary widely, even within highly inbred strains cultured under defined conditions, and depend on temperature, genetic background, and nutrition. In the summary table below, "-" indicates that fewer than 10% of the offspring possessed tumors, "+" that 11-40% were tumorous, and "++" that more than 41% of the flies were tumorous.

Table 1: Tumorous offspring from reciprocal matings between strains with second chromosome loci initiating melanotic tumor formation.

	FEMALES						
MALES	wild	tu bw	tu bw;st su-tu	tu 55g	tu B_3	tu A_2	tu^K
wild	-	-	-	-	-	-	-
tu bw	-	++	++	++	+	-	-
tu bw;st su-tu	-	++	+	+	-	-	-
tu 55g	-	++	+	++	++	-	-
tu B_3	-	++	+	++	++	-	-
$tu_K A_2$	-	-	-	-	-	-	-
tu^K	-	-	-	-	-	-	-

Thus it is seen that the second chromosome genes tu bw, tu 55g, and tu B_3 behave as if they were allelic, whereas tu A_2 clearly is not, a fact demonstrated earlier by Barigozzi and di Pasquale (1956, Ist. Lomb., Rend. Sci., 90:484). It also appears that the suppressor of tu bw in heterozygous condition exerts as great or greater effect on tu 55g and tu B_3 as on tu bw. The penetrance of tu A_2 is normally quite low, and that of tu^K is even lower when reared on standard media, although its penetrance is increased on media deficient in certain nutrients (Sang and Burnet, 1963, Genetics 48:235).

Since Glassman (1956, DIS 30:116) has already reported allelism between tu bw and tu e^{11}, it would seem that a sizable group of second chromosome melanotic tumor-producing loci are probably allelic; the designations of these loci should be standardized to reflect these allelic relationships.

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Comparative visible mutation rates in the X-chromosome of Drosophila at various stages in oogenesis.

Among approximately 40,580 female progeny of Muller's Vix stock (heterozygous at 13 visible loci in the X chromosome), a total of 77 mutations (45 whole body and 32 mosaic) were recovered at the visible loci under study, or about 1 in 530, after treatment of the female parents with CB 1506 (2-chloroethyl-methanesulfonate) vapor, and about a 20% sex-linked lethal rate from a smaller sample. Most of these mutations were recovered in the first five three-day brooding periods, but none among the relatively few progeny in the first brood, in which most of the oocytes treated in stage 14 would be represented. On the basis of work reported by R. Valencia, we calculate that the mutation rates at the same loci as the above (but in Muller's "jynd" stock) were about 10 times as high after X-ray treatment of stage 14 oocytes (the stage