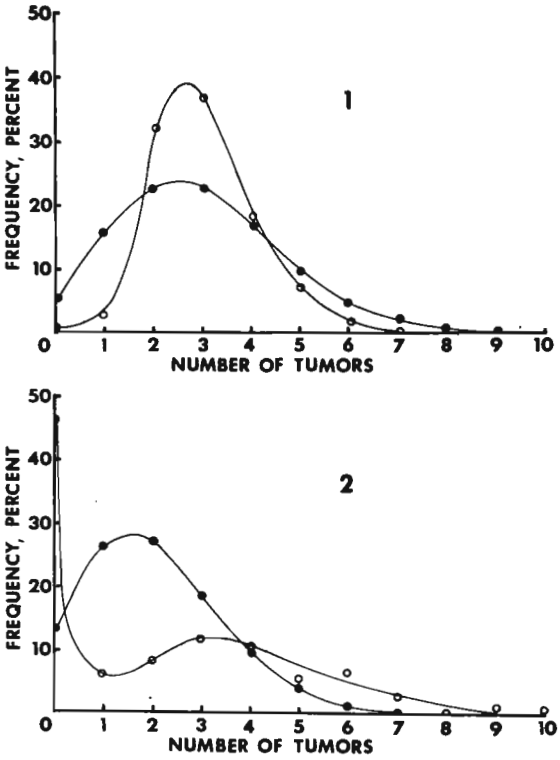
Bryant, P.J. University of California, Irvine, California.* Statistical distribution of melanotic tumors.

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.

Table 1. Percent male fertility and mean number of progeny per male at each temperature

Stock	% fertile			mean no. progeny per male		
	22°C	28°C	29°C	22°C	28°C	29°C
Urbana S.	100	75	**	37.5	19.0	*
Samarkand	100	20	0	55.2	.8	0
Swedish C	100	20	0	52.3	1.8	0
Lausanne	95	75	0	52.2	18.2	0
Samarkand 204	100	60	35	66.1	21.4	2.6
Oregon 369	95	90	5	66.4	36.6	.3
Canton S.	100	95	40	67.9	70.0	.4
Amherst^{tr}	75	75	60	30.0	47.9	31.9

**flies died in the early pupal stages at 29°C

The restrictive temperature of 29°C is obviously close to the borderline of normal biological function of Drosophila melanogaster. Since all eight mutant X chromosomes which we recovered at 29°C still conferred male sterility at 28°C, we feel that this is a far better restrictive temperature to use.

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Miller, D.D. University of Nebraska, Lincoln, Nebraska. On the identity of the "sex ratio" X chromosome of "eastern" D. athabasca.

As reported by Miller and Voelker (1969, Journ. Hered. 60: 231-238, 307-311), the all-female progeny of two wild females of "eastern" D. athabasca collected in Minnesota in 1966 were heterozygous for XL sequence MIII MIX-MX and XS sequence MIII-MIV, suggesting that the X chromosome characterized by these sequences was the carrier of a "sex ratio" factor. Additional evidence for this relationship has been gotten recently from a few single-male matings with strains of "eastern" athabasca from Englewood, New Jersey, and South Williamstown, Massachusetts (from collections of Dr. Max Levitan, via Dr. Robert Voelker). The following table gives the results of four such matings from each of these localities. The symbols represent X chromosome inversions described by Miller and Voelker (1969), except for XL MXIII, a subbasal inversion recently found in several "eastern" strains and to be illustrated and/or described another time.

Mating	Female larvae X chromosomes		Adult Sex Ratio		
	XL	XS	♀♀	♂♂	Total
New Jersey					
-1	MIV MVI MXIII	MVI	55	55	110
-2	MIV MVI MXIII	MVI	26	31	57
-3	MIII MIX-MX (homozygous)	MIII-MIV	61	5	66
-4	MIII MIX-MX (heterozygous)	MIII-MIV	90	0	90
Massachusetts					
-1	MI-MII MVI MVII-MVIII	MI-MII	79	85	164
-2	MI-MII MVI MVII-MVIII	MI-MII	20	22	42
-3	MIII MIX-MX (heterozygous)	MIII-MIV	55	0	55
-4	MIII MIX-MX (homo- and heterozygous)	MIII-MIV	64	3	67