

Engels, W.R. and C.R. Preston. University of Wisconsin, Madison, Wisconsin: Characteristics of a "neutral" strain in the PM System of hybrid dysgenesis.

The great majority of *D. melanogaster* strains can be classified by sterility tests as either paternally-contributing (P) or maternally-contributing (M) in the PM system of hybrid dysgenesis (Kidwell et al. 1977). A few strains, however, appear to be neutral ("Q strains") by

their lack of sterile offspring when crossed to either P males or M females (Kidwell 1979). One such strain, designated v_6 , is an inbred line derived from a Madison wild population in 1975. The neutrality of v_6 was demonstrated as follows: Under conditions restrictive for sterility (Engels and Preston 1979) a large number of crosses between single v_6 males and females from the M strain, $bw;st$, were performed. Sterility tests of the daughters by the tissue culture plate method of Engels and Preston (1979) yielded less than 1% sterility (4/427), which is indistinguishable from background effects. This lack of sterility cannot be attributed to a suppressor of sterility in the v_6 genome, as shown by tests of each of the v_6 major chromosomes in the absence of the others. The procedure was similar to that reported previously (Engels 1979a) for π_2 , a typical P strain. Sterility frequencies from the lone action of the X, second, and third chromosomes of v_6 were respectively 1/103, 1/91, and 0/93. Therefore, v_6 lacks the potential for sterility when crossed to M females. To determine the cytotype of v_6 (see Engels 1979a), 133 v_6 females were crossed individually to π_2 males under restrictive conditions. None of these crosses produced an appreciable proportion of sterile daughters, with the overall sterility frequency being less than 1% (14/2071). We may therefore say that v_6 has the P cytotype, which confers immunity to the sterilizing action of the π_2 chromosomes (Engels 1979a).

There is a strong correlation among wild genomes between their ability to cause sterility and their ability to bring about male recombination when in the M cytotype (Engels and Preston 1980). One might therefore expect v_6 to produce little or no male recombination in its dysgenic hybrids. This was not the case, however, as shown by experiments measuring male

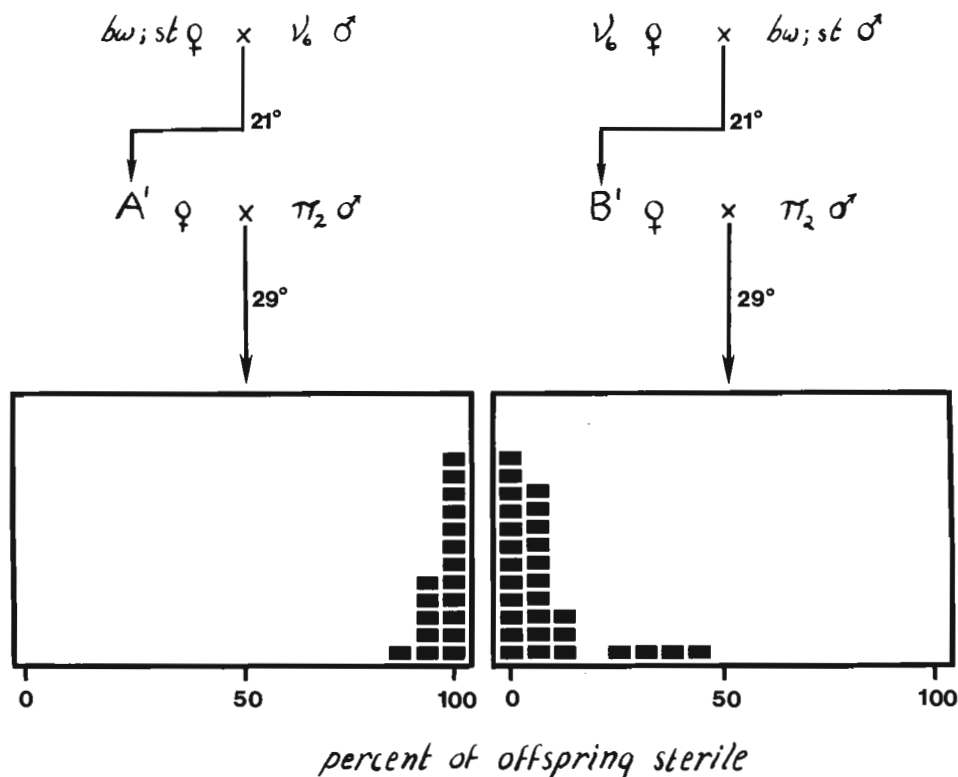


Fig. 1. Determination of cytotype for two reciprocal types of hybrids between v_6 and $bw;st$. Each block represents sterility tests of 16 daughters of a single A^1 or B^1 female.

recombination between *cn* and *bw*, and also segregation distortion at chromosome 2 in the two reciprocal kinds of hybrids involving v_6 and M strain (see Engels 1979b for stocks and procedures). Among approximately 200 progeny from each of 40 males (20 of each reciprocal type) the recombination frequency was $0.3\% \pm 0.1$ for the dysgenic class, and 0 for the reciprocal class. The transmission frequencies of the v_6 second chromosome were 50% and 54% (both $\pm 1\%$) respectively. (See Engels 1979c for the method of calculating standard deviations when clustering is present.) Treating the progeny of each male as an independent observation, the difference between the two reciprocal crosses was significant at $p < 0.01$ by the Rank Sum test for both comparisons. Thus v_6 behaves like a typical P strain regarding male recombination and segregation distortion.

Finally, the following set of experiments shows that the cytotype of v_6 as well as its chromosomal determinants are essentially identical to those of π_2 . Genetically-identical females from the two reciprocal crosses between v_6 and *bw/st* were grown in permissive conditions, then mated to π_2 males under restrictive conditions to determine their cytotype. The results are in Fig. 1. It is clear that the cytotype of v_6 is transmitted matroclinously through two generations, and is therefore not determined by strictly Mendelian factors or simple maternal effects. A self-replicating property of the cytoplasm (or nucleoplasm) which was previously demonstrated (Engels 1979a) is again indicated. To continue substituting the v_6 genome into *bw;st* cytoplasm, the A^1 females were backcrossed to v_6 males for several generations to produce A^2 , A^3 , etc., females. Each generation about 100 of these females were crossed to π_2 to determine their cytotype as above. We see (Fig. 2) that with each successive generation, more of the females have switched their cytotype from M to P. By comparing these results to Fig. 2 in Engels (1979a) it is clear that the ability of the v_6 genome to switch the cytotype is at least equal to that of π_2 .

We may conclude that v_6 is neutral for gonadal dysgenic sterility, but it behaves like a typical P strain regarding its influence on cytotype and on the production of some other dysgenic traits. These observations imply that the determinants of cytotype and other traits,

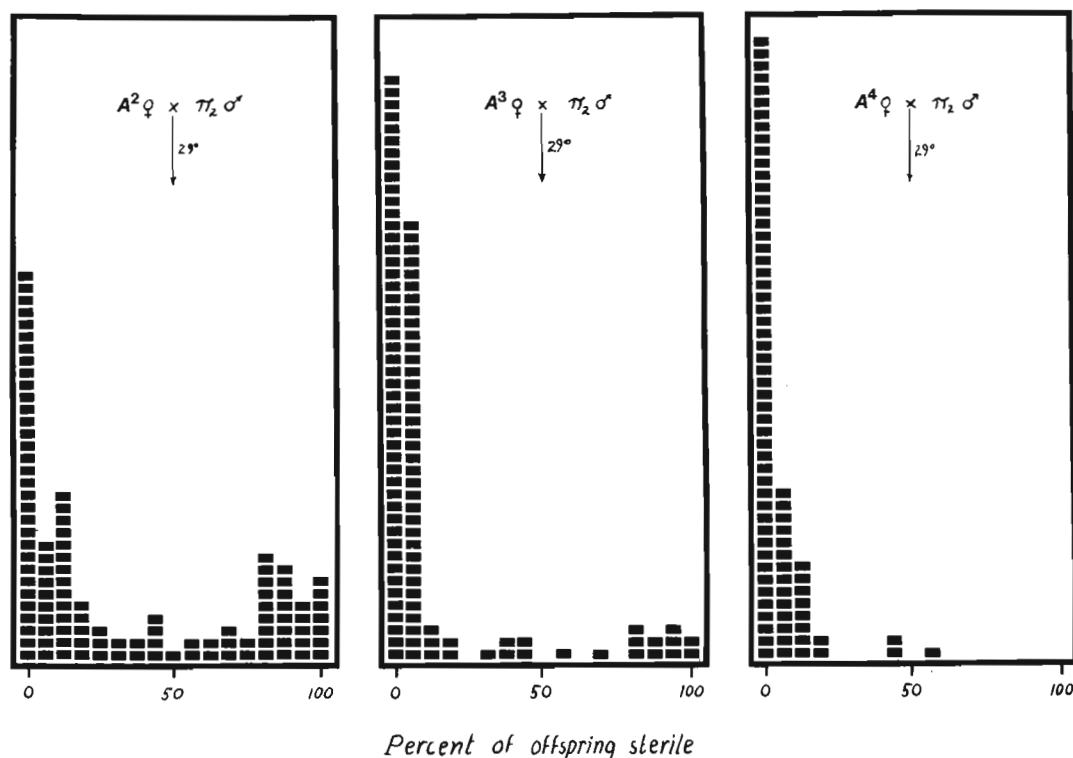


Fig. 2. Determination of cytotype after each successive backcross generation of genomic substitution. Each block represents sterility tests of 16 daughters of a single A^1 female.

are separable from those of sterility. One possibility is that v_6 carries a P factor (presumed to be a movable, multicopy genetic element [Engels 1980]) which lacks its usual sterility function but retains its other capabilities.

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References: Engels, W.R. 1979a, *Genetical Research* 33:219-236; ____ 1979b, *Genetical Research* 33:137-146; ____ 1979c, *Environmental Mutagenesis* 1:37-43; ____ 1980, C.S.H.S.Q.B. Vol. 45 (eds. A. Bukhari and J. Hicks), in preparation; Engels, W.R. and C.R. Preston 1979, *Genetics* 92:161-174; ____ and ____ 1980, *Genetics* 95 (in press); Kidwell, M.G., J.F. Kidwell and J.A. Sved 1977, *Genetics* 86:813-833; Kidwell, M.G. 1979, *Genetical Research* 33:105-117.

Falk, R. The Hebrew University, Jerusalem, Israel, and University of Oregon, Eugene, Oregon. Somatic mosaics produced by a loss of a centric fragment.

Developmental analysis in *Drosophila* depends heavily on the ability to induce genetically labeled clones at predetermined times during embryonic and larval development. Such clones have been produced mainly through chromosome nondisjunction and loss and through induced

mitotic recombination events. Here we report on still another method to obtain marked clones, namely by nondisjunction of free chromosome fragments with appropriate markers.

Novitski and Puro (1978) derived a small free ring chromosome from the second autosome, bearing the dominant bristle morphology mutant $B1: Dp(2;f)B1$. Flies with two wild type alleles on their chromosomes and with the mutant allele on the ring are $B1$ in phenotype. The spontaneous loss of the ring during development can be observed by the appearance of non- $B1$ bristles on the thorax and the head. We followed seven macrochetæ on the dorsal side of the head, the two major humeral bristles and 13 macrochetæ on the thorax (including two sternopleurals). 213 out of 1070 scored flies had non- $B1$ spots (19.9%); of these 193 had one spot, 18 had two spots and 2 had three spots each. This is in good agreement with the expectation of random distribution of independently originating spots ($X^2_{(3)}=0.86$). The size of the spots ranged from those of single bristles to one comprising half the thorax and head:

No. of bristles	No. of flies	No. of bristles	No. of flies
1	131	10	2
2	52	11	1
3	16	:	
4	9	13	2
5	6	14	1
6	7	15	3 (half thorax)
7	2 (half head)	:	
:		22	1 (half body)

Only 6 single non- $B1$ bristles were found in 715 flies of two different $B1/+$ laboratory stocks. The frequency of spots can be increased by taking flies with spots as parents for the next generation. Of 114 progeny of such mosaic flies 33 had one spot and 4 had two spots each (32.5%). Thus the non- $B1$ spots in the free-ring stock appear to be due to genuine fragment losses rather than to variations in the expression or the penetrance

of the mutant character.

The distribution of spots of all sizes indicates that fragment loss may occur at any time during development, being merely a function of the number of cells at each stage that undergo cell division, starting with few large clones induced at early stages of embryogenesis to many small ones shortly before puparium formation. However, the distribution of the spots may depend also on the presence of borderlines for developing clones and on the denseness of the bristle pattern at each site. Thus of the 10 large clones comprising most of the mesothorax only one included also the head disc, while 8 included also the humeral disc; two of these included both left and right humeral discs. These clones were obviously established even before the imaginal disc borders were determined, some even before the midline was laid down (note that the humeral focus is nearer to the midline on the blastoderm fate map than are the mesothoracic foci). In two flies an anterior dorso-central bristle was included in half-thorax clones of the "other side". It could be that these clones too were established before midline determination. Only three of the small clones crossed disc border lines (one 3-bristle spot crossed from head, through humerals to mesothorax). It is possible that these were also large early clones that extended mainly to the inside of the animal.