Among collected flies, as shown in the Table, we found only six different species which all belonged to the so-called cosmopolitan, domestic or widespread species (Patterson & Stone 1952; Dobzhansky 1965, David & Tsacas 1980).


In 1968 a factor of instability was discovered in Drosophila simulans. The H factor, as it was called, sharply increases the rate of somatic recombination and spontaneous mutation in the gametes of those individuals that carry it (Khovanova 1977). The H factor exercises semi-dominant effects, it is active when received from males or females, is localized at the end of the X chromosome and can get accumulated in it, so that individuals with more than one "dosage" of the H factor were found and became the starting points of the various stocks. To test the ability of H to migrate to the autosomes and be transferred to other loci within the autosomes of the carrier stock, a reciprocal autosome substitution was effected in two stocks: (1) sn v wy (2H+) & C(I), yw , stock No. 269(H+), the males contain two H dosages; (2) +(H+)/Y & C(I), yw , stock No. 2, contains no H factor (H−).

Females with compound-X chromosomes were obtained from the yw(H−) stock and carried no H factor in the X chromosomes. The order of chromosomes in the compound was not established. The autosome substitution was carried out as follows:

a) \( \sigma \sigma \) sn v wy + from stock No. 2,\( H^- \)
\[ F_1 \sigma \sigma \text{ sn v wy } \times \frac{??}{??} C(I),yw \]  (from stock No. 2, H−)
\[ F_2 \sigma \sigma \text{ sn v wy } \times \frac{??}{??} C(I),yw \]  (from stock No. 2)
\[ F_{14} \sigma \sigma \text{ sn v wy } (H?) \]

b) \( \sigma \sigma +(H^-)/Y \) from stock No. 269,\( H^- \)
\[ F_1 \sigma \sigma +(H^-)/Y \times \frac{??}{??} C(I),yw \]  (from stock No. 269, H+)
\[ F_2 \sigma \sigma +(H^-)/Y \times \frac{??}{??} C(I),yw \]  (from stock No. 269, H+)
\[ F_{14} \sigma \sigma +/Y (H?) \]
To obtain each generation, 10 to 15 males from the previous generation were crossed to females of the appropriate stock (No. 2 for (a) and No. 269 for (b)). The substitution required about one year. F₁ males from series (a) and (b) were individually crossed to females of the appropriate stock (No. 2 for (a) and No. 269 for (b)). The substitution required about one year. F₁, males from series (a) and (b) were individually crossed to yw(H) females. Crosses of No. 2 and No. 269 males to yw(H) females served as control. The rate of somatic mosaicism was determined in F₁ females resulting from these crosses. Individual analysis of n for the presence of H showed that n F₁ sn v wy (H'), (a) with autosomes replaced by the autosomes of stock No. 2 (H') had not lost H (contained the same two H doages as n= No. 296, H') and n F₁ (b) which had received the autosomes of stock No. 296 (H') had not received the H factor with them. Now the autosomal substitution in the (b) series led to an unexpected result. All the F₁ (b) males, which were phenotypically indistinguishable from the parental No. 2 males (red-eyed), produced brown-eyed females in the F₁ of the cross to yw females. Since the F₁ females were heterozygous with respect to the white gene, we supposed that all the F₁ (b) males carried a coloured white allele phenotypically indistinguishable from the wild-type w allele.

Further analysis, including crosses to w1187, w1393 and wlemon alleles independently obtained at different times, confirmed the above supposition. We termed the new white allele white-mysterious (wmy). The w1187/wmy, w1393/wmy, wle/wmy heterozygotes have dark brown eyes. The colouring of wmy/wmy females and wmy/Y males is phenotypically indistinguishable from the wild type.

The wmy mutation seems to have emerged in the process of autosome substitution. Possibly the wmy males develop at a somewhat faster rate than the wild-type males, which would have given them a higher probability of getting from F₁ to F₁+i. The case described is an instance of genetic drift in small laboratory populations.


In order to test for each system of hybrid dysgenesis, two crosses, denoted A and A*, were routinely made en masse with each strain as indicated in Table 1. Sterility frequencies of the gonadal (GD) and sterilite female (SF) types characteristic of the P-M and I-R systems, respectively, were estimated using the methods described by Kidwell (1979). The results of the sterility tests were interpreted and the strains were characterized according to the criteria given in Table 2. The distinctions made between P and Q and between R and N strains are somewhat arbitrary and may reflect quantitative rather than qualitative variation.

Table 1. Details of reference strains used in mass matings in order to test strains of unknown dysgenic potential with respect to the two systems of hybrid dysgenesis.

<table>
<thead>
<tr>
<th>Hybrid dysgenesis system</th>
<th>Type of cross</th>
<th>Developmental temp.</th>
<th>Sterility assay</th>
</tr>
</thead>
<tbody>
<tr>
<td>P-M</td>
<td>Canton-S (M)</td>
<td>Harwich (P)</td>
<td>29° GD frequency</td>
</tr>
<tr>
<td>I-R</td>
<td>seF (R) or Cocksaponsett</td>
<td>Luminy (I)</td>
<td>20° SF frequency</td>
</tr>
</tbody>
</table>

A large survey of D. melanogaster strains has been conducted in order to determine their potential for the P-M and I-R systems of hybrid dysgenesis (for review see Bregliano & Kidwell 1983). The summarized results and analysis of this survey will be published elsewhere (Kidwell 1983). Here a list of tested strains is provided together with the results of standard tests for hybrid dysgenesis.

In order to test for each system of hybrid dysgenesis, two crosses, denoted A and A*, were routinely made en masse with each strain as indicated in Table 1. Sterility frequencies of the gonadal (GD) and sterilite female (SF) types characteristic of the P-M and I-R systems, respectively, were estimated using the methods described by Kidwell (1979). The results of the sterility tests were interpreted and the strains were characterized according to the criteria given in Table 2. The distinctions made between P and Q and between R and N strains are somewhat arbitrary and may reflect quantitative rather than qualitative variation.

A list of tested strains together with sterility frequencies observed and strain designations with respect to hybrid dysgenesis are presented in Table 3. With respect to GD sterility, the cross A results provide an estimate of P factor activity and the cross A* results indicate the cytotype. With respect to SF sterility, the cross A results provide an estimate of I factor activity and the cross A* results estimate the degree of reactivity. Individual