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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.

Altenburg, E. and L. S. Browning. University of St. Thomas, Houston, Texas.

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

studied by Valencia) than the rate we recovered after treatment of the earlier stages of oogenesis with an equivalent mutagenic dose of CB 1506. The "equivalence" is based on the lethal rates induced by the agents under study, and on the assumption that the lethal rates induced by radiation of earlier stages in oogenesis (not obtained in our experiments) would be about the same as the rates usually recovered after radiation of the mature sperm cells, though actually they are somewhat lower in the former than the latter case. The visible rates, relative to the lethal, were not widely different after X-ray treatment of stage 14 oocytes, as reported by Valencia, and CB 1506 treatment of the earlier stages, herein considered. The extreme radiosensitivity of stage 14 oocytes has been pointed out by Valencia.

Scharloo, W., M. S. Hoogmoed and A. E. ter Kuile. Universities of Groningen and Leiden, Netherlands. Disruptive and stabilizing selection an a cubitus interruptus mutant.

Scharloo (1964) reported that disruptive selection with random mating, practised on the 4th vein interruption of $ci^{D-G}$, caused a large increase of the genetic variance. Stabilizing selection caused a decrease of both the genetic variance and the environmental variance. In both lines the within fly variance did not change. In a new series of experiments from the same Pacific base population the following selection lines were made:

1. A line maintained under disruptive selection with random mating. The increase in genetic variance was even larger than in the first experiment. Extreme individuals began to overlap with wild type at generation 15. They showed extra venation, probably caused by a plexus allele. At the other side of the frequency distribution individuals with a very short (about 20% present) 4th vein appeared in generation 6. They lacked the 2nd cross vein. Even in the absence of $ci^{D-G}$ the cross-vein defect and an interruption of the 4th vein occurred. This phenotype is dependent on the presence of the right part of the 2nd chromosome distal to c. The within fly variance increased slightly.
2. Two lines under disruptive selection with negative assortative mating (forced mating of high and low extremes). In both lines the genetic variance, the common environmental variance and the within fly variance increased.
3. Two lines under stabilizing selection. In both lines the phenotypic variance decreased as a consequence of a decrease of the genetic variance, but in one line it stayed relatively high throughout the whole experiment (15 generations). In the other line environmental variance and within fly variance decreased as well as genetic variance.

Scharloo, W. and W. Vreezen. Universities of Groningen and Leiden, Netherlands. Correlated responses in 4th and 5th vein selection in Hairless mutants.

Scharloo and Vreezen (DIS 40:63) reported on selection for a large 4th vein interruption caused by Hairless mutants. In these lines selected after introduction of the mutant in the Kaduna and Pacific cage populations, measurements of the 5th vein were also made. The results show:

1. Pacific H selection 1. Only a minor response of the 5th vein.
2. Pacific H selection 2. A strong correlated response of the 5th vein so that after generation 8 practically no 5th vein material was present posterior to the 2nd crossvein, and individuals with a break proximal to the crossvein appeared.
3. Pacific $H^{57c}$ selection. In the first 5 generations both the 4th and 5th vein interruption increased, but after generation 8 the 5th vein increased in length to about its original value.
4. Kaduna $H^{57c}$ selection. 2 lines were selected concurrently for larger 4th vein interruption and larger 5th vein interruption respectively. The correlated responses of the veins not selected for, were very small compared with the direct responses.

Thus different base populations and even different selections from the same base population may show a different pattern of correlated response.