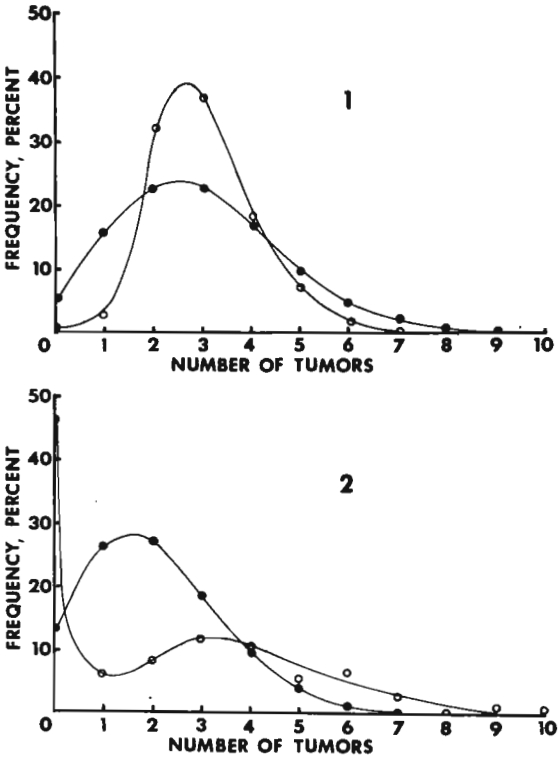
Bryant, P.J. University of California, Irvine, California.* Statistical distribution of melanotic tumors.

Many studies of melanotic tumor genes have employed penetrance as a measure of the expression of the gene. However, this becomes inaccurate when penetrance approaches 100%. In such cases, it is necessary to use a parameter related to the number of tumors developed per fly, and in order to do this effectively it is desirable to know how the tumors are distributed throughout the population.



The a priori expectation is that tumor distribution would be of the Poisson type, and this involves assuming that tumors form independently; that is, the probability of tumor formation is not altered by the formation of other tumors in the larva. In some stocks, the distribution of tumors differs markedly from the Poisson distribution. Fig. 1. shows a population of tu bw; $+^{su-tu}$ and Fig. 2. a population of Oregon K, both grown under sterile conditions. These distributions (hollow circles) are compared with Poisson distributions (solid circles) having the same means as the observed populations. tu bw;$+^{su-tu}$ shows a smaller spread than the Poisson curve, and Oregon K shows a wider spread. This is true for all populations of these strains studied, representing a wide range of expression values.

These results indicate that tumors do not form independently in these larvae. The reason for this is not known, but two kinds of explanation are possible. The first kind of explanation is that in tu bw;$+^{su-tu}$, initial tumor formation is inhibitory to further tumorigenesis and that in Oregon K, initial tumor formation stimulates further tumorigenesis. The second possible explanation is that tumors form independently in both stocks, but that they fuse (tu bw;$+^{su-tu}$) or fragment (Oregon K) subsequently, perhaps during metamorphosis. We have been unable to detect any appreciable loss of tumor number during metamorphosis in the tu bw strains, indicating that in those cases there may be real inhibitory effects between tumors during their formation.

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Ayles, B.* University of British Columbia, Vancouver, B.C. Male fertility of wild type stocks of Drosophila melanogaster at different temperatures.

Three years ago, it was decided to screen for mutations on the Y chromosome which produced male sterility at 29°C but fertility at 22°C. However, we found that most wild type strains of D. melanogaster are sterile at 29°C. We therefore screened eight different stocks for fertility at 22°, 28° and 29°C. Twenty males (48-60 hours after eclosion) from each stock were individually mated at each temperature to 3 virgin y/y females. The parents were then discarded after 48 hours and all of the progeny scored.

The percentage of male fertility and mean number of progeny per male is shown in Table 1. The females are fertile at all temperatures as shown by the Amherst^{tr} cross. All but the Amherst^{tr} and Urbana S males were sterile at 29°C. The Urbana S stock produced progeny which died as early pupae at 29°C. At 28°C, all but Samarkand and Swedish C males were highly fertile.

The Amherst^{tr} is a temperature resistant strain selected from the five surviving progeny of a similar test involving an Amberst stock obtained from P.T. Ives of Amherst College in 1967. The Am^{tr} strain has now been maintained in our laboratory by mass mating for over 50 generations at 29°C and it appears to be equally fertile at all three temperatures.