

Trippa, G., A. Lovere, A. Micheli and I. Miola. Istituto di Genetica, Università di Roma, Italia. Frequencies of SD chromosomes in natural populations of *Drosophila melanogaster*.

Samples of wild flies of *Drosophila melanogaster* from eight natural populations were collected in Puglia (Castellaneta, Otranto and Corato), Calabria (Sambiase) and Sicily (Ranna, Pedalino, Vittoria and Archi) during September-October 1971. Males, with $+I/+II$ constitution for the second chromosomes, were individually mated with

virgin bw;st females from a line selected for its high degree of sensitivity to SDR-1 chromosome (Nicoletti et al., Atti A.G.I. 14:29, 1969).

From the F_1 progeny of each cross, three males were collected and mated individually to bw;st females; from the F_2 progeny of only one cross, three males of the same constitution $+I/bw;st/st$ or $+II/bw;st/st$ were collected and mated to bw;st females. The k values (number of $+I/bw;st/st$ individuals/total progeny) were calculated in the F_3 generation.

The k values varied between 0.5 and 1.0, depending on whether the tested second chromosome behaved as a normal or as an SD chromosome. The results are presented in Table 1.

Table 1. The distribution and the properties of the SD-carrying chromosomes found in eight natural populations.

Populations	Number of tested chromosomes	\bar{k}^*	Absolute frequency of SD chromosomes with k values between			TOTAL	Percent frequency of the SD chromosomes
			.65-.75	.75-.85	.85-1.00		
Castellaneta	235	.52 \pm .003	1	1		2	0.85
Otranto	221	.53 \pm .003	3	1	5	9	4.07
Corato	162	.52 \pm .004	6	3		9	5.56
Vittoria	10	.55 \pm .017			1	1	10.00
Ranna	279	.52 \pm .003	4	5	2	11	3.94
Archi	268	.51 \pm .003	12	1		13	4.85
Pedalino	121	.52 \pm .005	1	1		2	1.65
Sambiase	284	.52 \pm .003	8	9	5	22	7.75

* the k values referring to the following progenies: 1) with less than 40 individuals; 2) with a k lower than 0.35; 3) with a k higher than 0.65 have not been computed while estimating the normal mean k.

As it can be seen, SD-carrying chromosomes have been found in all the eight populations examined. Though a SD chromosome would be expected to attain high frequencies in few generations, it is a common observation that this is not the case. There can be many ways by which natural selection might balance the effects of the behaviour of SD, such as a reduction of the reproductive fitness of SD heterozygous males (Nicoletti et al., Accad. Naz. Lincei XLII: 383, 1967) or the association of SD to lethal genes, or the spontaneous appearance of insensitive SD⁺ alleles.

SD chromosomes with inversions (Sandler et al., Genetics 44:233, 1959; Hiraizumi, and Nakazima, DIS 40:72, 1965) as well as without inversions (Nicoletti and Trippa, Atti AGI 12: 361, 1967) have been identified by means of salivary gland chromosomes examination. The present material has not yet been examined from this point of view.

Counce, S.J. Duke University, Durham, North Carolina. Variation in germ cell number and distribution in primitive dipterans.

In dipterans, germ cells are derived from cells which bud off at the posterior pole during late cleavage or early blastoderm stages. The number of primary pole cells may be constant, e.g. one in *Miastor*, two in *Sciara*, or vary, as in *Drosophila*, where three or more primary cells form

(see Sonnenblick in BIOLOGY OF DROSOPHILA). These pole cells continue to divide, and in the first group, the number of these divisions is usually constant also, producing a predictable number of definitive pole cells. Later, during gonad differentiation, the germ cells may also divide a set number of times. For example, in *Miastor*, there are three divisions of the pole cells (8 definitive pole cells) which, in the gonads, divide three more times, producing 64 oogonia.

In *Miastor*, however, although only a single pole cell is initially produced, the number of definitive pole cells does vary. In around 20% of the more than 5000 embryos I have studied, a departure from the expected 8 pole cells has been observed as follows (see table):

no. P.C.:	>8	9	10	11	12	13	14	15	16	Five embryos with 24-32 pole cells were observed, but are not included in the above data. Obviously, one or more pole cells quite frequently undergo a supernumerary division; rarely, fewer than the normal number of
n	29	329	189	80	127	27	54	29	57	
%	3.1	35.7	20.5	8.7	13.8	2.9	5.9	3.1	6.2	

divisions may occur. It is not known whether supernumerary divisions also occur during oögonial divisions in the gonad.

During gastrulation, the pole cells are passively transported to the interior as the germ band extends along the dorsal surface of the egg. During this time, they become segregated into two groups of primary germ cells (in *Miastor* normally 4 and 4) surrounded by lateral mesoderm which will give rise during gonad differentiation not only to supporting elements and follicle cells but to nurse cells as well. In about 20% of all older embryos (103/529) asymmetrical distribution of germ cells to the gonads was found. There was no difference between embryos with normal germ cell numbers (8) and those with aberrant numbers in the frequency with which abnormal gonadal distribution occurred. (Of course, in embryos with odd numbers of germ cells, in "normal" distribution, one of the two gonads would be expected to have an additional cell; this was taken into consideration.)

In the related genus, *Heteropeza*, Reitberger (Chromosoma 1) also noted a variation in the number of definitive pole cells formed. In some 600 embryos of this species I looked at, some 20% had more than the expected number of definitive pole cells (4). Asymmetrical distribution to the gonads was observed in about 20% of the older embryos, and as in *Miastor*, was not related to discrepancies in the number of definitive pole cells.

There is some disagreement as to whether all presumptive gonidia give rise to embryos, or whether some of them become nurse cells (cf. Camenzind, Chromosoma 18; Counce, Nature 218; and Panelius, Chromosoma 23). The extent to which the number of pole cells, and hence the number of definitive germ cells, may vary in these species must be kept in mind in arguments based upon the number of offspring a single mother larva may be expected to produce.

Colaianne*, J.J. and A.E. Bell. Purdue University, Lafayette, Indiana. On the action of transformer (*tra*) in males of *D. melanogaster*.

The third chromosome mutant transformer (*tra*) was initially described as transforming homozygous female zygotes, *tra/tra*, into male-like phenotypes (Sturtevant, 1945). It has generally been thought that the gene has no effect on *tra/tra* male zygotes. Brown and King (1961) have

hypothesized that *tra* is an amorphic mutant whose wild-type allele either suppresses the masculinizing action of other autosomal genes or enhances the feminizing activity of sex-linked loci. However, a recent study (Colaianne and Bell 1972) on the influence of sex of progeny on the lethal expression of the sonless gene (*snl*) yielded results which indicate a masculinizing effect for transformer in *tra/tra* males.

In this study we found that the degree of expression of sonless is directly influenced by the sex of the progeny. Given the prerequisites that both mothers and progeny are homozygous for *snl*, the more male-like an offspring on the male-female continuum the more susceptible it is to *snl* lethality. Specifically relating to the action of transformer were twenty single-pair matings (*snl* +/*snl* + *Ubx/tra* x + *B/Y*, *Ubx/tra*) which produced 1834 daughters and 51 sons. The sons consisted of 49 *Ubx/tra* and 2 *tra/tra*, a highly significant deviation from the expected 2:1 ratio, while the daughters were present in the ratio of 1231 *Ubx/tra*: 603 *tra/tra*.

Thus *tra/tra* males do differ from normal males at least in viability. Our results suggest that the *tra/tra* sons are, in some sense, "super males" in that they are more susceptible than normal males to the sex influenced, lethal action of *snl*.

References: Brown, E.H. and R.C. King 1961, Genetics 46:143-156; Colaianne, J.J. and A.E. Bell 1972, Genetics 72 (in press); Sturtevant, A.H. 1945, Genetics 30:297-299.

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