DROSOPHILA INFORMATION SERVICE

Number 63

Including the Special Report:

THE MOLECULAR GENOME OF Drosophila melanogaster:
Catalogs of cloned DNA, breakpoints
and transformed inserts by chromosome location.

by

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SPECIAL REPORT

The Molecular Genome of Drosophila melanogaster:
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by J. Merriam, S. Smalley, A. Merriam and B. Dawson
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ANNOUNCEMENTS

NOTICE OF PRICE CHANGE: Effective with Volume 65 (June 1987), the cost of a subscription will rise to $7.00 per copy if paid in advance ($8.00 if invoiced). This is the first price raise that DIS has had in quite a few years and is due to rising costs of printing and postage, as usual. Back issues (vol. 64 and prior) will remain at $5/copy.

Notice of Publication: The next installment of The Genome of *Drosophila melanogaster* by Dan Lindsley and Georgianna Zimm will appear as DIS 64 in the fall of 1986. It is anticipated that it will include mutations beginning with the letter L (including the lethals), Duplications, Inversions, Rings, Translocations, and Transpositions. In addition, there will be an ordered list of rearrangement breakpoints and polytene gene localizations prepared by Andrew Cockburn from a computer search of the current data base for the revised edition, plus the as yet unrevised material from the Red Book.

27th ANNUAL DROSOPHILA RESEARCH CONFERENCE

Held April 10-13, 1986, at the Asilomar Conference Center in Pacific Grove, California.

Plenary Session - Chair: Larry Sandler
   Speakers: M. Gatti, R.S. Hawley, A.J. Hilliker, A. Chovnick, L. Sandler
   Friday, April 11

Concurrent Sessions:
   Population genetics and evolution (Chair - Margaret Kidwell)
   Pattern formation (Chair - Judith Lengyel)
   Gene expression (Chair - Steven Henikoff)
   Friday afternoon, April 11

Concurrent Sessions:
   Oncogene homologues (Chair - John McDonald)
   Heat shock (Chair - Nancy Petersen)
   Friday evening, April 11

Concurrent Sessions:
   Enzymes and physiology (Chair - Janis O'Donnell)
   Gene interactions (Chair - Paul Bingham)
   Hormone-inducible genes (Chair - Ross Hodgetts)
   Saturday morning, April 12

Concurrent Sessions:
   Neurobiology and behavior (Chair - Margrit Schubiger)
   Homeotic genes (Chair - Richard Carber)
   Chromosomes (Chair - Barbara Wakimoto)
   Saturday afternoon, April 12

Concurrent Sessions:
   Reproductive behavior (Chair - Laurie Tompkins)
   Adh in evolution (Chair - W. Dickinson)
   Saturday evening, April 12

Plenary Session - Chair: Gerold Schubiger
   Speakers: Bruce Alberts, Corey Goodman, Carl Parker, Terrence Lyttle
   Sunday, April 13

28th Annual Drosophila Research Conference will be held May 20-24, 1987, at the Bismarck Hotel in Chicago, Illinois. Local arrangements are being handled by Janice Spoofford (University of Chicago); please write to get on the mailing list to: Office of Continuing Medical Education, SBRI J 131 (Box 139), University of Chicago, Chicago, Illinois 60637, or call 312-962-1056. The conference will start Wednesday evening (May 20) and run thru Sunday noon, May 24 at the Bismarck Hotel, which is close to the rapid transit from O'Hare airport. Room rates: $45 single and $55 double. All meetings will be at the hotel. William Engel is coordinating the program arrangements (Lab of Genetics, 509 Genetics Bldg, 445 Henry Mall, University of Wisconsin, Madison, WI 53706). The regular mailing will be sometime in the Fall 1986.
New Publication: *Diptera: Drosophilidae*, by Gerhard Bächli and Hans Burla, Vol. 7 (1985) of *Insecta Helvetica*, edited by the Swiss Entomological Society, 116 pages, 3 color plates; 20 Swiss Francs (about $9). This volume, written in German, is thought to include all Central European species. It contains information on collecting, biology and morphology, as well as illustrated keys to genera and species.

New laboratory: Einar Arnason has organised a new *Drosophila* laboratory at the Institute of Biology, University of Iceland, Grensávegur 12, 108 Reykjavik, Iceland (tel: 354-1-685433). We request being put on mailing lists where such exist. Fields of interest are evolutionary biology: population genetics.

Stock not available: We have been receiving requests for the *Drosophila melanogaster* stock Barlike-eyes (3-94) Ble, which was erroneously listed in Stock List #3 (DIS 57). We regret to say that we do not, and never did, have the stock in question.

Recommends needed service: Prof. Robert Arking, Wayne State University, recommends Watkins Tweezer Refinishing (P.O. Box 1402, 1209 Hilltop Drive, Mount Dora, Florida 32757 Tel 904-386-8037) for repairing laboratory forceps at ca. $2.50/tweezers. He has been very satisfied with their work. In the past Prof. Arking had to rely on replacing damaged forceps and was pleased to find this service through a watchmaker friend.

ANNOUNCING A 1987 UCLA SYMPOSIUM:

**Molecular Biology of Invertebrate Development**


The development of a single cell into a multicellular organism comprised of several differentiated cell types and organ systems requires the integration of gene expression, regulation of membrane transport, and the recognition of both intracellular and extracellular signals. These and other related phenomena are being investigated in diverse plant and animal species. Invertebrates continue to offer excellent experimental systems to study the molecular mechanisms involved in the regulation of development, due in no small measure to the facility with which they can be cultured, their relatively small genome, and the number and array of the critical signals (e.g., hormones, pheromones, neuropeptides). By focusing on invertebrate development, this meeting will permit a relatively detailed examination of the principal events of development in the most extensively investigated organisms, while at the same time permitting comparative analysis.

Plenary Session topics will include: Vitellogenesis/Oogenesis, Molecular Sequelae of Fertilization, Early Molecular Events in Cell Lineage, Segmentation and Early Pattern Events, Cell Lineages and Commitment, Molecular Neurobiology, Genetic and Cellular Mechanisms in Imaginal Disc Development, Juvenile Hormone/Gene Expression/Metamorphosis, Cis and Trans Regulatory Elements, Regulation at the Post-transcriptional Level.


For further information, please call or write to: UCLA Symposia, Molecular Biology Institute, University of California, Los Angeles, CA 90024. Tel 213-206-6292. Telex: UCLA Symposia 9103427597. Applications will be accepted at any time for meetings which are not over-subscribed.
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Hydroxyurea, a known DNA synthesis inhibitor, is used in the present investigation for evaluating its in vivo synchronization effect on the replicating polytene nuclei.

The inhibitory action of Hydroxyurea on DNA synthesis is probably by blocking the activation of reductase activity by specific deoxyribonucleoside triphosphates (Swindlehurst et al. 1971).

Early third instar giant mutant female larvae of Drosophila melanogaster (gt w¹/Df(1)162 g¹8) were fed on sucrose (1M) solution containing 1mg/ml Hydroxyurea for 48 hr using an apparatus designed for chemical mutagenesis (Achary & Dutta 1984). ³H-TdR autoradiograms were prepared from larval salivary glands and the labelling patterns were scored following the classification of Chatterjee & Mukherjee (1975).

Table and histogram show predominance of mid-part of S-phase nuclei (3C and 3D patterns). While the frequencies of early part (DD, 1C and 2C patterns) was 13.75 percent and late part (2D and 1D patterns) was 11.89 percent, the mid-part (3C and 3D) showed a percentage as high as 73.29.

The synchronizing effect of Hydroxyurea at the mid-part of S-phase simulates those of FdUrd (Achary et al. 1981) and Aphidicolin and Ricin (Duttagupta & Banerjee 1984), where more than sixty-five percent of mid-part nuclei were recorded. Cold thymidine chase experiments, for releasing the block and to obtain further synchronization at other parts of S-phase, are in progress.

Acknowledgement: We gratefully acknowledge Dr. A.K. Duttagupta, Calcutta University, for providing laboratory facilities.


Table 1. Frequencies (in %) of different types of ³H-TdR replicating patterns of salivary gland polytene nuclei of giant Drosophila melanogaster larvae fed with Hydroxyurea for 48 hr. Numbers in () show number of nuclei observed.

<table>
<thead>
<tr>
<th>Replication patterns</th>
<th>CONTROL: tn = in total nuclei</th>
<th>tn = in labelled nuclei</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>45.74 18.09 4.26 8.51 11.70 5.32 6.38 -- --</td>
<td>(68.09 20.21 11.70)</td>
</tr>
<tr>
<td>HYDROXYUREA: tn = in total nuclei</td>
<td>tn = in labelled nuclei</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.00 2.96 4.15 16.60 20.36 5.34 2.37 48.22 51.78</td>
<td>(0) (15) (21) (84) (103) (27) (12) (244) (262)</td>
</tr>
<tr>
<td></td>
<td>0.00 5.73 8.02 32.06 39.31 10.31 4.59 -- --</td>
<td>(13.75 72.37 11.89)</td>
</tr>
</tbody>
</table>

Figure 1. Histogram showing frequencies of early (DD-2C), mid (3C-3O) and late (2D-1D) patterns within the labelled nuclei. The open bars represent control and solid bars represent experimental (Hydroxyurea). LI indicates labelling index.

A larval arrest in development of Drosophila melanogaster was first detected in crowded conditions by Mensua & Moya (1983) in different wild strains. Afterwards it has been also shown in other Drosophila species, other Drosophilids (Botella & Mensua 1985a), as well as in other holometabolous insects (Tribolium castaneum) data to be published elsewhere. Recently it has been shown (Botella & Mensua 1985b) that stopped larvae seem to have decreased levels of molting hormone. This fact has been used as a starting point to investigate the larval stop in mutants known to be affected in the levels of molting hormone, such as lethal giant larvae (Welch 1957).

For this purpose crowded cultures (70 larvae in 0.5 ml of Lewis medium) of the strain (2)gl a px or/SM5 a2 Cy Itv cn sp2) were reared at 25°C. At different days from the seeding day, different sets of crowded cultures were subjected to overfeeding (Moya & Mensua 1983) allowing larvae to migrate spontaneously to larger vials (10 x 25 cm with 10 ml of Lewis medium in an inclined position). In this way the initial population was divided into two subpopulations, inner (non-migrated individuals) and outer (individuals recovered by overfeeding following their spontaneous migration).

Two controls, uncrowded and crowded (never subjected to overfeeding), were also taken. A total of five replications were made of every set of vials.

Table 1 shows survival in inner, outer and total population, and mean developmental time in inner and outer populations throughout overfeedings and controls.

As can be seen from this table, the number of larvae recovered by overfeeding decreases as the cultures develop and becomes zero in the two last overfeedings.

As reflected by the mean developmental time (MDT), larvae recovered in the three first outer populations may be considered stopped.

However, from a certain age on (14 day old) no larvae were recovered by overfeeding, and hence third instar larvae of this age die, possibly as a consequence of the mutation (2)gl.

Table 2 shows larval mortality before and within the third instar. In contrast with other strains in which dead larvae cannot directly be seen, careful inspection of these cultures reveals the presence of dead larvae in third instar.

The main point to be noted is the increase in larval mortality both before and within third instar as the overfeedings are later. The situation of the crowded control becomes similar to the cultures overfed at 16th day.

The effect of competition seems to enhance the larval mortality, and we suspect that some heterozygous larvae for the mutant (2)gl die when the competition becomes stronger. These results are only a preliminary report to be further investigated but which appears to indicate a phenocopy effect of (2)gl due to an interaction between environment (competition) and heterozygous constitution for this mutant.

**References:**

---

**Table 1.** Survival in inner, outer and total population, and mean developmental time (MDT) in inner and outer populations throughout overfeedings and control.

<table>
<thead>
<tr>
<th>Survival (inner)</th>
<th>Survival (outer)</th>
<th>Survival (total)</th>
<th>MDT (inner)</th>
<th>MDT (outer)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Uncrowded control:</td>
<td>50.2±1.6</td>
<td>50.2±1.6</td>
<td>---</td>
<td>12.8±0.1</td>
</tr>
<tr>
<td>Crowded control:</td>
<td>13.5±2.6</td>
<td>13.5±2.6</td>
<td>13.9±0.3</td>
<td>---</td>
</tr>
<tr>
<td>Overfeeding (days):</td>
<td>8</td>
<td>6.0±0.6</td>
<td>42.8±1.6</td>
<td>48.8±1.1</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>14.0±2.3</td>
<td>22.4±5.6</td>
<td>36.4±3.9</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>13.7±3.4</td>
<td>8.5±6.4</td>
<td>22.2±6.0</td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>11.0±2.0</td>
<td>11.0±2.0</td>
<td>13.3±0.2</td>
</tr>
<tr>
<td></td>
<td>16</td>
<td>10.0±1.9</td>
<td>10.0±1.9</td>
<td>12.8±0.4</td>
</tr>
</tbody>
</table>

*Regression values: a(intercept at origin) = 5.41; b(slope) = 1.33; R² = 0.99.*

**Table 2.** Larval mortality before and within 3rd instar.

<table>
<thead>
<tr>
<th>% dead larvae before 3rd instar</th>
<th>% dead larvae* in 3rd instar</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (crowded)</td>
<td>44.4%</td>
</tr>
<tr>
<td>Overfeeding (days):</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>24.9%</td>
</tr>
<tr>
<td>10</td>
<td>37.4%</td>
</tr>
<tr>
<td>12</td>
<td>31.6%</td>
</tr>
<tr>
<td>14</td>
<td>35.7%</td>
</tr>
<tr>
<td>16</td>
<td>44.6%</td>
</tr>
</tbody>
</table>

* this percentage of dead larvae was calculated referring to individuals which attain 3rd instar.

It has been reported (Mensua & Moya 1983) that competition induces larvae of D. melanogaster to arrest their development in third larval instar. This is true both, at different temperatures (Botella & Mensua 1985a) and at different crowding densities (Mensua et al. 1983). Nevertheless, the number of arrested larvae depends on the degree of crowding (Mensua et al. 1983). On the other hand, long-term competition in other Drosophilids (such as Chymomyza costata) induces third instar larvae to enter diapause. This being so, and to further investigate on the effects of continuous crowding in D. melanogaster, two sets of experiments have been carried out in this species, reared at two temperatures (19 and 25°C). For this purpose two different sets of 5 x 0.8 cm vials with 0.5 ml of Lewis medium were seeded with 70 newly hatched larvae from an isogenic Oregon-R strain. A total of 30 vials were started at 19°C and 26 vials at 25°C. At different intervals, every 9 days at 25°C and every 18 days at 19°C, the still remaining larvae were extracted from the original cultures, and transferred by groups of 70 larvae to vials identical to the previous ones, keeping in this way the same crowding. The number of emerged adults was recorded daily until the exhaustion of the cultures.

Table 1 is a life table showing the following parameters: the total number of remaining larvae at the end of each transfer, the number of dead larvae, the total number of pupated larvae, the number of dead pupae and the number of emerged adults, each accompanied by their corresponding percentage referred to the total number of larvae in each period for 19°C and 25°C. Bearing in mind that previous experiments carried out in our laboratory have shown that mean developmental time in the same strain in uncrowded cultures is about 19-23 days at 19°C and about 9-13 days at 25°C, a delay in mean developmental time can be observed in both temperatures as a consequence of long-term competition. In our case development has been prolonged until 32 days at 25°C and 53 days at 19°C. It is worth pointing out that pupal mortality increases as competition lasts longer. This effect may be due to the increased intoxication of larvae subjected to the competition process which are being forced to ingest their metabolic wastages (uric acid among them) (Botella et al. 1985). It is also interesting to note the higher rates of mortality obtained at 19°C owing to the fact that 25°C is a better temperature for Drosophila development than 19°C (Ashburner & Thompson 1978).

On the other hand, in contrast with the result obtained in Drosophilids with larval diapause, such as Chymomyza costata (Botella & Mensua 1985b), long-term competition cannot stop larval development for months in third instar. In fact, as can be seen from the table, all larvae finally pupated or died. In this sense larval arrest in Drosophila may be viewed rather as a kind of quiescent state susceptible to end as soon as environmental conditions become slightly more favourable throughout the successive transfers to fresh medium. This mechanism would be parallel to the adaptive meaning of larval diapause in non-diapausing species such as Drosophila, but in a more flexible way than diapause do.

Table 1. Life Table from the experiments of long-term competition in Drosophila melanogaster at 19°C and 25°C.

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Days</th>
<th>Dead Larvae (%)</th>
<th>Pupated Larvae (%)</th>
<th>Dead Pupae (%)</th>
<th>Emerged Adults (%)</th>
<th>Remaining Larvae (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>19°C</td>
<td>1-18</td>
<td>255(12.1)</td>
<td>372(17.3)</td>
<td>99(26.6)</td>
<td>273(73.4)</td>
<td>1473(70.1)</td>
</tr>
<tr>
<td></td>
<td>19-36</td>
<td>495(33.7)</td>
<td>908(61.8)</td>
<td>312(34.4)</td>
<td>596(65.6)</td>
<td>67( 4.6)</td>
</tr>
<tr>
<td></td>
<td>37-54</td>
<td>43(64.2)</td>
<td>24(35.8)</td>
<td>17(70.8)</td>
<td>7(29.2)</td>
<td>0</td>
</tr>
<tr>
<td>25°C</td>
<td>1-9</td>
<td>489(26.9)</td>
<td>350(19.3)</td>
<td>45(12.8)</td>
<td>306(87.2)</td>
<td>980(53.8)</td>
</tr>
<tr>
<td></td>
<td>10-18</td>
<td>216(23.7)</td>
<td>545(59.9)</td>
<td>69(12.7)</td>
<td>476(87.3)</td>
<td>149(16.4)</td>
</tr>
<tr>
<td></td>
<td>19-27</td>
<td>50(35.7)</td>
<td>82(58.6)</td>
<td>33(40.2)</td>
<td>49(59.8)</td>
<td>8( 5.7)</td>
</tr>
<tr>
<td></td>
<td>28-36</td>
<td>5(72.5)</td>
<td>3(37.5)</td>
<td>3(100)</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

* % of emerged adults and dead pupae were referred to total no. of pupated larvae. a = days of culture. b = % of 910 larvae which were seeded.

Alahiotis, S.N. and G.N. Goulielmos.
University of Patras, Patras, Greece. Fertile
F₁ males and females from crosses between
Drosophila mauritiana females and D.melanogaster or D.simulans males.

D.mauritiana, sterile females or males (in respect with the melanogaster sex used) are yielded in the F₁ generation. In spite of this general consensus, we present now data showing that fertile males and females can be obtained from some crosses between the sibling species pair mentioned above. Thus, in one out of 55 pair matings, where mauritiana was used as female and melanogaster as male, fertile F₁ males and females appeared. The fertility of these individuals was tested by mating them and noticing if F₂ (or F₃) progeny are yielded. These interspecific fertile hybrids have been named mame and are kept with success in our laboratory as a stock for 10 generations so far. In the opposite cross (♀ melanogaster x mauritiana ♂), seven out of 43 pair matings were found to yield hybrid females (named mema) which are sterile when crossed with melanogaster or mauritiana males. It must be noted that as melanogaster we used the Cyl.⁴/Pm stock, while in a previous effort using another melanogaster stock (homoyzogous for the malate dehydrogenase fast form), we failed to get one successful mating (yielded offspring) out of 92 performed.

In another interspecific cross-type where mauritiana was used as female and simulans as male, two out of 82 pair matings yielded fertile F₁ males and females. These interspecific fertile hybrids have been named masi and are kept in our lab as a stock, like mame. In the case where mauritiana is used as male and simulans as female, 63.41% (26/41) successful matings obtained. Each such successful pair mating yielded females and males which are sterile when crossed with each other but fertile when crossed with mauritiana (males) and simulans (males or females). When mauritiana females are crossed with the above described interspecific hybrids (named sima), no progeny are produced. The same is also true in the cross ♀ mauritiana x mame ♂. In crosses ♀ mame x masi ♂ (and the reciprocal), some pair matings are successful, yielding females and males which possibly get genes from three different species (melanogaster, simulans, mauritiana). In Table 1 we show all the above information described and in Table 2 we give a brief summary of the reproductive isolation status among the three sibling species examined as it was found in our lab (with the strains of the species we used).

It must be noted that the combination melanogaster-simulans was tested and our findings verify the previous consensus (see Table 2), that is, sterile F₁ females or males are produced, dependent on the melanogaster sex used. However, in a mass cross (♀ melanogaster x simulans ♂) a part of the sterile females obtained as expected, 3 hybrid males were also

Table 1. Successful and unsuccessful pair-matings between pairs of D.melanogaster (mel), D.simulans (sim) and D.mauritiana (maur), as well as between fertile interspecific hybrids* and their parents.

<table>
<thead>
<tr>
<th>species pair</th>
<th>successful</th>
<th>unsuccessful</th>
<th>total</th>
</tr>
</thead>
<tbody>
<tr>
<td>♀ sim x maur ♂</td>
<td>26</td>
<td>15</td>
<td>41</td>
</tr>
<tr>
<td>♀ maur x mel ♂</td>
<td>1</td>
<td>54</td>
<td>55</td>
</tr>
<tr>
<td>♀ maur x sim ♂</td>
<td>2</td>
<td>80</td>
<td>82</td>
</tr>
<tr>
<td>♀ mel x maur ♂</td>
<td>7</td>
<td>36</td>
<td>43</td>
</tr>
<tr>
<td>♀ masi x sim ♂</td>
<td>15</td>
<td>27</td>
<td>42</td>
</tr>
<tr>
<td>♀ masi x maur ♂</td>
<td>16</td>
<td>1</td>
<td>17</td>
</tr>
<tr>
<td>♀ maur x masi ♂</td>
<td>0</td>
<td>49</td>
<td>49</td>
</tr>
<tr>
<td>♀ sim x masi ♂</td>
<td>1</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>♀ mame x mel ♂</td>
<td>13</td>
<td>0</td>
<td>13</td>
</tr>
<tr>
<td>♀ mame x maur ♂</td>
<td>1</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>♀ maur x mame ♂</td>
<td>0</td>
<td>38</td>
<td>38</td>
</tr>
<tr>
<td>♀ mel x mame ♂</td>
<td>8</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>♀ mema x mel ♂</td>
<td>0</td>
<td>14</td>
<td>14</td>
</tr>
<tr>
<td>♀ mema x maur ♂</td>
<td>0</td>
<td>28</td>
<td>28</td>
</tr>
<tr>
<td>♀ sima x sim ♂</td>
<td>7</td>
<td>3</td>
<td>10</td>
</tr>
<tr>
<td>♀ sima x maur ♂</td>
<td>3</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>♀ maur x sima ♂</td>
<td>0</td>
<td>16</td>
<td>16</td>
</tr>
<tr>
<td>♀ sim x sima ♂</td>
<td>9</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>♀ masi x mame ♂</td>
<td>5</td>
<td>18</td>
<td>23</td>
</tr>
<tr>
<td>♀ mame x masi ♂</td>
<td>7</td>
<td>25</td>
<td>32</td>
</tr>
</tbody>
</table>

*The name of the interspecific hybrids is composed of two syllables. The first is from the ♂ and the second from the ♀ species-parent name.

Table 2. Hybridization possibilities between the 3 sibling species, melanogaster, simulans, mauritiana, as it has been found in our lab.

<table>
<thead>
<tr>
<th>parents</th>
<th>♀</th>
<th>♂</th>
</tr>
</thead>
<tbody>
<tr>
<td>♀ mel x sim ♂</td>
<td>sterile</td>
<td>---</td>
</tr>
<tr>
<td>♀ sim x mel ♂</td>
<td>---</td>
<td>sterile</td>
</tr>
<tr>
<td>♀ mel x maur ♂</td>
<td>sterile</td>
<td>---</td>
</tr>
<tr>
<td>♀ maur x mel ♂</td>
<td>fertile</td>
<td>fertile</td>
</tr>
<tr>
<td>♀ sim x maur ♂</td>
<td>fertile*</td>
<td>fertile*</td>
</tr>
<tr>
<td>♀ maur x sim ♂</td>
<td>fertile</td>
<td>fertile</td>
</tr>
</tbody>
</table>

*but sterile when crossed with each other (see text)
Table 3. Mating propensities in multiple choice experiments involving the interspecific fertile hybrids *masi* and its parental species, *mauritiana* and *simulans*.

<table>
<thead>
<tr>
<th>Cross</th>
<th>No. of chambers*</th>
<th>Sexual Isolation Index ± S.E.</th>
</tr>
</thead>
<tbody>
<tr>
<td>sim x masi</td>
<td>25</td>
<td>22</td>
</tr>
<tr>
<td>maur x masi</td>
<td>5</td>
<td>14</td>
</tr>
</tbody>
</table>

* in each chamber existed 12 virgin females and 12 males from each stock.

The situation described here regarding the reproductive isolation status between three sibling species in the *D. melanogaster* subgroup differs with that which it was so far known. These differences may be based on the genetic composition of our strains used (e.g. Cyl4/Pm for *D. melanogaster*; *D. simulans* was captured recently from a Greek natural population) or on some evolutionary changes of *D. mauritiana* under the laboratory conditions where lately is maintained.

The implication of these hybrids to the study of the speciation mechanisms is obvious and can be proved important in understanding the evolution of interspecific reproductive isolation. The elucidation of the detailed genetic organization of these hybrids (the status of which was also verified by electrophoretic and cytogenetic criteria) will contribute greatly to the approach of the above purpose.


It is well known that *D. melanogaster* can readily become tolerant for a wide range of toxic chemicals when present in its environment. To obtain insight in the process of developing tolerance, two different populations of *D. melanogaster* were subjected to selection on five xenobiotics. Selection was performed by rearing the flies in cages on food supplemented with the xenobiotics. During the experiment the concentrations were increased regularly in 7-8 steps and the initial concentrations and the concentrations after 17 months (the moment the tolerance levels were determined) of the different chemicals are given: (1) phenobarbital (sodium salt): 250 – 1300 ppm; (2) rotenon (a commercial anti-flea powder containing 0.9% rotenon was used): 6 – 24 ppm; (3) malathion (a commercial preparation containing 50% malathion was used): 0.15 – 0.65 ppm; (4) carbaryl (also a commercial preparation containing 50% carbaryl was used): 6 – 28 ppm; (5) DDT: 15 – 65 ppm. The first chemical is used as a drug; the other four are or have been used as insecticides.

For the experiments two different sets of each six population cages were established. The Bogota populations were initiated with 36 independent lines isolated from the Bogota base population as described by Bijlsma (1980). The 50 x 50 populations were initiated with 40 lines from the second reisolation from the original 50 x 50 base population as described by Bijlsma & Van Delden (1977). All cages of each set were provided with standard food (for description of the food see Bijlsma 1980) for the first week to get them well established. Thereafter one cage of each set was kept on this food (control) while the others were supplied with standard food supplemented with one of the five toxic compounds. To standardize the selection pressures somewhat, the initial concentrations were chosen in such a way that the larval viability was approximately 40-60%; as a result the density in the cages was kept well over a thousand individuals. When the concentrations were increased during the experimental period, it was also ensured that the population density stayed above this level.
Table 1. Mean egg-to-adult survival (mean ± S.E. in angles) for the selected populations on food supplemented with the different xenobiotics together with the survival of the control populations at the same concentration.

<table>
<thead>
<tr>
<th></th>
<th>Bogota</th>
<th>50 x 50</th>
<th></th>
<th>50 x 50</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>selected</td>
<td>control</td>
<td>selected</td>
<td>control</td>
</tr>
<tr>
<td>phenobarbital</td>
<td>1750 ppm</td>
<td>48.46±1.04</td>
<td>13.47±1.34</td>
<td>53.77±1.19</td>
</tr>
<tr>
<td>rotenon</td>
<td>30 ppm</td>
<td>43.44±3.97</td>
<td>28.05±0.83</td>
<td>50.71±5.36</td>
</tr>
<tr>
<td>malathion</td>
<td>0.90 ppm</td>
<td>59.14±0.94</td>
<td>5.18±1.11</td>
<td>55.02±2.60</td>
</tr>
<tr>
<td>carbaryl</td>
<td>36 ppm</td>
<td>50.41±1.29</td>
<td>0</td>
<td>51.82±1.98</td>
</tr>
<tr>
<td>DDT</td>
<td>90 ppm</td>
<td>53.46±1.02</td>
<td>17.34±1.50</td>
<td>51.83±1.66</td>
</tr>
</tbody>
</table>

Table 2. Estimated concentrations of the xenobiotics (in ppm) that give an egg-to-adult survival of 50% both for the selected populations and the control populations together with the ratio of these two.

<table>
<thead>
<tr>
<th></th>
<th>Bogota</th>
<th>50 x 50</th>
<th></th>
<th>50 x 50</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>selected</td>
<td>control</td>
<td>ratio</td>
<td>selected</td>
</tr>
<tr>
<td>phenobarbital</td>
<td>3500</td>
<td>950</td>
<td>3.7</td>
<td>4425</td>
</tr>
<tr>
<td>rotenon</td>
<td>25.5</td>
<td>12.5</td>
<td>2.0</td>
<td>56</td>
</tr>
<tr>
<td>malathion</td>
<td>2.90</td>
<td>0.18</td>
<td>16.1</td>
<td>2.0</td>
</tr>
<tr>
<td>carbaryl</td>
<td>41</td>
<td>10</td>
<td>4.1</td>
<td>42</td>
</tr>
<tr>
<td>DDT</td>
<td>210</td>
<td>17</td>
<td>12.4</td>
<td>210</td>
</tr>
</tbody>
</table>

The level of tolerance was determined by measuring the egg-to-adult survival of both selected and control flies on food supplemented with a series of different concentrations of the xenobiotic studied (selected flies were tested only on the chemical they were reared on). For the tests flies were collected from the cages and reared for one generation on normal food. Thereafter females were allowed to lay eggs on 2% agar gels for 6 h, after which the eggs were collected and cultured in vials, 100 eggs per vial. Table 1 shows the result when selected and control flies were reared at the same concentration. It is clear that the selected flies of both populations show a much higher survival than the control flies for all xenobiotics. Student-t testing of the difference in survival between selected and control flies showed that the difference was highly significant for all comparisons (P<0.001). This indicates a significant increase in tolerance in all selected populations. As the egg-to-adult survival was measured on different concentrations of the xenobiotics, it was possible to estimate the concentrations at which 50% survival would be expected. These concentrations are shown in Table 2 for both selected and control flies. Furthermore the relative increase in tolerance in the selected populations (represented by the ratio of these two) is shown in this Table. It is clear that the two populations have reacted in more or less the same way; the relative increase for each xenobiotic shows the same order of magnitude for both. On the other hand there are marked differences between the compounds.

Phenobarbital, rotenon and carbaryl show an increase in tolerance of 2-5 times, whereas the increase in tolerance for malathion and DDT is much greater, 15-20x. Both malathion and DDT treated flies seem to have become resistant to concentrations much higher than they have experienced during the experiment (highest concentrations used, respectively, 0.65 and 65 ppm). Especially the difference in tolerance increase between carbaryl (4 x) and malathion (15 x) is remarkable, as it is well known that both insecticides bring about their toxic effect in the same way by inhibiting acetyl-choline esterase activity in insects. The reason why some xenobiotics cause a much higher increase in tolerance is not clear at the moment. One possibility is that malathion and DDT have caused higher selection pressures in the cages and therefore might have effected a different tolerance mechanism. Investigation of the genetic bases of the increase in tolerance and the possible existence of cross-resistance is needed to elucidate this question.

The Drosophila genus is a material usually used for the genetic research, because flies are easily captured and reproduced in laboratory; their fecundity is high (Fowler 1973) and their vital cycle is short (Ashburner & Thompson 1978). Drosophila has 15 subgenus (Wheeler 1983); in our work we study two of them: Dorsilopha (one species, *D. buskii*), and Drosophila (49 species reported in Europe).

We sample Drosophilidae along the river valley Paramo-Nalon, in Asturias (North of Spain). This valley goes along 60 km. from 1300 m. of altitude to sea level, in S-N direction. His climate has a gradient from cold maritime in the high area to temperate maritime in the coast belt (Felicisimo & Alvarez 1980). This is why this valley is adequate for studying altitude influence over the Drosophilidae communities.

So, we can bring some new data about Drosophila species ecology and distribution.

Capture sites are in the map of Figure 1, as well as the ecosystem type of each one. We show in Table 1 the ecologic and climatic parameters that we have measured for each capture site. Thermopluviometric data are obtained in the nearest meteorologic stations. These data are missing for those sites where pluviosity and temperature records couldn't be taken.

Flies were captured during the last week of August. Eight traps with banana and yeast have been placed in nature for three days. Begg & Hogben (1946) and Parsons & Stanley (1981) have described this method as effective for capturing the species of Drosophila in the wild, though banana is not their natural nutritive source. Data are shown reuniting both sexes.

We have found 10 species of the Drosophila genus (Table 2). *D. simulans* and *D. melanogaster* are constant in all collection stations. *D. obscura, D. buskii, D. immigrans* and *D. funebris* are less constant, while the other four species are rare.

![Capture sites in Asturias, along the Paramo-Nalon valley.](image)
Table 1. Parameters determined in each site of capture.

<table>
<thead>
<tr>
<th>Sites</th>
<th>Altitude</th>
<th>Temperature</th>
<th>Pluviosity</th>
<th>Orientation</th>
<th>Dominant species</th>
<th>Total no. of individuals</th>
<th>Ratio: $D_{sim.}/D_{mel.}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Puerto Ventana</td>
<td>1500 m.</td>
<td>13.3°</td>
<td>9.0 mm</td>
<td>E (90°)</td>
<td>$D_{obscura}$</td>
<td>7</td>
<td>--</td>
</tr>
<tr>
<td>Cabanas</td>
<td>1090 m.</td>
<td>13.3°</td>
<td>8.2 mm</td>
<td>NW (330°)</td>
<td>$D_{simulans}$</td>
<td>9</td>
<td>7.04</td>
</tr>
<tr>
<td>Fresnedo</td>
<td>690 m.</td>
<td>18.0°</td>
<td>--</td>
<td>SE (120°)</td>
<td>$D_{simulans}$</td>
<td>95</td>
<td>16.13</td>
</tr>
<tr>
<td>Entrago</td>
<td>450 m.</td>
<td>--</td>
<td>35.0 mm</td>
<td>N (0°)</td>
<td>$D_{simulans}$</td>
<td>68</td>
<td>4.25</td>
</tr>
<tr>
<td>Proaza</td>
<td>210 m.</td>
<td>20.2°</td>
<td>27.2 mm</td>
<td>SE (110°)</td>
<td>$D_{simulans}$</td>
<td>478</td>
<td>8.06</td>
</tr>
<tr>
<td>Trubia nc.</td>
<td>180 m.</td>
<td>19.2°</td>
<td>32.4 mm</td>
<td>E (50°)</td>
<td>$D_{simulans}$</td>
<td>942</td>
<td>3.42</td>
</tr>
<tr>
<td>Trubia c.</td>
<td>90 m.</td>
<td>19.2°</td>
<td>32.4 mm</td>
<td>N (0°)</td>
<td>$D_{simulans}$</td>
<td>942</td>
<td>3.42</td>
</tr>
<tr>
<td>Sandiche</td>
<td>60 m.</td>
<td>19.5°</td>
<td>29.8 mm</td>
<td>Mid-valley</td>
<td>$D_{melanogaster}$</td>
<td>640</td>
<td>0.67</td>
</tr>
<tr>
<td>Soto del Barco</td>
<td>10 m.</td>
<td>18.0°</td>
<td>32.0 mm</td>
<td>NW (320°)</td>
<td>$D_{melanogaster}$</td>
<td>149</td>
<td>0.33</td>
</tr>
</tbody>
</table>

Table 2. Number of individuals of each captured species in each site.

<table>
<thead>
<tr>
<th>Sites</th>
<th>$D_{melanogaster}$</th>
<th>$D_{simulans}$</th>
<th>$D_{obscura}$</th>
<th>$D_{subobscura}$</th>
<th>$D_{funebris}$</th>
<th>$D_{buskii}$</th>
<th>$D_{phalerata}$</th>
<th>$D_{dimigrans}$</th>
<th>$D_{tes-tacea}$</th>
<th>$D_{hydei}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Puerto Ventana</td>
<td>--</td>
<td>--</td>
<td>7</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Cabanas</td>
<td>1</td>
<td>7</td>
<td>1</td>
<td>8</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Fresnedo</td>
<td>4</td>
<td>64</td>
<td>19</td>
<td>--</td>
<td>8</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Entrago</td>
<td>12</td>
<td>51</td>
<td>4</td>
<td>1</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Proaza</td>
<td>94</td>
<td>752</td>
<td>10</td>
<td>--</td>
<td>11</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Trubia nc.</td>
<td>72</td>
<td>293</td>
<td>28</td>
<td>--</td>
<td>11</td>
<td>3</td>
<td>71</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Trubia c.</td>
<td>212</td>
<td>724</td>
<td>1</td>
<td>--</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Sandiche</td>
<td>342</td>
<td>230</td>
<td>17</td>
<td>2</td>
<td>42</td>
<td>--</td>
<td>5</td>
<td>1</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td>Soto del Barco</td>
<td>102</td>
<td>34</td>
<td>13</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
<td>--</td>
</tr>
</tbody>
</table>

Drosophila abundance, measured as the total number of captured individuals, is the first index of the habitat resources for this genus (Table 1). Altitude is a limitant factor, though at 700 m. we captured a number of individuals not different from that of the captures at sea level (Garcia-Vazquez et al. 1985).

Diversity (Figure 2), as index of the community's variety, is measured by the Shannon formula. We find a negative correlation between diversity and altitude ($r = -0.65, 7$ df., $p<0.05$), mainly due to influence of the higher areas (above 700 m.). Climatic conditions in this area are not suitable for the Drosophila development. So, in Puerto Ventana only the resistant $D_{obscura}$ develops. $D_{melanogaster}$ and $D_{simulans}$ appear in Cabanas (1090 m.), but they are very less abundant than in the lowest areas. Favourable environment in low areas enable more diverse communities development.

Altitude has a different importance for each captured species. $D_{funebris}$ is associated to forest ecosystems, although it seems to be independent from the dominant tree species. $D_{funebris}$ does not compete with the other species because there is not a diminution of them when $D_{funebris}$ is more abundant. Altitude is not a decisive factor for this species.

$D_{buskii}$ and $D_{immigrans}$ appear respectively from Proaza and Trubia nc. to low areas; altitude
seems to be a limitant factor (stronger in D. simulans than for D. buskii), as we cannot associate their appearance with other ecological factor.

D. simulans is the most abundant species in this sampling. It is dominant in most of the capture sites, though D. melanogaster coexists with it. D. melanogaster and D. simulans are sibling species, which compart the same habitat (Parsons 1975); they have an effective sexual isolation (Mourad & Mallah 1960). Nevertheless we find a different development for both species according to altitude. D. melanogaster has a negative correlation between altitude and abundance (measured as the number of captured individuals, see Table 2): $r = -0.67, 6 \text{ df.}, p<0.05$; D. simulans seems to be the most resistant to adverse ambient conditions, and it accepts a greater ambiental variation. It is established in more extensive areas than D. melanogaster, and easily becomes the dominant species.

In high areas, temperature has much day-night variation; furthermore, the minimum temperature is less than in low areas. The fact that D. simulans have been favoured in that environment is contrary to findings of other authors. Parsons (1975) found that D. melanogaster is favoured against D. simulans when temperature variation is great; Cuesta & Comendador (1982) proved that D. simulans is less resistant to low temperatures than D. melanogaster.

Also, a seasonal substitution of D. melanogaster by D. simulans is cited: D. melanogaster is dominant in spring, whereas D. simulans is dominant in autumn (McKenzie & Parsons 1974). We think that this replacement is more rapid in high areas, under unfavourable ambient conditions.


Alexandrov, I.D. Research Institute of Medical Radiology, Obninsk, USSR. Modification of radiation-induced rates of intra- and inter-genic changes at the black locus of Drosophila melanogaster by the genotype, caffeine, actinomycin-D, sodium fluoride and radiation quality. In the same experiments described earlier (Alexandrov 1985), two hundred and forty-seven different black mutations (region 34D of 2L chromosomes) were recovered simultaneously with the yellow ones among the F1 progeny resulting from the mating of the irradiated males to ln(1)sc51 Sc8R dpl49, y sc51 Sc8 w; b cn vg females. Each of the black mutants was at first mated to b Pm/ln(2LR)Cy, net dp[t]C1 y b pr BlL3 cn2 L4 sp2 tester flies of the appropriate sex to obtain, when all was said and done, the black homozygotes. According to data of the genetic analysis, all the black mutations, as with the yellow ones, have been classified into 3 main and regularly occurring mutant types: (1) sterile F1 visibles (SV) (80 out of 247 black mutations scored), (2) transmissible visibles with recessive lethality (LV) (76 out of 167 fertile F1 mutants), and (3) transmissible and viable in homozygote visibles (VV) (all the other fertile mutants).

A further analysis of LV showed that one third of them (25 out of 76) had a dominant sterility and a low viability (LV5) (i.e., the F2 heterozygotes bx/b Pm or bx/ln(2LR)Cy, when inter-se crossed, yielded no progeny at all), but the rest of the LV were fertile ones (LVF) (51 out of 76). Some of the latter, when bxBI heterozygotes constructed were inter-se crossed, had a recessive lethality inseparable from the black phenotype (true LVF being deficiencies, inversions, etc., according to data of the genetical [Alexandrov & Alexandrova, this issue: New Mutants] analysis), whereas the others had an independent recessive lethal separated from the black mutation by crossing over in the bxBI female heterozygotes (so-called "twin" black mutants). Polytene chromosome analysis of LVF and the 68 VV preserved was also carried out, and the number of VV associated (VVch) as well as unassociated (VVB) with chromosome rearrangements was detected (Alexandrov & Alexandrova, ibid.).

A knowledge of a genetical nature of the black mutations scored has made it possible now to estimate the relative proportion of intra-locus changes (VVB) versus all inter-genic (i.e., chromosome SV, LV5, LVF and VVch) alterations affecting the chromosome region of interest after action of the variable studied (Table 1) (LVF are given as so far non-tested for the presence of independent recessive lethals). Corrected a.m.f. for VVB (i.e., taking account of the "twin" black LVF, among which gamma-ray-induced "twin" mutants are found to arise nearly twice as frequently as the neutron-induced ones) and for the chromosome rearrangements as a whole are listed in Table 2.
<table>
<thead>
<tr>
<th>Conditions of experiment</th>
<th>VVg</th>
<th>SV</th>
<th>LVg</th>
<th>LVf</th>
<th>Micro de- Visible re-</th>
<th>Lost before analysis</th>
<th>Total</th>
<th>a.m.f.**</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. gamma-rays, 40 Gy, D-32, No. = 192939</td>
<td>9</td>
<td>12</td>
<td>7</td>
<td>11</td>
<td>2</td>
<td></td>
<td>41</td>
<td>/1.2/</td>
</tr>
<tr>
<td>2. gamma-rays, 40 Gy, D-18, No. = 156127</td>
<td>19</td>
<td>14</td>
<td>3</td>
<td>5</td>
<td></td>
<td></td>
<td>41</td>
<td>/3.0/</td>
</tr>
<tr>
<td>3. caffeine (0.2%) + gamma-rays, 40 Gy, D-32, No. = 91729</td>
<td>18</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td>26</td>
<td>/4.9/</td>
</tr>
<tr>
<td>4. actinomycin-D (100 μg/ml) + gamma-rays, 40 Gy, D-32, No. = 69046</td>
<td>8</td>
<td>9</td>
<td>6</td>
<td>5</td>
<td>1</td>
<td></td>
<td>29</td>
<td>/2.9/</td>
</tr>
<tr>
<td>5. sodium fluoride (0.2%) + gamma-rays, 40 Gy, D-32, No. = 30861</td>
<td>8</td>
<td>6</td>
<td></td>
<td>3</td>
<td></td>
<td></td>
<td>17</td>
<td>/6.5/</td>
</tr>
<tr>
<td>6. 0.1, 0.35 and 0.85 MeV fission neutrons, 8-10 Gy, D-32, No. = 79839</td>
<td>2</td>
<td>10</td>
<td>5</td>
<td></td>
<td>13</td>
<td></td>
<td>31</td>
<td>/2.5/</td>
</tr>
<tr>
<td>7. 252Cf, 14 Gy, D-32, No. = 24072</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td>8</td>
<td>/5.9/</td>
</tr>
<tr>
<td>8. 0.85 MeV fission neutrons, 10 Gy + gamma-rays, 10 Gy, D-32, No. = 13377</td>
<td>1</td>
<td>4</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td>9</td>
<td>/3.7/</td>
</tr>
<tr>
<td>9. X- or gamma-rays, 40 Gy, c(3)G, No. = 143305</td>
<td>10</td>
<td>16</td>
<td>2</td>
<td>8</td>
<td></td>
<td></td>
<td>38</td>
<td>/1.7/</td>
</tr>
<tr>
<td>10. 0.35 and 0.85 MeV fission neutrons, c(3)G, 5 Gy, No. = 20605</td>
<td>1</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>3</td>
<td>/9.7/</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0</td>
<td>/0.0/</td>
</tr>
</tbody>
</table>

* Sterile in homozygote black mutants without visible rearrangements; ** a.m.f. = average mutation frequency, locus/r x 10^-8.

As seen, VV8 (corrected a.m.f.) arise nearly as frequently as chromosome rearrangements in the wild-type (D-32, D-18) and c(3)G male germ cells (the post-meiotic stages as a whole) after gamma-irradiation. On the other hand, fission neutrons and 252Cf are more efficient than gamma-rays in producing both the chromosome and the gene black mutations in different genotypes although this effect is more profound for the first than for the latter. Therefore, the relative proportion of intra- versus intergenic changes induced by high-LET radiations at the locus in question turn out to be 1:4 and 1:2 for the wild-type and c(3)G male germ cells, respectively. It is of interest that the consecutive irradiation by neutrons and gamma-rays increases the yield of the chromosome black mutations as well as VV8. This finding for the black VV8 is analogous to that for VV8 at the yellow locus (Alexandrov 1985). However, unlike the latter, pre-treatment of the D-32 males with caffeine or actinomycin-D (other things being equal) enhances the occurring of VV8 whereas the actinomycin-D, but not the caffeine, increases the yield of the black chromosome mutations (cf. the yellow ones, Alexandrov 1985).

The modifying effect of sodium fluoride (the males were fed on 0.2% NaF in 10% sucrose for 24 hr prior to irradiation) is marked for both gene and chromosome black mutations (Table 2), and this picture is found to be exactly opposite to that for the sex-linked loci. In particular, among 15522 X chromosomes studied in the same experiments, not one yellow mutation and merely 9 white mutants (among which only one was VV8) were scored. Therefore, sodium fluoride significantly increases the yield of gamma-ray-induced autosomal, but not sex-linked, visible mutations, at least under our experimental conditions. To answer the question, whether the action of sodium fluoride is a chromosome-
Table 2. Corrected (i.e., with accounting of the "twin" black mutants*) a.m.f.** for the gene (VV9) and chromosome black mutations scored in different experiments listed in Table 1.

<table>
<thead>
<tr>
<th>No. of</th>
<th>Total LVF</th>
<th>No. of &quot;twin&quot; LVF</th>
<th>Corrected a.m.f. for VVG ***</th>
<th>Chromosome rearr.***</th>
</tr>
</thead>
<tbody>
<tr>
<td>exp.</td>
<td>LVF tested</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.</td>
<td>11</td>
<td>8</td>
<td>2.6 (17)</td>
<td>2.7 (24)</td>
</tr>
<tr>
<td>2.</td>
<td>5</td>
<td>non-tested</td>
<td>3.0 (19)</td>
<td>3.5 (22)</td>
</tr>
<tr>
<td>3.</td>
<td>2</td>
<td>0</td>
<td>4.9 (18)</td>
<td>2.2 (8)</td>
</tr>
<tr>
<td>4.</td>
<td>5</td>
<td>3</td>
<td>4.0 (11)</td>
<td>6.5 (18)</td>
</tr>
<tr>
<td>5.</td>
<td>3</td>
<td>1</td>
<td>7.3 (9)</td>
<td>6.5 (8)</td>
</tr>
<tr>
<td>6.</td>
<td>13</td>
<td>4</td>
<td>7.5 (6)</td>
<td>31.3 (25)</td>
</tr>
<tr>
<td>7.</td>
<td>1</td>
<td>0</td>
<td>5.9 (2)</td>
<td>17.8 (6)</td>
</tr>
<tr>
<td>8.</td>
<td>3</td>
<td>2</td>
<td>11.2 (3)</td>
<td>22.4 (6)</td>
</tr>
<tr>
<td>9.</td>
<td>8</td>
<td>4</td>
<td>2.4 (14)</td>
<td>19.4 (20)</td>
</tr>
<tr>
<td>10.</td>
<td>2</td>
<td>0</td>
<td>9.7 (1)</td>
<td>19.4 (2)</td>
</tr>
</tbody>
</table>

* See text; ** See Table 1; *** Figures in () show number of mutations recovered.

When comparing data secured for both the black (this Note) and the yellow (Alexandrov 1985) loci, one must point out that, in the picture of the radiomutability of the two loci in question after action of the variables studied, there are very marked features in common (e.g., the same proportion of intra-versus inter-genic alterations after gamma-irradiation predominate the inter-over intra-geneic changes among neutron-induced mutations, etc.) as well as obvious distinctions (e.g., the different effects of the modifiers studied upon the yield of the yellow and black VV8) which appear to be conditioned by unique features of the fine structure of the loci themselves and/or of the neighbouring chromatin.

References:

Figure 1. Results of cytogenetic and genetic mapping of the radiation-induced chromosome breakpoints (*) in the 34A - 35E region of 2L chromosome of D.melanogaster. One asterisk corresponds to one breakpoint. Genetic and chromosome maps as described by Lindsley & Grell (1968).

Table 1. Cytology and genetics of the radiation-induced chromosome rearrangements recovered as the lethal black mutations.

<table>
<thead>
<tr>
<th>Rearrangements</th>
<th>Cytology</th>
<th>Genetics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tp(2) b71k1</td>
<td>34D2-4; 34D8-E1.2; 43C2-4</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b74c6</td>
<td>34D2-4; 34D8-E1.2</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b77c</td>
<td>34D2-4; 34F4-35A1.2</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b78j</td>
<td>34D2-4; 35A3-4</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b79a3</td>
<td>34D2-4; 34D8-E1.2</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b79b3</td>
<td>34C7-01.2; 35A4</td>
<td>b(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b79b4</td>
<td>34D2-4; 34E6-F1.2</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b79b8</td>
<td>34D2-4; 34F4-35A1.2</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>In(2L) b79d5</td>
<td>34D4(^+); 35B10(^+)</td>
<td>b(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>T(2,3) b79d6</td>
<td>34A2-3; 34D8-E2; 79B; 80C</td>
<td>b(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Tp(2) b79h1</td>
<td>34D2-4; 34D8-E2; 41</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b80k</td>
<td>34D2-4; 35B10-C1</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b80r</td>
<td>34D2-4; 34E2-E4.5</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b81a</td>
<td>34D2-4; 34D8-E2; 41D1-E1</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b81f1</td>
<td>34D2-4; 35A3-4</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Ef(2L) b81f2</td>
<td>34D2-4; 35A4-6</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>*In(2L) b81f3</td>
<td>34D2-4; 35B10(^+)</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>In(2L) b81g7</td>
<td>34D2-4; 40F</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>*In(2L) b82c44</td>
<td>34D2-4; 40F</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>In(2L) b82b22</td>
<td>34D2-4; 35B10(^+)</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b8329a</td>
<td>34D2-4; 35E2-E6</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b8311</td>
<td>34D2-4; 34E2-E5</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) + Df(2L) b8422</td>
<td>34C2; 34E5,6 (^+)</td>
<td></td>
</tr>
<tr>
<td>T(2;3) b8312</td>
<td>T(2;3) 34B12-C1; 35A2-3; 83A7-8; 83C</td>
<td>b(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b81d4</td>
<td>34D2-4; 34F4-35A1.2</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b84h30</td>
<td>34D2-4; 35C1-C3</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b85b1</td>
<td>34D2-4; 34E2-E4.5</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b85b2</td>
<td>34D2-4; 34D6-E1.2</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b85c1</td>
<td>34D2-4; 34F4-35A1.2</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>T(2;3) b85c2</td>
<td>34C7-D1.2; 34E1.2; 95C4-D1</td>
<td>b(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df(2L) b85f2</td>
<td>34D2-4; 34F3-4</td>
<td>nub(^+) (-) - j(^+) - rku (-) pu(^+)</td>
</tr>
<tr>
<td>Df + In(2L) b85f2</td>
<td>Df(2L) b8422</td>
<td>34E6-F1 (^+)</td>
</tr>
<tr>
<td>In(2L) 33A1-2; 35E3-4</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Viable in homozygote.

mutations in the region of interest (Woodruff & Ashburner 1979). Indeed, the pu phenotype shown to have first the pu/Df(2L)b87f2 heterozygote (lacking the bands up to 35B4), but not the pu/Df(2L)b87f1 one (lacking the bands up to 35A3). All the other and larger deficiencies, when they are heterozygous for pu, have the pu phenotype too (Table 1).

Acknowledgement: I am grateful to Dr. R.C. Woodruff, Bowling Green, Ohio, for supplying the nub, j, rk and pu stocks.

Al-Taweel, A.A. Iraqi A.E.C., Agriculture and Biology Faculty, Baghdad, Iraq. The frequency of spontaneous and induced recessive lethals in the sex chromosome of the *Drosophila melanogaster* wild type from Iraq.

The frequency of spontaneous sex-linked recessive lethals which occurred in nature and the induced gamma-radiation were determined by the Muller-5 technique in two Iraqi populations of *Drosophila melanogaster*. The first population was collected from Mosul, 396 km north of Baghdad (lat. 36; long. 43), and the second population was collected from Basrah, 545 km south of Baghdad (lat. 29.5; long. 47). Collections were made during June-August 1980. Immediately upon collection the flies were maintained in culture at 25°C in the laboratory in half-pint bottles with 50 ml food medium. The ingredients of the food medium in 1000 ml of water were agar (20 gm), date syrup (100 gm), Wheat (100 gm), yeast (50 gm), propionic acid (5 ml) and nepagin (15 ml) and were designated as M and B, respectively.

After several generations the frequency of spontaneous and induced SLRL-Mutations by 0.5 kGy of gamma radiation were determined by mating each male to virgin female of Muller-5 [(Sc B In-S W Sc) (Muller-5 originated from Genetics Dept., Aberdeen University)]. The Fl females were individually mated to Muller-5 male and the criterion of scoring the Fl as lethal was the complete absence of wild type males and the presence of at least 7D-15 Muller-5 males.

### Table 1. Frequency of spontaneous and induced SLRL-mutations by gamma-rays in natural *D.melanogaster* populations collected from different localities in Iraq.

<table>
<thead>
<tr>
<th>Population</th>
<th>CONTROL 0.5 kGy</th>
<th>No. Chromosomes tested</th>
<th>% lethal</th>
<th>No. Chromosomes tested</th>
<th>% lethal</th>
</tr>
</thead>
<tbody>
<tr>
<td>M</td>
<td>1443 (10)**</td>
<td>0.69</td>
<td>405 (20)**</td>
<td>4.93</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>1035 (8)</td>
<td>0.77</td>
<td>468 (19)</td>
<td>4.06</td>
<td></td>
</tr>
<tr>
<td>Oregon-K*</td>
<td>2737 (13)</td>
<td>0.47</td>
<td>1547 (112)</td>
<td>7.23</td>
<td></td>
</tr>
</tbody>
</table>

* Oregon-K sent to us from Genetics Dept., Aberdeen U. ** numbers in () represent lethal chromosomes recorded.

Table 1 showed the frequency of spontaneous and induced SLRL-mutations by gamma-radiation in natural populations of *D.melanogaster* in comparison with that of the Oregon-K and from these results we can draw the following conclusions:

1. No significant differences were observed between the spontaneous or induced SLRL-mutations by gamma-radiation in both Iraqi population (Chi-square test based on the observed frequencies were 0.0529 & 0.3935, respectively).

2. Also, no significant differences were noticed if the spontaneous SLRL-mutations were compared with that of the Oregon-K (M. vs. Oregon-K X^2=0.8205; B vs. Oregon-K X^2=1.2069).

3. Just not significant and a significant difference were observed if the induced SLRL-mutations were compared with that of the Oregon-K (M. vs. Oregon-K X^2=2.6982; B vs. Oregon-K X^2=5.981).

Finally the Iraqi populations have shown a resistance to induce SLRL-mutations in comparison with Oregon-K because they have lived for a long time under natural environmental conditions and recently adapted to the laboratory conditions while the Oregon-K has been adapted to the laboratory conditions for several years.


The sexual behavior of *drosophila* has been described by many authors, Ewing (1983) being one of the most recent. They emphasize the stereotyped components of male courtship giving rise to the choice of the female between males of different species. In spite of this cliche there is an individual variability which is not only a genetic effect.

Several authors have shown that the courtship behavior of *drosophila* is modified by the social experience of the male. For example, Mainardi (1967) has demonstrated that the male choice between two kinds of females (savage Oregon or yellow) depends on its individual experience. "Males reared in isolation perform their courtship at random" and court the Oregon female when reared in groups. This indicates that social experience during the beginning of the male imago life may modify its courtship.

More recently, Siegel & Hall (1979) found that a male having courted an unreceptive female presents an experience-dependent modification designated as "conditioned courtship" which lowers the possibility of copulation with a new female. This demonstrates that social experience immediately preceding courting may also modify the male's courtship.

The present paper shows that the physical-environmental factors (under which the male is bred during the first week of the imaginal life) do influence its sexual behavior.
Males from an outbred strain of *Drosophila melanogaster* (Brazzaville) are stored, just after hatching, individually in a test-tube and fed with a synthetic chemically defined medium (Marenco 1983). Four kinds of test-tubes are used: (1) glass test-tube about 75 cm$^3$ (T.L), (2) darkened glass test-tube about 75 cm$^3$ (T.D), (3) transparent plastic test-tube about 5 cm$^3$ (t.L), and (4) dark plastic test-tube about 5 cm$^3$ (t.D).

Females are stored in groups of ten in a 75 cm$^3$ glass test-tube with the same nutritive medium. For all the flies, this nutritive medium was changed every three days (under red light for T.D and t.D). Breeding and experiments are carried out under controlled conditions of humidity (80%), temperature (25°C), and for T.L and t.L photoperiod (L.D: 12/12, 100 lux from 8 a.m. to 8 p.m.). Courtship occurs in an observation-cell: a 20 mm high cylinder with an internal diameter of 25 mm lit by a 500 lux white light. Without anesthesia, male and female are individually placed in small transparent test-tubes. These two tubes are put into two opposite funnels driving the flies to the observation-cell. One to four minutes later, a sliding stick which separates the funnel from the cylinder, is withdrawn and the female is able to get into the observation-cell. Then the stick is pushed back and the same procedure is applied to the male. Observation begins when the male enters the cell and for 30 minutes we note the occurrence of all courtship elements. We only give below results concerning the precopulatory duration (from the time the male enters to copulation) and the copulatory duration.

A two-ways analysis of variance was made with a logarithmic transformation (Log$_e$) of these time periods (owing to an asymmetry in the distribution of raw scores). Table 1(A) presents this analysis; both dimensions show a significant effect, without interaction. As it can be seen in Figure 1, the fastest couples are those whose males were bred in a large test-tube (T.L), under a photoperiod. The precopulatory time-periods are considerably longer for the couples whose male was of t.L type, longer still for the couples whose male was T.D and the longest for couples whose male was bred in a small dark test tube (t.D).

The same statistical treatment was performed for the duration of copulation. The two-way analysis of variance (on the logarithmic transformation of the duration, Table 1(B)) shows the significant effect of the absence of photoperiod during the first week of the imago life. This effect interacts with the breeding space where the male is confined (5 cm$^3$ or 75 cm$^3$).

Such results are not only reducible to the consequences of a strong lightening occurring after a week of darkness for T.D and t.D groups. Such males are not only slow in action for their courtship, but also

---

**Table 1. Analysis of variance of data.**

**(A) Precopulatory duration:**

<table>
<thead>
<tr>
<th>Source</th>
<th>D.F.</th>
<th>Mean square</th>
<th>F value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Space</td>
<td>1</td>
<td>19.85</td>
<td>6.4 *</td>
</tr>
<tr>
<td>Photoperiod</td>
<td>1</td>
<td>37.50</td>
<td>12.1 ***</td>
</tr>
<tr>
<td>Interaction</td>
<td>1</td>
<td>4.81</td>
<td>1.5</td>
</tr>
<tr>
<td>Residual</td>
<td>198</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**(B) Copulatory duration:**

<table>
<thead>
<tr>
<th>Source</th>
<th>D.F.</th>
<th>Mean square</th>
<th>F value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Space</td>
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<td>.25</td>
<td>3.6</td>
</tr>
<tr>
<td>Photoperiod</td>
<td>1</td>
<td>.33</td>
<td>4.8 *</td>
</tr>
<tr>
<td>Interaction</td>
<td>1</td>
<td>.59</td>
<td>8.5 **</td>
</tr>
<tr>
<td>Residual</td>
<td>113</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* P<0.05; ** P<0.01; *** P<0.001

---

**Figure 1.** Precopulation and copulatory durations. Mean of Log$_e$ duration.
in copulation—even if they mate in less than 30 minutes—we think that the male-adaptation to the light is over before copulation, nevertheless the behavior of the couple is influenced by this lightening deficiency. The influence of space, even though less important than that of light, modifies courtship: couples whose males are bred in small tubes have a longer precopulatory period.

These preliminary results show that imaginal breeding conditions (individual experience) must influence the sexual behavior of Drosophila melanogaster.


Band, H.T. Michigan State University, East Lansing, Michigan. Emergence of D. simulans and other Drosophila from a variety of fruit/nut substrates.

D. simulans has become a colonizing species. Neither McCoy (1962) nor Sabath (1974) included it among the drosophilids collected in their studies in Indiana. However, it has been consistently collected in Michigan along with other Drosophila in the 1980s (Band & Band 1983; Band et al. 1984). Its emergence has now been documented from an array of substrates in Fall 1985, along with other drosophila. This is shown in Table 1.

Table 1. Emergence of Drosophilids from a variety of substrates in Fall 1985. D.m.=D. melanogaster; D.s.=D. simulans; D.b.=D. busckii; D.i.=D. immigrans; C.a.=Chymomyza amoena; o.c.= ornamental crabapples.

<table>
<thead>
<tr>
<th>site</th>
<th>date</th>
<th>substrate</th>
<th>females</th>
<th>males</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Oct.</td>
<td>apples</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>A</td>
<td>Oct.</td>
<td>walnuts</td>
<td></td>
<td>4</td>
</tr>
<tr>
<td>B</td>
<td>Oct.</td>
<td>o.c.</td>
<td>21</td>
<td>4</td>
</tr>
<tr>
<td>B</td>
<td>Sept.</td>
<td>pears</td>
<td>270</td>
<td>194</td>
</tr>
</tbody>
</table>

Emergence of D. simulans from ornamental crabapples at site B has been reported previously (Band & Band 1983). Carson (1965) found D. busckii breeding in walnut husks. However, D. immigrans has not been reported emerging from the walnut substrate.

The presence of D. melanogaster, D. simulans, D. busckii and D. immigrans at site A in 1985 represents an invasion since the site was last employed in C. amoena cold hardiness work. Apple and walnut trees are adjacent; fruits lie mixed on the ground. Selective substrate utilization at site A is evident. D. melanogaster and D. simulans occur in the rotting fruits; D. busckii in the walnut husks; D. immigrans and C. amoena in both substrates at site A.

The fact that D. busckii emerged from green fallen unripe apples in the mid-south (Band 1985) but from walnut husks from the site A farm is in agreement with the observations that this species does not ordinarily use fermenting fruits (Carson 1965; Atkinson & Shorrocks 1977). Emergence of D. immigrans from apples and walnuts agrees with previous findings that it is a generalist species (Carson, loc cit; Atkinson & Shorrocks, loc cit). Peak emergence at the site A farm had probably already occurred by the 27 Oct. collecting date. Numbers of D. immigrans and D. busckii emerging are lower than the numbers of pupae; many were used to determine cold hardiness characteristics. The incidence of D. immigrans is too low at site B to say whether or not this species is able to use ornamental crabapples for breeding; however, its emergence from this substrate has not been noted previously (Band & Band 1983).

Band, H.T. Michigan State University, East Lansing, Michigan. Evidence that Drosophila oviposits on ripe and rotting fruits on trees.

McCoy (1962) presented data that D. melanogaster in tomato fields in Indiana oviposit only on damaged fruits less than 8 inches off the ground. He concluded from his study that Drosophila used only damaged fruits at or near ground level.

Cavener and Clegg (1981) reported that D. melanogaster in apple orchards in Georgia were ovipositing on fruits on trees. Band et al. (1984) used D. melanogaster emerging from overripe pears pulled from a tree in 1982 in their study of the existence of LSP-1B5 polymorphisms in mid-Michigan D. melanogaster.

Data were accumulated in Sept. 1985 to establish that at least 4 species oviposited on ripe-to-rotting fruits on trees. All pears came from the same tree as in 1982. The umbrella shape of the canopy and moderate height insured accessible fruits. Twenty-five D. melanogaster (and simulans) flew out of the first pear plucked; the single female from the escapes from the hole at the calyx made them easy to count. Nine pears gathered on 9/22/84 and three on 9/29/85 were dissected. Four of the first group and one of the second, containing Drosophila eggs or larvae, were placed in individual jars and capped with a sturdy grade tissue. Potting soil was added as a substrate when 2 pears became watery and many larvae drowned. Table 1 shows the emergence data to 10/14 when all cultures were discarded because of mites.

In each collection one pear contained only dead larvae. A pH of 3 was recorded. All pears were frassy or had begun to rot when gathered and one had 46 Drosophila eggs in frass along a tunnel from the center to the calyx. Two also had Chymomyza amoena eggs and lesser apple worm eggs (Graptolitha prunivora) but cultures were discarded before the date of development. These pears did not contain codling moth larvae (Cydia pomonella) although two others did (but had no evidence of drosophilid invasion).

Although urea/uric acid have been found to have a negative effect on D. melanogaster development (Botella et al. 1985), the Drosophila species ovipositing on and their emergence from parasitized fruits in Michigan parallels findings of Drosophila breeding in fallen frassy unripe apples in the mid-South (Band 1985). The total difference between the laboratory larvae in medium, yeast and temperature and natural substrates, diversity of microflora and fluctuating temperatures may minimize the effects of the presence of excreta, both that of the initial occupant and of co-occurring individuals.

In any event, whatever the source of the attraction to ripe and overripe fruits on trees, D. melanogaster females do not oviposit only on fruits at or close to ground level. Neither do other Drosophila species.

Table 1. Emergence of Drosophila from larvae. A pH of 3 was recorded. All pears were frassy or had begun to rot when gathered and one had 46 Drosophila eggs in frass along a tunnel from the center to the calyx. Two also had Chymomyza amoena eggs and lesser apple worm eggs (Graptolitha prunivora) but cultures were discarded before the date of development. These pears did not contain codling moth larvae (Cydia pomonella) although two others did (but had no evidence of drosophilid invasion).

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Band, H.T. Michigan State University, East Lansing, Michigan. Occurrence of three chymomyzid species at Mt. Lake Biological Station in Virginia.

Chymomyza amoena and at least two additional undescribed species of chymomyzids exist at Mt. Lake Biological Station. On the station grounds all three could be observed displaying on freshly cut oak (Quercus sp.) wood in the early morning (7-8:30 a.m.) and early evening (7-8:30 p.m.). The first unidentified species was observed in mid-July. Five flies were trapped 7/18/85; three still survived on apples 7/22/85 but only one male was successfully mailed to the University of Chicago. A female, which escaped, had a shovel-like ovipositor. By the time trapping and media availability for mailing a series of captured flies could be coordinated, the first species had been replaced by a second unknown species. Seven males were successfully mailed to the University of Chicago on 8/16/85. Trapping methods were as previously employed for MI adult C.amoena and consist of placing an empty receptacle over displaying flies, causing them to fly upward into the trap.
Table 1. Observations on 3 chymomyzid species, July - August 1985, at MLBS in Virginia. Two with unbanded wings plus *C.amoena*; m=males; f=females; p=progeny.

| date    | time | # unbanded | #^
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>7/18</td>
<td>p.m.</td>
<td>7</td>
<td>0</td>
</tr>
<tr>
<td>7/19</td>
<td>a.m.</td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td>7/22</td>
<td>a.m.</td>
<td>15</td>
<td>2</td>
</tr>
<tr>
<td>7/29</td>
<td>p.m.</td>
<td>20</td>
<td>2</td>
</tr>
<tr>
<td>8/6</td>
<td>p.m.</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>8/12-14</td>
<td>p.m.</td>
<td>at least 7</td>
<td>7m</td>
</tr>
</tbody>
</table>

Chymomyza amoena co-occurred with at least one unknown chymomyzid species and was the only species with banded wings. Numbers of all three species were low although C.amoena was consistently less frequent than adults of either of the other two species. This is shown in Table 1. Wheeler (1952) lists chymomyzids as being found around peeled areas of tree trunks: aspen, alder, fir, pine. Attraction to Oak is not mentioned. However, C.amoena has a lesser tendency to be associated with wood than other species (Wheeler, ibid). Watabe (1985) has also reported the morning and evening display times of chymomyzid males at timberyards in Japan. Oaks were among the cut logs.

All chymomyzids observed at MLBS were aggressive. All were wing-wavers, a typical chymomyzid characteristic that has been absent in some species in New Guinea (Okada 1981). Males attempted to mate indiscriminately between species. All could be kept alive on ripe apples although there was no record of emergence of either of the undescribed species from apples collected at 3 sites outside the station grounds in studies on C.amoena oviposition behavior; no unidentified chymomyzid larvae or unemerged pupae were later found in apples brought back to Michigan State University to suggest apples might be feeding/overwintering sites for larvae of the unknown species.

Only two adult C.amoena were observed at one of the three sites where apples were collected although a total of 95 C.amoena emerged from those found to contain C.amoena eggs. Eighty-four adults emerged from apples on the ground and 11 from apples pulled from trees. Pupation was mostly within apples; only 23 pupal cases were recovered from soil substrates supplied for individual records per apples with C.amoena eggs.

A single C.amoena male successfully trapped was mated with newly emerged Michigan C.amoena (from apples) on 7/19/85. Eggs were observed by 7/30/85, hatching by 8/3/85. Emergence to oviposition in 10 days and oviposition to hatching in 4 days is consistent with unpublished studies on interpopulation crosses in Michigan from apple substrates and interstate crosses between Michigan and Missouri C.amoena.

The total of 12 males, representing 3 species, and 1 female trapped on separate occasions agrees with Japanese findings that males predominate in collections. Differences in courtship displays were not noted but see Watabe (1985). Numbers of all species are lower than for chymomyzid collections at the Jyozankei timberyard (Watabe 1985). However, total population sizes at any one time were also small.

Acknowledgements: Thanks are gratefully extended to Lynn Thockmorton for establishing the existence of two undescribed chymomyzid species in the Virginia mountains, to Bruce Wallace at VPI&SU and the Drosophila group at the University of Virginia for supplying media and mailing cartons on different occasions, and to Jerry Wolff, Director of MLBS, for supplying space for research.


Michigan State University, East Lansing;
*Universidad de la Laguna, Tenerife, Canary Islands, Spain. Comparison of the two numbering systems for the larval hemolymph proteins of Drosophila melanogaster.

Two different numbering systems have been applied to the larval hemolymph proteins of D.melanogaster. Gonzalez et al. (1982) used the numbering system of Loukas et al. (1974) for D.subobscura (see also Cabrera et al. 1983). Singh et al. (1982) and Singh & Coulthart (1982) applied the numbering system of Doane & Treat-Clemons (1981); they surmised that proteins 16, 15, 11, 10, and 9 were SGS-3, LSP-2, LSP-1q, LSP-1β, and LSP- 1 of Akam et al. (1978) and Roberts & Roberts (1979). Band et al. (1984) used 2-dimensional electrophoresis to partition LSP-1 at pH 7 into the 3 component proteins of Roberts & Roberts (1979) in order to study the existence of LSP-1β in northern U.S. populations; it verified that the numbering system of Doane & Treat-Clemons (1981, 1982) was the appropriate one for D.melanogaster. There was no way to relate the two numbering systems to one another.

To correlate the two numbering systems, stocks of D.melanogaster from Tenerife, Canary Islands, Spain and Cordoba, Spain, were mailed to East Lansing. Hemolymph proteins from larvae (and pupae) were electrophoresed along with OR-R controls as described by Band et al. (1984) and then compared to a
Table 1. Comparison of the numbering systems that have been applied to the larval hemolymph proteins of *Drosophila melanogaster.*

<table>
<thead>
<tr>
<th>SGS</th>
<th>Pt. 16</th>
<th>Pt. 15</th>
</tr>
</thead>
<tbody>
<tr>
<td>LSP-2</td>
<td>Pt. 15</td>
<td>Pt. 10</td>
</tr>
<tr>
<td>LSP-1 α</td>
<td>Pt. 11</td>
<td></td>
</tr>
<tr>
<td>LSP-1 β</td>
<td>Pt. 10</td>
<td>Pt. 8</td>
</tr>
<tr>
<td>LSP-1 γ</td>
<td>Pt. 9</td>
<td>Pt. 7</td>
</tr>
</tbody>
</table>

SGS = salivary glue secretion (salivary gland); LSP = larval serum protein (fat body).

Photograph of *D.melanogaster* larvae showing proteins numbered according to the system of Loukas et al. (1974) and applied by Gonzalez et al. (1982).

It is possible to construct the following identities between the two systems. This is given in Table 1. The results thus indicate that pt. 10 and pt. 7 and 8 represent fat body proteins in an array of species: *D simulans, D mauritiana* (Gonzalez et al. 1982); *D pseudoobscura* (Lewontin 1974; Singh & Coulthart 1982; Cabrera et al. 1983); *D guanche, D madeirensis, D subobscura, D ambigua* (Cabrera et al. 1983); *D persimilis* (Lewontin 1974).

Of special interest is the fact that LSP-2 F, which is rare in *D melanogaster* (Singh et al. 1982) and carries a lethal or semi-lethality (Hoogwerf & Roberts 1982), is the predominant polymorphism in *D simulans* where it also may tend to lethality or semilethality as a homozygote (Band, unpubl.); it is the form of LSP-2 in *D mauritiana* (Gonzalez et al. 1982). Other species do not seem to have fixed this allele of LSP-2. LSP-2 as pt. 10 may also be involved in intra- and interpopulation cold resistance in *D pseudoobscura* pupae (Marinkovic et al. 1969; Jefferson et al. 1974; Coyne et al. 1983). β is on chromosome 2; LSP-2 as pt. 10 is on chromosome 3 in *D melanogaster* (Lewontin 1974). This is the protein called larval hemolymph protein (LHP) by Beverley & Wilson (1982, 1984) and has been used by them to construct phylogenetic trees among the drosophilids and other dipterans.

The failure to detect all 3 LSP-1 proteins by Gonzalez et al. (1982) may be a consequence of the technique, if limited migration prevents proper separation of the α and β proteins. LSP-1 F was present in at least one larva in the photograph sent to E. Lansing, so the faster migrating alleles for β may represent, respectively, α and β homozygotes and α heterozygotes.

However, LSP-1 over an array of drosophilids does not display 3 independently migrating proteins (Brock & Roberts 1983; Band & Band, unpubl.). Also, separate α and β proteins are difficult to discern in *D simulans* when β is β F. Included as an allele of β, between species comparisons do enable determination of the existence of comparable rapid migrating LSP-1 proteins across an array of species. LSP-1 proteins in the *obscura* group tend to migrate at a faster rate than in the *melanogaster* group; LSP-2 tends to migrate slower among the *obscura* group than in the *melanogaster* group (Gonzalez et al. 1982; Cabrera et al. 1983).

Thus, speciations both within and between the 2 groups have involved the larval fat body proteins and their polymorphisms. Since to date there is no evidence that drosophilids are synthesizing glycerol, other polyols or relying on sugars for cold tolerance (Kruinic et al. 1980; Band & Band 1980, 1982; Kimura 1982) while the fat body is reconstituted in drosophilids that show a discrete adult diapause (Carson & Stalker 1948; Begon 1976) and fat deposition increases in at least one species, *D subobscura,* which doesn't have an adult diapause (Begon ibid.), fat body proteins may play a role in overwintering in addition to functioning as storage proteins for development.


Ritossa & Spiegelman (1965) have shown that the rDNA quantity was directly proportional to the number of nucleolar organisers (NO) in genome. However, Ritossa et al. (1966) revealed that D. melanogaster females with four NO per genome lost about half of the rRNA genes over a period of several generations. Krider & Plaut (1972) also found that in homozygous females with four NO, X chromosomes despite having two NO frequently carried mutations for the bobbed locus. The partial loss of rDNA during several generations of multinucleolar flies was also observed by Kubaneishvili et al. (1983). These data are in contradiction with those obtained by Spear (1974) who did not observe instabilities in the rDNA content over 10 generations of multinucleolar genotypes, though in one of the generations he noted rRNA gene number variation but accounted it as artifact.

One shortcoming was shared by all these experiments: in all of them the In(1)sc4Rsc8L chromosome was used for synthesis of multinucleolar genotypes, though the regions important for maintenance of rRNA gene number stability are affected in this chromosome. Therefore, we performed the synthesis of female genotypes with three NO (termed trinucleolar females) using only structurally normal X chromosomes. An additional rRNA gene dosage was introduced into genome with an extra Y chromosome. The rRNA gene number was determined by using RNA/DNA hybridization technique. The results of typical experiments are presented in Table 1. The data obtained for other trinucleolar genotypes are represented in Fig. 1. It is obvious that in all these cases there is a considerable loss of rRNA gene copies in comparison with the theoretically expected values. This loss amounts to 70-300 genes. The rRNA gene reduction in a single NO of trinucleolar genotypes is termed by us retrocompensation. The matings between individuals from the stock reared with selection of multinucleolar females and wild (with respect to NO) individuals showed that trinucleolar females transmitted fewer ribosomal cistrons to the progeny than their normal brothers with two NO. The trinucleolar females in the

### Table 1. rDNA content in the 5th-6th generations of the stock reared with selection of the females y ac sc w/y ac sc w/BY wY+. Y

<table>
<thead>
<tr>
<th>Genotype</th>
<th>rDNA (%)</th>
<th>rRNA gene no.</th>
<th>X</th>
<th>Y</th>
<th>A</th>
</tr>
</thead>
<tbody>
<tr>
<td>y ac sc w/y ac sc w</td>
<td>0.470</td>
<td>537±12</td>
<td>269</td>
<td></td>
<td>--</td>
</tr>
<tr>
<td>y ac sc w/BY wY+Y</td>
<td>0.426</td>
<td>486±13</td>
<td>269</td>
<td>217</td>
<td></td>
</tr>
<tr>
<td>y ac sc w/y ac sc w/BY wY+Y</td>
<td>0.333</td>
<td>419±15</td>
<td></td>
<td></td>
<td>335</td>
</tr>
</tbody>
</table>

X - number of X chromosomal rRNA genes.

\[ X = \frac{rRNA \text{ gene number in } \text{XX}}{2} \]

Y - number of Y chromosomal rRNA genes.

\[ Y^2 = (rRNA \text{ gene number in } \text{XY}) - X \]

A - difference between the observed and theoretically expected (T) rRNA gene number in XXY females.

\[ T = 2X + Y \]

Figure 1. Relative rDNA contents in XXY females of different genetic backgrounds (the % ratio of the observed rDNA contents to theoretically expected ones). 1 - y ac sc w/y ac sc w/BY wY+; 2 - y ac sc w/y ac sc w/bwY y+; 3 - Canton S/Canton S/BY wY+; 4 - y ac sc w/y ac sc w/BY wY+Y; 5 - y2su(w3) w3/y2su(w3) w3/gBY y+; 6 - Swedish b/Swedish b/BY wY+; 7 - In(1)sc8/In(1)sc8/BY wY+; 8 - In(1)wmb4/In(1)wmb4/gBY y+. 

[Graph showing rDNA content for different genetic backgrounds with bars representing the % ratio]
The observed modulations of the rDNA content in these cases are probably provoked by the fact that during restoration of the rRNA gene number in the "retrocompensating" chromosomes the rDNA quantity rose above the normal level. This, in turn, reactivated the regulatory genes provoking retrocompensation. The system controlling the number of rRNA genes appears to be reorganized not always in time. Thus, the number of rRNA genes sometimes fails to be corrected in a necessary direction even when this can result in lethality. For example, see Fig. 2a where it is shown that the decrease in the rDNA content starting in the early generations did not stop leading to the death of stock in the 10th generation.

According to our results, retrocompensation does not occur in the first generation of trinucleolar females. The F₁ females with additional NO demonstrate additivity of contributions of all three NO into the total rDNA content. Hence, retrocompensation is a stepwise process and continues throughout several generations. Thus, in continuously maintained genotypes with an additional NO, retrocompensation occurs over a period of several generations which results in an essential DNA loss, probably in each of three NO. rDNA retrocompensation is in a certain sense opposed to magnification.

Two *D.ananassae* strains (Ana-2 and Ana-5) were started with three females collected in the same place at Olimpia city (SP) Brazil, and were kept up isolated in a corn-wheat flour medium during 2435 days. The data in Table 1 show the values obtained for the descendant of the crosses with the two strains, $F$ values of the analysis of variance and $W_{0.05}$ (Tukey's test) for comparison among the means. Those means followed by the same letter are statistically equal; if not, they are different.

<table>
<thead>
<tr>
<th>Type of Crosses</th>
<th>Female</th>
<th>Ana-2</th>
<th>Ana-5</th>
<th>Ana-2</th>
<th>Ana-5</th>
<th>Values</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>Ana-2</td>
<td>Ana-5</td>
<td>Ana-5</td>
<td>Ana-5</td>
<td>Ana-2</td>
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<td></td>
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<tr>
<td>NFL</td>
<td>245.17±22.70c</td>
<td>115.67±13.42b</td>
<td>40.75±12.29a</td>
<td>65.75±14.20ab</td>
<td>29.01**</td>
<td>63.72</td>
<td></td>
</tr>
<tr>
<td>BI</td>
<td>209.01±19.96c</td>
<td>116.12±13.18b</td>
<td>35.64±10.53a</td>
<td>58.85±12.61a</td>
<td>28.22**</td>
<td>55.01</td>
<td></td>
</tr>
<tr>
<td>VI</td>
<td>0.95±0.01</td>
<td>0.93±0.01</td>
<td>0.81±0.06</td>
<td>0.87±0.05</td>
<td>2.41**</td>
<td>1.31</td>
<td></td>
</tr>
<tr>
<td>DT</td>
<td>16.15±0.23b</td>
<td>15.87±0.19b</td>
<td>14.68±0.28a</td>
<td>14.95±0.22ab</td>
<td>4.21*</td>
<td>1.31</td>
<td></td>
</tr>
</tbody>
</table>

ns = not significant ($P>0.05$); * = significant ($P<0.05$); ** = significant ($P<0.01$).

The viability (VI) was measured by the formula: number of flies (NFL) divided by the sum of the number of flies, number of larvae and number of pupae. For development time (DT) the formula $DT = \left(t + (t - t')/2\right) + T$ (Carvalho 1981) was used; where $t$ is the number of days between the withdrawal of the parental flies from bottles (1/4 of liter) and the first imago counting; $t'$ is the interval between two countings (one day) and $T$ is the maintenance time of the paternal flies in the bottles (three days). Each cross type was mounted with 20 couples and 12 repetitions.

The results concerned with the number of flies and biomass (mg) showed that crosses between the two different strains had lower productivity than the crosses of the same strains individuals, which seems to indicate any reproductive isolation degree between the two strains, and the Ana-2 strain was superior to Ana-5 in productivity. There was no variation concerned with viability, and the developmental time of the hybrid individuals were shorter than the homozygote flies. On the other hand, the crosses were not enough to show any maternal effect of the flies.


Botella, L.M. & J.L. Mensua. Universidad de Valencia, Spain. Arrested development in second and third larval instars of *D.melanogaster* in highly crowded cultures. Mensua & Moya (1983) put into evidence the existence of a larval stop in the third larval instar of *D.melanogaster* when reared in crowded conditions. Further studies (Mensua et al. 1983) have shown that the larval arrest occurs at third instar in a range of densities from 21 to 83 larvae in 0.5 ml of food at 25°C. Nevertheless, Perez Tome (1980) reported that in highly crowded cultures of *D.hydei* larval size showed two peaks corresponding, respectively, to the 2nd and 3rd instar. In these conditions this author reported that only a small fraction of the total competing population progresses towards the third instar.

In order to establish a comparison between this phenomenon occurring in *D.hydei* and the larval stop reported in *D.melanogaster*, the following experiments were designed. Different sets of 5 (5 x 0.8 cm) vials supplied with 0.5 ml of Lewis medium were seeded with either 150, or 300, or 500 recently hatched larvae of an isogenic Oregon-R strain. Cultures were incubated at 25°C. At different times from the seeding day (8th, 10th, 12th, 14th and 16th days), crowding was interrupted by the overfeeding technique (Moya & Mensua 1983). The results obtained concerning larva-adult viability and mean developmental time are shown in Figure 1. As regards viability, the decreases are sigmoidal-like, though far more marked when the initial density is of 300 or 500 larvae, the reduction in 50% of the initial population being reached two days earlier in these latter densities than at the density of 150 larvae.
Figure 1. Larva-adult viability and mean developmental time in highly crowded cultures of *D. melanogaster*.

Figure 2. Absolute adult emergence in time in 3 earlier overfeedings at density of 500 larvae in 0.5 ml food.
In relation to mean developmental time, at a density of 150 larvae, the regression over the different overfeedings is similar to that found by Mensua & Moya (1983) working with 70 larvae in 0.5 ml (the slope close to 1), but the regression lines at higher densities (300 and 500) differ considerably.

Samples of 100 larvae were analyzed at the different overfeedings by jaw analysis under the light microscope to determine their stage of development. While at a density of 150 all larvae were stopped in third instar, it was found that at higher densities a great proportion (amounting to 60% at a density of 500 larvae) was stopped in 2nd instar. This result moved us to consider that perhaps at these high densities, the phenomenon detected in D. hydei was being reproduced. In an attempt to further study the development of larvae at the highest density (500 larvae in 0.5 ml), the number of adults emerged daily over the development of the cultures has been presented in Figure 2 for the three earlier overfeedings. It can be seen, above all when crowding is interrupted earlier (first overfeeding at the 8th day) that two peaks of adult emergence in time become apparent. The first peak (the sooner in emergence) is smaller and corresponds to larvae which have previously been stopped in 3rd instar, and the second and more numerous peak corresponds to larvae stopped in the second instar before the overfeeding took place. When the regression of mean developmental time was calculated for each peak over the three first overfeedings, the slope was equal to 1 for both peaks, which seems to support the existence of a larval stop in development in both instars: 2nd and 3rd.

Altogether, we may say that competition in densities within the chaos range (Hassell et al. 1976) gives rise to a double stop in larval development of D. melanogaster. Populations might have evolved to face highly unfavourable conditions through the acquisition of a "double brake" in their development. In the absence of this mechanism the final outcome of competition would be catastrophic, since the amount of food available in such crowded conditions is hardly enough for the development of a 2% of total population. However, the possibility of developmental arrests enables the larval population to wait for the eventual supply of new food resources.


The existence of a larval arrest in D. melanogaster development (Mensua & Moya 1983) may be explained by means of two hypotheses which are not mutually exclusive. One of these hypotheses is based on the idea that an accumulation of residual metabolites, laid by the competing larvae in a limited space, would lead to a stopped development in the last larval instar. This hypothesis has been supported by the results obtained with urea and uric acid as residual compounds by Botella et al. (1985). The second possibility is that the nutritional scarcity in some essential food principle may interrupt also larval development.

This idea has been tested by the use of axenic synthetic media, either complete or deficient in each single essential food principle for Drosophila development. The complete synthetic medium employed was that proposed by Sparrow & Sang (1975). The experiments were performed as follows. Crowded cultures were established by seeding 70 recently emerged larvae from an isogenic Oregon-R strain in small vials containing 0.5 ml of a yeasted sterile medium. These cultures were reared at a temperature of 25°C, and crowding was interrupted at different days from the starting in different sets of 5 small vials by the technique of overfeeding (Moya & Mensua 1983). The media used in each overfeeding (in an amount of 10 ml in inclined disposition) were either complete or deficient in the following essential nutrients: cholesterol, choline, casein, inosine, and vitamins. A negative control medium only made out of water, agar, salts and sugar was also employed. Adults emerging every day in the overfeeding vials were recorded until the exhaustion of the cultures. All the experiments were carried out in sterile conditions.

The results obtained as regards viability in complete and deficient medium for the larvae recovered in the different overfeeding vials are shown in Figure 1. All the different media showed slightly lower viability than the medium containing the lowest viability was obtained in a casein deficient medium. Thus, proteins seem to be the main scarce principle in our crowded conditions. In Figure 2 mean developmental times over the different overfeedings in the different synthetic media are represented. No significant differences among the slopes of the regression lines were found. This result means that food deficiency cannot be the actual and only cause for the arrest in development. If a larva is stopped because it needs some particular nutrient requirement, it should remain in this stage for the deficient medium does not provide it. However, the adults emerged in time in the different deficient media following the same regularity as in the complete medium throughout the overfeedings. As a conclusion from this group of experiments, we can say that food scarcity may explain the decrease in viability operated in crowded
Figure 1. Larva-adult viability of the stopped larvae in different synthetic media in different overfeedings.

conditions, but does not seem to be the direct and actual cause of the larval arrest in 3rd instar developmental stage of Drosophila.


The process of selection for development time in Drosophila has usually led authors to find an asymmetrical response with a slight or almost absent progress in the fast direction (Sang & Clayton 1957; Robertson 1963). Bakker & Nelissen (1963) explain this type of response to selection because developmental time is a selectively optimized character in those species, such as Drosophila, which depend on temporary food resources. On the other hand, it is well documented that mean developmental time may be considerably delayed by competition for food (Bakker 1961; Robertson 1963; Miller 1964; Barker & Podger 1970; Mensua & Moya 1983). Nevertheless, there are no references in literature on what occurs in a process of selection for developmental time when it is carried out under crowded conditions. We have focused our study on this point, since it is well known that many responses shown under suboptimal conditions, may be hidden in more favourable environments (Robertson 1963, 1964).

Selection for faster and slower developmental time was practised as follows. Two lines of selection were started from a stock of Drosophila melanogaster consisting of a mixture of three different wild stocks: one from laboratory origin (Or-R) and the other two from natural origin. In each line, a total of eight 5 x 0.8 cm vials with 0.5 ml of a boiled yeasted medium (Lewis medium) were seeded with 70 recently hatched larvae each. In the fast line, competition was interrupted at the 8th day from the seeding by means of the "overfeeding technique" (Moya & Mensua 1983). In this way the initial population was divided into two subpopulations: an "inner subpopulation" constituted by those larvae which have pupated inside the vials before the 8th day, and an "outer subpopulation" composed by those larvae recovered by overfeeding. The four earliest couples to emerge as adults from the "inner population" were selected as parents in successive generations. In the slow line, overfeeding was postponed until the 12th day. Larvae recovered by overfeeding constituted the "outer subpopulation". The four latest couples to emerge as adults in this latter subpopulation were chosen as parents of the next generation. Selection proceeded for 10 generations in the slow line and for 15 generations in the fast line. Two replications were run simultaneously for each line.

Figure 1 shows the mean developmental time obtained over the selection process in both directions. The straight lines are the best fit regressions. As can be seen, there is a slight significant trend to a decrease in mean developmental time in the course of selection in the fast direction, if one excludes the initial generation. This tendency is observed in both replications, F1 and F2. In the slow direction there is an increase during the early generations (from 0-5th in SL1, and from 0 to 4th in SL2).

In the case of the SL2 line, two regressions have been represented which correspond from the 1st to the 15th generation and from the 2nd to the 15th generation, respectively. It is also worth mentioning that the drop in developmental time found between generations 5 and 6 in SL1, and between 4th and 5th generations in SL2 was due to a failure in the temperature regulation system. However, there was no significant increase in developmental time later. The results are altogether in concordance with others reported in literature by Sang & Clayton (1957) and Robertson (1963), in relation to the asymmetry and type of response obtained in uncrowded conditions. However, when selection in crowded cultures is further studied with the help of an "overfeeding technique", a new type of response becomes apparent.

Table 1 presents the differences between developmental times in outer and inner subpopulations in each generation over the process of selection in both directions. Two main points deserve attention: (i) Differences in the slow lines are larger than in the fast lines, which is not surprising since crowding was kept for a longer time in these latter (12 days in slow lines compared with 8 days in fast lines). (ii) In the fast direction, differences tend to decrease in time in a more or less regular way. This trend may be explained in terms of a response to selection pressure in both subpopulations "inner" and "outer", though more marked in this latter, where developmental rates would be further increased.

### Table 1. Differences between outer and inner mean developmental times in the fast and slow lines throughout the process of selection.

<table>
<thead>
<tr>
<th>Generations</th>
<th>F1</th>
<th>F2</th>
<th>SL1</th>
<th>SL2</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>4.33</td>
<td>3.46</td>
<td>6.99</td>
<td>5.56</td>
</tr>
<tr>
<td>1</td>
<td>4.57</td>
<td>4.36</td>
<td>5.69</td>
<td>6.50</td>
</tr>
<tr>
<td>2</td>
<td>4.37</td>
<td>2.72</td>
<td>4.48</td>
<td>7.81</td>
</tr>
<tr>
<td>3</td>
<td>3.69</td>
<td>3.86</td>
<td>6.63</td>
<td>5.87</td>
</tr>
<tr>
<td>4</td>
<td>3.99</td>
<td>3.48</td>
<td>5.88</td>
<td>7.43</td>
</tr>
<tr>
<td>5</td>
<td>3.67</td>
<td>3.29</td>
<td>7.64</td>
<td>5.19</td>
</tr>
<tr>
<td>6</td>
<td>3.39</td>
<td>3.62</td>
<td>5.98</td>
<td>6.02</td>
</tr>
<tr>
<td>7</td>
<td>3.74</td>
<td>3.37</td>
<td>6.10</td>
<td>5.58</td>
</tr>
<tr>
<td>8</td>
<td>2.89</td>
<td>2.49</td>
<td>7.85</td>
<td>6.70</td>
</tr>
<tr>
<td>9</td>
<td>2.81</td>
<td>2.94</td>
<td>5.88</td>
<td>8.28</td>
</tr>
<tr>
<td>10</td>
<td>3.13</td>
<td>3.21</td>
<td>5.35</td>
<td>5.69</td>
</tr>
<tr>
<td>11</td>
<td>3.12</td>
<td>2.64</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>2.17</td>
<td>3.29</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>3.80</td>
<td>2.69</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>2.99</td>
<td>4.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>3.10</td>
<td>3.41</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*a=Values of regression over generations: slope = -0.10, intercept at origin=4.23; p<0.001. b=slope = -0.03, intercept at origin=3.53; n.s. c=slope= 0.016, intercept at origin=6.14; n.s. d=slope=0.02, intercept at origin=6.31; n.s."
On the contrary, the slow lines show a typical pattern where an increased difference is always followed by a decreased one over the process of selection. This alternating pattern between successive generations may be obtained as a consequence of the following "feed-back" or "relais" mechanism. When inner subpopulations take longer to develop, crowding is stronger, and hence the least adapted genotypes will be eliminated. The best adapted genotypes in crowding conditions (recovered by overfeeding) will develop faster in outer subpopulations. Thus, the intragenerational difference in outer and inner mean developmental times would be short. The best adapted genotypes will be selected as parents of the next generation, and will give rise to a progeny, part of which will take short to develop in crowded conditions. The remaining larvae, recovered by overfeeding (less adapted genotypes) will take longer to emerge as adults in outer subpopulation, and then, the intragenerational difference between outer and inner mean developmental times would be large.


Capy, P. and J. Rouault. C.N.R.S., Gif-sur-Yvette, France. Sampling on decaying fruit of Opuntia. In mediterranean countries, one of the most frequent breeding sites for Drosophila species is the fruit of Opuntia ficus-indica. Generally, fruit flies and their parasites are found in the cavity of decaying fruit. This cavity is closed to the outside except for a small hole. When fruit is touched insects fly away one by one because of the narrowness of the hole.

Using two fly aspirators, two consecutive samples have been collected from the same fruits. The first sample corresponds approximately to the first twenty seconds, and the second one to the last thirty seconds of the collection. This sampling method was carried out in November 1982 when a large number of fruits were available (Nasr'Allah, Tunisia).
Three Drosophila species were found: D. melanogaster, D. simulans and D. buzzatii; and one parasite: Leptopilina boulardi. These results are similar to those previously reported by Rouault & David (1982) and David et al. (1983).

The number of individuals caught per species and per sample are given in Table 1. Owing to the determination error due to the likeness of D. melanogaster and D. simulans females, individuals of these species have been grouped.

The size of the two samples are similar. However, there is a significant difference between the two samples of Drosophila species. It appears that D. melanogaster and D. simulans fly away before D. buzzatii. Furthermore, within a species, both sexes have been caught in the same proportions. Finally, females of Leptopilina boulardi were caught almost exclusively in the second sample. All these results are presented in Figure 1. The 95% confidence intervals of the observed proportions exhibited in this figure show that intraspecific differences observed between the two samples are statistically significant for each species.

These preliminary observations lead to the conclusion that to get an accurate sample, it is necessary to collect all individuals living in the fruit cavity. All truncated samples would give a biased representation of a population.


In a previous work about sexual isolation between D. melanogaster females and D. simulans males, we found a positive correlation between the speed at which females reached sexual maturity and the frequency of hybridization (Carracedo & Casares 1985). By following suggestions made then, we intend to prove now whether or not these two traits are related in some way to the females’ level of receptivity. For this purpose we have chosen 8 isofemale lines of D. melanogaster which had previously shown different hybridization frequencies with D. simulans (Carracedo & Casares 1985), designated as M1, M2 ... M8; these lines were rechecked for hybridization with a single simulans line using the same method. The hybridization percentages calculated as the number of melanogaster females out of fifty that hybridized with simulans males were: 76, 42, 32, 28, 24, 18, 12 and 8%, for the M1 to M8 lines, respectively.

The maturity speed of melanogaster females was measured as follows. Five pairs of virgin flies from the same line, aged two hours, were kept in a vial for 30 hr. Then, females were individually placed in small vials with some food. Mating, which was inferred from the presence of larvae in the vials, was taken as evidence of female sexual maturity. The arc-sine transformed percentages of the number of fertile females out of five constituted the basic measure of the females’ maturity speed. Ten replicates per line were carried out.

The level of the females’ receptivity was measured by the time they needed to accept a standard melanogaster male. In order to make maturity speed and receptivity measurements independent factors, it was necessary to use sexually mature females. For this, we used 3-day-old virgin flies, the age at which females from the 8 lines had proven to be completely mature (Carracedo et al., submitted). The 8 lines were tested with two different lines of melanogaster males, the M1 and M4 lines. Females of each line were placed individually in a vial with two males from the same line. No anaesthesia was used. The basic measure was the time elapsed till the onset of copulation. Since a pilot experiment showed that over 99% of the females from these lines mated within a 15-min period, unmated females were discarded after this time. Each male x female combination was replicated 45 times giving a total of 8 x 2 x 45 females to be scored. The measurements were taken at 21.5°C between 9-11 a.m.

Table 1 shows the result of an analysis of variance for the time to copulation values in log.of secs., in which females and males were the sources of variation. Significant differences were only found between females. Because of this, the two male values were pooled, and the mean values of each melanogaster female line were calculated. These, after being retransformed to minutes, appear in Table 2 together with the values of the females’ maturity speed.

Table 1. Analysis of variance of the female’s “time to copulation” (in log of secs.) for 8 lines of D. melanogaster females and two lines of D. melanogaster males.

<table>
<thead>
<tr>
<th>sources of variation</th>
<th>df</th>
<th>MS</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>males</td>
<td>1</td>
<td>0.185</td>
<td>3.5</td>
</tr>
<tr>
<td>females</td>
<td>7</td>
<td>0.989</td>
<td>18.9*</td>
</tr>
<tr>
<td>males x females</td>
<td>7</td>
<td>0.070</td>
<td>1.3</td>
</tr>
<tr>
<td>error</td>
<td>704</td>
<td>0.052</td>
<td></td>
</tr>
</tbody>
</table>

* P<0.001

A negative correlation between maturity speed (in arc-sine percentages) and receptivity (in log of secs.) was found (r=-0.82; P<0.05) which means that the sexually mature melanogaster females that needed less time to mate, were those showing faster sexual maturity.

Our results not only show a close relationship between two important fitness components in Drosophila, sexual maturity and mating time, but also show connection between these homospecific traits and the hybridization frequency of melanogaster females and simulans males in the laboratory. As noted before, (Carracedo & Casares 1986), the success of such hybridization is mostly explained by the speed at which melanogaster females reach sexual maturity. Now, going a step further in the matter, our results suggest that hybridization is primarily determined by the level of melanogaster female receptivity; the greater this level, the greater the mating probability with simulans males. At least at the intrapopulational level, this suggestion does not support the existence of a species-specific discrimination key accounting for sexual isolation between these sibling species.

In this note we present an advance of some observations made about pupation behaviour in Drosophila. All the tests started with 75 newly hatched larvae seeded in a vial (18 x 200 mm) with 6 ml of standard baker’s yeast food. Development was at 21°C and under 12:12 h L:D cycles. Two lines of D.melanogaster (M1 and M2) and one line of D.simulans (S1) were used in Test 1, with 10 replications per line. When all larvae had pupated in the vials, the pupae were classified according to their pupation height. Three groups per vial, named high, intermediate and low pupation height, were made for D.melanogaster, approximately the same number of pupae in each; two groups, high and low, were made for D.simulans. The sex of the adults emerging from the pupae of each group was recorded, and the data, after pooling the replicates, appear in Table 1 for each line. Contingency chi-squares with 1 or 2 df, testing for sex differences between groups, were significantly different from zero. There were more males at the highest sites of the vials and more females at the lowest; that is, males pupated, on average, higher than females, for both species.

Test 2 was carried out with the M2 line of D.melanogaster and two new lines, S2 and S3, of D.simulans, with 11, 12 and 13 replicates, respectively, for each line. In each vial we recorded both the time at which larvae pupated and the height they attained. The pupae were classified, according to the duration of larval development (egg-to-pupa development time), as coming from early, intermediate or late pupating larvae, and the mean pupation height was calculated for each group. Thus, the relation between larval development time and larval pupation height was analyzed.

Table 1. Sex of the adults emerging from pupae located at the high, intermediate and low pupation sites in the culture vials. The chi-squares test for sex differences in pupation height.

<table>
<thead>
<tr>
<th>Line</th>
<th>High pupae</th>
<th>Intermediate pupae</th>
<th>Low pupae</th>
<th>X²</th>
</tr>
</thead>
<tbody>
<tr>
<td>M1</td>
<td>67</td>
<td>95</td>
<td>124</td>
<td>136</td>
</tr>
<tr>
<td>M2</td>
<td>93</td>
<td>126</td>
<td>115</td>
<td>115</td>
</tr>
<tr>
<td>S1</td>
<td>87</td>
<td>121</td>
<td>--</td>
<td>--</td>
</tr>
</tbody>
</table>

*** P < 0.001

Table 2. Top: analyses of variance of the pupation height values shown by larvae pupating early, intermediate or late (3 groups) in the culture vials. Bottom: mean values of pupation height for each group and for each line. All the F-values were significant with P<0.001.

<table>
<thead>
<tr>
<th>Source of variation</th>
<th>Line M2</th>
<th>Line M3</th>
<th>Line S3</th>
<th>Line S4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Between groups</td>
<td>2</td>
<td>455.6</td>
<td>2</td>
<td>4301.8</td>
</tr>
<tr>
<td>Error</td>
<td>30</td>
<td>29.2</td>
<td>33</td>
<td>60.7</td>
</tr>
<tr>
<td>Means of pupation height:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early</td>
<td>61.7</td>
<td>59.6</td>
<td>42.2</td>
<td></td>
</tr>
<tr>
<td>Intermediate</td>
<td>58.6</td>
<td>32.8</td>
<td>29.2</td>
<td></td>
</tr>
<tr>
<td>Late</td>
<td>49.4</td>
<td>20.4</td>
<td>26.7</td>
<td></td>
</tr>
</tbody>
</table>

Sex differences in pupation height were first reported by Bauer (1984). Later, however, Bauer & Sokolowski (1985) did not find any sex difference, despite their use of the same material and method. Our results show that the male larvae of D.melanogaster and D.simulans pupate higher than females do. Also, a higher pupation site occurs for larvae with shorter development. These two conclusions might be interrelated in view of the general shorter development time for male larva than for female (see ref. in Ashburner & Thompson 1978). We realize that these facts could have important repercussions and be responsible for some of the failures reported on experiments selecting for pupation height (Mensua 1967; Ringo & Wood 1983).


The ventral surface of third instar larvae of Drosophila simulans and D. melanogaster shows thin, short, black spiculae or denticles, grouped in several rows forming dentine belts (Fig. 1). These belts appear at the boundaries of consecutive larval segments and because of this are referred to as thoracic or abdominal belts. The denticles display different size, shape and orientation. They help the larvae to move through the food which might be related with some described larval behaviours connected with food acquisition. (1)

Figure 1. Ventral view of a third instar Drosophila simulans larva showing dentine belts.

Figure 2. Dentine belts on the third thoracic (a) and first abdominal (b) segments of a Drosophila simulans larva. Bar represents 300 μ.
We have analyzed the number of denticles on the third thoracic and first abdominal segments, two segments with different denticle distribution patterns (Fig. 2). Eight isofemale lines of *D. simulans* from the same population, four of them kept for two years in standard food (medium S, with 12% agar) and the other four in a thicker one (medium H, with 15% agar), and one strain of *D. melanogaster* reared in medium S were the biological material. Ten larvae from every *D. simulans* line and twenty-nine from *D. melanogaster* were fixed in Farmer's fluid for 6 hr and subsequently stored in 70% ethanol. Larvae were dorsally placed on a slide with a drop of lactic acid (20%), and a cover slide applied without pressure. The third thoracic and the first abdominal segments were photographed under a microscope. From each photograph we registered the number of denticles of the third thoracic (in a 200 x 200 \( \mu m^2 \) area) or first abdominal (in a 300 x 300 \( \mu m^2 \) area) segment in *D. simulans*. In *D. melanogaster* both segments of the same larva were counted.

In Table 1 the mean values of the number of denticles found in the *D. simulans* lines (L1 to L8) are given. Within each food class, the differences between some pairs of lines were checked by Student's t tests. All differences were significant except for L3 and L4 lines.

The mean values for the number of denticles corresponding to *D. melanogaster* are shown in Table 2. These values were contrasted with those of the *D. simulans* lines developed in medium S, and the differences were clearly significant for both larval segments. The number of denticles was higher for *D. simulans* than for *D. melanogaster*, which is in accordance with Agnew's results. (2) Following this author the above signifies that *D. simulans* might have some advantage over *D. melanogaster* when eating hard foods.

The differences found between the *D. simulans* isofemale lines and between both species suggest the existence of an extensive phenotypic variability for this character. The number of denticles could be in connection with the degradation of natural food and therefore with the fitness of Drosophila populations. At the present we are engaged in a wider investigation on the effect that genes and environment have on this character.


**Chandrashekaran, S. and R.P. Sharma.** Indian Agricultural Research Institute, New Delhi, India. A new dominant temperature sensitive lethal induced on the SM5 balancer In(2LR) al^2Cy itY cn^2 sp^2 chromosome.

Dominant temperature sensitive (DTS) lethals are useful in crossing schemes where a large number of homozygous mutagenised lines are to be established for screening purposes. It is all the more advantageous if the DTS lethal is on a balancer chromosome, so that non-DTS recombinant-escapers are avoided. DTS lethals on the second chromosome balancers CyO and Pm were reported by Falke & Wright (1970) and third chromosome balancers with DTS lethals were constructed by Marsh (1978).

Reported here is a new dominant temperature sensitive lethal induced on the SM5 balancer. The DTS lethal was induced by feeding 0.03 M Ethyl Methane sulphonate in 2% sucrose to 2 day old SM5/+ males. The mating and screening scheme is shown in Fig. 1. The vials where no Curly winged progeny hatched at 28°C were maintained after restesting, by isolating SM5/+ females and male flies from the 19° vial.

Of the 3,293 cross II vials set up, 3,100 were scored at 28°C and 8 putative DTS lethals isolated. Seven were found to be leaky when reared in larger populations producing 2-10% Curly escapers.
Figure 1. Scheme for isolating DTS lethals on the SM5 balancer.

Cross I Canton-S ♀♂ x In(2LR) SM5 a1Cy Ycn2sp2/+ (Fed with 0.03 M EMS) (mass mated in 250 ml bottles)

Cross II Canton-S ♀♂ x SM5*/+ single σ (pair mated in vials)

Vial 1 Vial 2 Flies from vial 1 transferred to fresh vial and reared at 19±2°C
d (Fed with 0.03 M EMS)

SCREEN VIALS: Select vials which produce SM5-Curly flies only at 19° and not at 28°.

Table 1. Lethality at the embryonal, larval and pupal phases in F1 individuals from a CS ♀ x SM5 448/+ mating at 19° and 28°C.

<table>
<thead>
<tr>
<th>Growth temperature</th>
<th>Number of un-</th>
<th>% Embryonal hatched</th>
<th>Expected Pupa seen</th>
<th>% larval lethality</th>
<th>Adults</th>
<th>% pupal lethality</th>
</tr>
</thead>
<tbody>
<tr>
<td>19°C</td>
<td>1014</td>
<td>174</td>
<td>17.15</td>
<td>840</td>
<td>540</td>
<td>35.71</td>
</tr>
<tr>
<td>28°C</td>
<td>1167</td>
<td>720</td>
<td>61.69</td>
<td>447</td>
<td>153</td>
<td>65.77</td>
</tr>
</tbody>
</table>

Table 2. Lethality at the embryonal, larval and pupal stages of individuals from a reciprocal mating of SM5 DTS/+ and +/+ flies at 25°C.

<table>
<thead>
<tr>
<th>Female parent</th>
<th>Male parent</th>
<th>Growth temperature</th>
<th>Total progeny</th>
<th>Cy Progeny</th>
<th>Cy+ Progeny</th>
<th>Proportion of Cy progeny*</th>
</tr>
</thead>
<tbody>
<tr>
<td>SM5 DTS/+</td>
<td>+/+</td>
<td>19°C</td>
<td>484</td>
<td>128</td>
<td>128</td>
<td>108</td>
</tr>
<tr>
<td>SM5 DTS/+</td>
<td>+/+</td>
<td>25°C</td>
<td>685</td>
<td>141</td>
<td>126</td>
<td>224</td>
</tr>
<tr>
<td>SM5 DTS/+</td>
<td>+/+</td>
<td>28°C</td>
<td>438</td>
<td>0</td>
<td>0</td>
<td>220</td>
</tr>
<tr>
<td>+/+</td>
<td>SM5 DTS/+</td>
<td>19°C</td>
<td>280</td>
<td>26</td>
<td>18</td>
<td>154</td>
</tr>
<tr>
<td>+/+</td>
<td>SM5 DTS/+</td>
<td>25°C</td>
<td>523</td>
<td>10</td>
<td>18</td>
<td>270</td>
</tr>
<tr>
<td>+/+</td>
<td>SM5 DTS/+</td>
<td>28°C</td>
<td>462</td>
<td>0</td>
<td>0</td>
<td>226</td>
</tr>
</tbody>
</table>

* Number of Cy flies divided by total number of flies.

Table 3. Lethality at the embryonal, larval and pupal stages of individuals from a reciprocal mating of SM5 DTS/+ and +/+ flies at 25°C.

<table>
<thead>
<tr>
<th>Female parent</th>
<th>Male parent</th>
<th>Number of un-</th>
<th>% Embryonal hatched</th>
<th>Expected Larvae</th>
<th>Pupa seen</th>
<th>% larval lethality</th>
<th>Adults</th>
<th>% pupal lethality</th>
</tr>
</thead>
<tbody>
<tr>
<td>+/+</td>
<td>SM5 DTS/+</td>
<td>1008</td>
<td>284</td>
<td>28.17</td>
<td>724</td>
<td>550</td>
<td>24.03</td>
<td>0</td>
</tr>
<tr>
<td>SM5 DTS/+</td>
<td>+/+</td>
<td>932</td>
<td>142</td>
<td>15.20</td>
<td>790</td>
<td>748</td>
<td>5.30</td>
<td>239</td>
</tr>
</tbody>
</table>

However, it is evident that the non-DTS individuals were also surviving poorly at both growth temperatures, the survival frequency of Cy+ being 0.4 at 19° and 0.09 at 28°C (calculated as observed Cy+ individuals divided by expected Cy+ individuals).

To further assess the viability of the DTS carrying SM5 flies and non-DTS flies, two way crosses with SM5 DTS 448/+ either as the female or male parent with Canton S as the other parent were made. Twenty pairs of flies were mated for 5 days each in 250 ml bottles at each temperature, transferred for another 5 days to fresh bottles and then discarded. Progeny from each set of bottles were counted and sexed. Data presented in Table 2.

The recovery of SM5 DTS/+ flies is 3.3 times more at 19°C and 7.4 times more at 25°C when the female parent is SM5 DTS/+ than when the female parent is +/+.

The data shows that the survival frequency of DTS 448 SM5/+ flies at 25°C is raised from 0 to 0.4 when the female is SM5 DTS/+ instead of being Canton S. The DTS 448 gene appears to be a pupal lethal since nearly 50% of the individuals are pupal lethal. However, the lethal effect can be rescued when the DTS 448+ individuals are derived from a DTS 448/+ female rather than from a wild type female. In this respect DTS 448 behaves as a recessive since it is resuable prior to fertilization by the DTS 448/+ gene product that may perdure in the egg cytoplasm. The rescue is not however possible at the non-permissive temperature probably because the mutant product is malformed beyond rescue.

Until now, we have analyzed more than 40 French natural populations of Drosophila melanogaster and it appears necessary to describe the different alleles found commonly at the Acph (acid phosphatase; 3 - 101.4), Adh (alcohol dehydrogenase; 2 - 50.1), Est-C (esterase - C; 3 - 49), Est-6 (esterase - 6; 3 - 36.8) α-Gpdh (α-glycerophosphate dehydrogenase; 2 - 20.5) and Pgm (phosphoglucomutase; 3 - 43.4) loci. The technique used was horizontal starch-gel electrophoresis.

**Gel preparation.** The kind of starch used was Connaught (Toronto) hydrolysed starch. A 12.5% solution of starch in gel buffer (volum: 500 ml) was heated to near boiling point. The starch solution was degassed and poured in a plexiglass mold 27 x 16 x 1.5 cm. The gel was covered with saran-wrap.

**Gel and electrode buffers.** The buffer system used was Poulik’s (1957); gel buffer: 76 mM TRIS and 5 mM citric acid, pH 8.65; electrode buffer: 300 mM boric acid and 60 mM NaOH, pH 8.1.

**Sample preparation.** Single flies were ground in small wells in a plastic block with 30 μl of gel buffer or distilled water. This crude homogenate was absorbed with a piece of filter paper (Whatman nr3) 1.2 x 0.2 cm. The paper pieces containing the samples were inserted along a slot in the gel made about 2 cm from the edge. The gel was placed horizontally over two buffer trays with electrodes. Two bridges in Cofram paper were used to establish contact between the gel and the electrode buffer. This set-up was connected to a rectifier (Apelex 400 mA, 300V) and placed in a refrigerated room at 4°C. The time of run was approximately 2H30 under 260 volts and 175 mA per gel. After the run was completed, the four ends of the gel were cut off. The rest was divided into two parts. Each part was sliced horizontally into six 2.5 mm thick slices with the help of a taut wire and 2.5 mm thick guides. The top slices were discarded since surface effects made them unusable. The slices of each part were used for six different enzyme assays.

**Enzyme assays** (values are given for one slice). Acph: the stain used was
50 mg Na-α-naphthyl acid phosphate, 25 mg Fast Garnet GBC salt in 75 ml 0.125 M acetate buffer pH 5.0. Adh and α-Gpdh were stained on the same slice of gel as follows: 4 ml EDTA 0.1 M, 125 mg α-glycerophosphate, 0.25 ml ethanol, 20.6 mg MTT, 3.75 mg PMS in 62.5 ml 0.05 M TRIS-HCL buffer, pH 8.5. Est-C: 1.25 ml 2% α-naphthyl-acetate solution (1:1 water: acetone was used as solvent), 75 mg Fast Blue RR salt, 8.25 ml propanol in 67.5 ml 0.1 M phosphate buffer pH 6.0. Est-6: 75 mg Fast Red TR salt, 3.75 ml propanol, 1.25 ml 2% α-naphthyl-acetate solution, 1.5 ml 2% β-naphthyl-acetate solution (1:1 water: acetone was used as solvent) in 75 ml 0.1 M phosphate buffer pH 6.0. Pgm: 66.5 mg glucose-1-phosphate (disodium salt), 7.5 ml MgCl₂ 0.1 M, 3.25 mg NADP, 4 units glucose-6-phosphate dehydrogenase, 25 mg MTT, 3.75 mg PMS in 62.5 ml 0.1 M TRIS-HCl buffer pH 8.0.

**Figure 3.** Alleles A, B, C and E at the Pgm locus in the French populations of *Drosophila melanogaster.*

**Alleles found at the different loci.** In the case of Acph, Adh and Est-C, two alleles F (Fast) and S (Slow) are commonly present in the French populations and have been previously described (Acph: McIntyre 1966; Adh: Johnson & Denniston 1964; Est-C: Beckman & Johnson 1964). For esterase-6, six alleles are present: the two common F and S which produce, respectively, the Fast (migrating rapidly towards the anode) and the Slow (migrating more slowly towards the anode) variant, two alleles (F₁ and F₂) which give allozymes migrating more rapidly than the Fast variant, and two other (S₁ and S₂) which give allozymes migrating more slowly than the Slow variant (Fig. 1). At the α-Gpdh locus, three alleles are present: the two common F and S and a rare UF (ultra fast) which produces a variant migrating more rapidly than the Fast one (Fig. 2). For Pgm, according to the nomenclature of Trippa et al. (1977), four alleles are encountered in the French populations: A and B corresponding, respectively, to the Fast and Slow variants, C and E producing, respectively, variants which migrate more rapidly than Fast or more slowly than Slow (Fig. 3).

Note: enzyme substrates and dyes are Sigma reagents.

I obtained XO males of *D. simulans* as rare segregants from a Y-autosomal translocation stock provided by E.H. Grell. This stock, T(Y;2)3,+/net b, contains homozygous net, black (II) females and wild-type males, the latter heterozygous for a net b second chromosome and a wild-type chromosome attached to a Y. About one in five hundred males in this stock are net black XO males produced by nondisjunction.

Twenty-one four-day-old XO males from this stock were dissected in Ringer's solution and their testes examined under phase-contrast microscopy. Primary spermatocytes were compared to those present in XO *D. melanogaster* males. As reported previously, the *D. melanogaster* males had crystal-containing spermatocytes. None of the 21 *D. simulans* XO males, however, showed crystal formation. All of these had full-sized testes which contained sperm in various stages of development, but none had motile sperm. The lack of crystals in these males supports Livak's suggestion that Y-linked copies of the presumptive Stellate locus regulate the copies on the X, and militates against the idea that crystal formation in spermatocytes is an inherent property of XO males.

**Acknowledgements:** I thank Ken Livak for the idea of looking at XO *D. simulans* males. This work was partially supported by grants from the National Science Foundation (BSR-83-18558) and the National Institutes of Health (32221).


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As previous investigations have shown (Sparrow 1978), a common feature among melanotic tumor mutants of *Drosophila melanogaster* is the participation of larval blood cells in tumor formation. According to Rizki & Rizki (1980a), plasmatocytes, with their morphological variants podo- and lamellocytes, and the crystal cells (Fig. 1) are the cellular components of the larval hemolymph. They are produced in the lymph gland.

The hemolymph of tumorous 3rd instar larvae is characterized by a large number of lamellocytes owing to their precocious transformation from plasmatocytes (Sparrow 1978). The recently isolated melanotic tumor mutant, tu-pb, differs from the other tumor mutants since melanized masses appear only in the adult stage and are confined to the head. tu-pb tumors develop at 23.5°C (penetrance: $\approx 35\%$; $\approx 10\%$) but not at 18°C (Di Pasquale Paladino & Cavolina 1983). In order to elucidate the mechanism of tumor formation in tu-pb, we examined the patterns of blood cell types in the hemolymph of late 3rd instar male and female larvae (120 h aged at 23.5°C) of tu-pb and Oregon-R (as control) stocks.

---

**Figure 1.** Crystal cell from the larval hemolymph.

**Figure 2.** Heat treated larvae: (a) Oregon-R, (b) tu-pb.
Table 1. Mean cell numbers in hemolymph of Oregon-R and tu-pb 120h aged larvae.

<table>
<thead>
<tr>
<th>Cell types</th>
<th>Male Oregon-R</th>
<th>Female Oregon-R</th>
<th>Male tu-pb</th>
<th>Female tu-pb</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasmatocytes</td>
<td>3618.6±967.38</td>
<td>3122.2±300.54</td>
<td>3552.6±797.86</td>
<td>3505.4±624.91</td>
</tr>
<tr>
<td>Lamellocytes</td>
<td>3310.8±306.96</td>
<td>1880.0±384.97</td>
<td>2843.4±526.75</td>
<td>2591.6±305.70</td>
</tr>
<tr>
<td>Crystal cells</td>
<td>237.4±8.42</td>
<td>48.6±10.95</td>
<td>210.4±29.50</td>
<td>55.8±3.43</td>
</tr>
<tr>
<td>Cell totals</td>
<td>7166.8±1202.03</td>
<td>5050.8±340.77</td>
<td>6606.4±1393.75</td>
<td>6152.8±846.3</td>
</tr>
</tbody>
</table>

Table 2. Hot water (70°C, 15') treated larvae.

<table>
<thead>
<tr>
<th>breeding temperature</th>
<th>stock</th>
<th>N</th>
<th>no mela-nization</th>
<th>spread mela-nized cells</th>
<th>melanized lymph gland</th>
</tr>
</thead>
<tbody>
<tr>
<td>23.5°C 96h</td>
<td>♀ ♀ Oregon-R</td>
<td>103</td>
<td>1.9</td>
<td>98.1</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>♂ ♀ Oregon-R</td>
<td>104</td>
<td>2.9</td>
<td>97.1</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>♂ ♂ tu-pb</td>
<td>111</td>
<td>98.2</td>
<td>0.0</td>
<td>1.8</td>
</tr>
<tr>
<td></td>
<td>♂ ♀ tu-pb</td>
<td>97</td>
<td>97.9</td>
<td>0.0</td>
<td>2.1</td>
</tr>
<tr>
<td>23.5°C 120h</td>
<td>♀ ♀ Oregon-R</td>
<td>117</td>
<td>0.0</td>
<td>93.2</td>
<td>6.8</td>
</tr>
<tr>
<td></td>
<td>♂ ♀ Oregon-R</td>
<td>109</td>
<td>0.0</td>
<td>95.4</td>
<td>4.6</td>
</tr>
<tr>
<td></td>
<td>♂ ♂ tu-pb</td>
<td>141</td>
<td>75.2</td>
<td>0.0</td>
<td>24.8</td>
</tr>
<tr>
<td></td>
<td>♂ ♀ tu-pb</td>
<td>127</td>
<td>70.1</td>
<td>0.0</td>
<td>29.9</td>
</tr>
<tr>
<td>18.0°C 240h</td>
<td>♀ ♀ Oregon-R</td>
<td>125</td>
<td>30.4</td>
<td>69.6</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>♂ ♀ Oregon-R</td>
<td>100</td>
<td>16.0</td>
<td>84.0</td>
<td>0.0</td>
</tr>
<tr>
<td></td>
<td>♂ ♂ tu-pb</td>
<td>113</td>
<td>88.5</td>
<td>0.0</td>
<td>11.5</td>
</tr>
<tr>
<td></td>
<td>♂ ♀ tu-pb</td>
<td>105</td>
<td>78.1</td>
<td>0.0</td>
<td>21.9</td>
</tr>
</tbody>
</table>

Counts of cell types were made according to the classification into plasmatocytes, lamellocytes and crystal cells (Rizki 1978). Results and statistical analysis (t-Student) are presented in Table 1. Comparing tu-pb with Oregon-R, blood cells mean number patterns appear similar except for the crystal cells which are numerically fewer in tu-pb.

Therefore, we can conclude that tu-pb does not hold a larger amount of lamellocytes in its hemolymph. This fact seems to be in agreement with the later tumor manifestation in this stock. On the other hand, the reduced number of crystal cells might be significant. As Rizki has recently pointed out (Rizki 1980b), melanization of crystal cells can be induced by treatment of larvae with hot water. By using this functional criterion, we compared the distribution of heat-induced black cells in tu-pb and Oregon-R larvae. Results are presented in Table 2. In consequence of heating the two stocks manifest a different phenotype: blackened crystal cells appear spread in the hemocoel of Oregon-R larvae, while in tu-pb they are maximally confined into the lymph gland (Fig. 2).

The blackening of lymph gland is more frequent in late 3rd instar larvae (120 h aged) than in the younger ones (96 h aged) and in larvae reared at 18°C; the trait appears also to be recessive because etherozygous tu-pb/Oregon-R larvae manifest the Oregon phenotype. According to Shrestha & Gateff (1982), mature crystal cells are very rare in the first hematopoietic lobes and absent in the second lobes; therefore, their relative abundance in tu-pb lymph gland can be considered an unusual condition.

These findings could suggest that the releasing of mature crystal cells by the hematopoietic organs may be abnormal in tu-pb; they could also account for the low number of free crystal cells found in the hemolymph of the tumorous stock.

The hypothesis of a possible relation between the above mentioned facts and the manifestation of the melanotic tumor trait must be confirmed by further investigations. In this regard, we take into consideration the possibility of following the fate of the crystal cells in tu-pb making them marked with the Black-cells (Bc) mutant. In Bc/+ individuals naturally occurring pigmented crystal cells can be seen in larval, pupal and adult stages. We are now trying to combine tu-pb with Bc.

**Table 1. Genetic mapping of Dia-3 in D.virilis.**

<table>
<thead>
<tr>
<th>Phenotype of visible markers</th>
<th>Number</th>
<th>Dia-3h</th>
<th>Dia-3l</th>
</tr>
</thead>
<tbody>
<tr>
<td>cv +</td>
<td>368</td>
<td>223</td>
<td>145</td>
</tr>
<tr>
<td>+ w</td>
<td>365</td>
<td>224</td>
<td>141</td>
</tr>
</tbody>
</table>

Electrophoretic study of 20 wild and laboratory stocks of D.virilis revealed that stock No. 112 is homozygous for an allele determining very low activity of diaphorase-3, i.e., Dia-3l (low activity) (Ralchev et al. 1986). Crosses of this stock with stock No. 147 possessing high activity of diaphorase-3, i.e., Dia-3h (high activity) showed sex-linked inheritance of this character (Fig. 1). For precise localization of Dia-3 gene on the X-chromosome genetic map stock No. 127 carrying mutations cv - crossveinless, 1-25.0, and w - white, 1-105.0 (Alexander 1976) was used. The following crosses were carried out:

\[
P \,(127)\, \text{cv w (Dia-3h)} \times \sigma (112)\, \text{++ (Dia-3l)}
\]

\[
F_1 \quad \text{cv w (Dia-3h)} \times \sigma \text{cv w (Dia-3h)}
\]

In F2 progeny 733 male flies, recombinant by visible markers, were selected and analysed electrophoretically to determine the phenotype of diaphorase-3. The results of this analysis (Table 1) showed that Dia-3 gene is located at 65.3 ± 2.3 position.

**References:**


Dosage compensation has been studied in 2 species: D. virilis and D. americana and in their hybrid (D. americana ♀ x D. virilis ♂). The chromosome complement of D. virilis is similar to that of the ancestral species with 5 acrocentric chromosomes (elements A to E) and a dot chromosome (element F). In D. americana fusion between B element and the X chromosome of the ancestral species has occurred. These two closely related species belonging to the same group (virilis) form hybrids (Patterson & Stone 1952) (Fig. 1).

We wanted to know whether D. americana (comparatively a younger species evolutionarily which evolved from D. virilis by X-B fusion) and D. virilis have the property of dosage compensation and whether the hybrid shows any change from the parental species or not.

Results reveal that the total transcripitive activity of the X chromosome is equivalent in both female and male D. virilis (X/A♀/X/A♂ = 1.06). Sitewise transcriptional analysis shows that all the 15 subsegments of the distal part of the X chromosome (1A-11C) are dosage compensated (Fig. 2).

Same type of results have also been obtained for D. americana which showed a similar activity in male and female as revealed from the total grain count analysis (X/A♀/X/A♂ = 1.08). Sitewise analysis of the distal part (15 subsegments from 1A-10B) shows no dose-dependent sites. The slight deviation in the female/male ratio from 1 for the sites 4A and 4B was found to be non-significant (P>0.7) (Fig. 3).

In the hybrid nuclei also, we fail to observe any significant deviation in the X chromosome/autosome ratio in male and female (female/male ratio = 1.27) (Fig. 4).

Figure 1. Morphology of hybrid (D. americana X D. virilis) male polytene chromosome.

Figure 2. Histogram showing the transcripitive activity of different X-chromosomal segments (1A-11C) of male and female D. virilis.
Figure 3. Histogram showing the transcriptive activity of different X-chromosomal segments (1A-1OB) of male and female D.americana.

Figure 4. Photograph showing the $^3$H-uridine incorporation in the hybrid (D.americana ♀ X D.virilis ♂) female X-chromosome.

We, therefore, conclude that the individual set of chromosomes maintain the property of compensation in the hybrid cytoplasm.

Acknowledgement: This work has been supported by a UGC scheme (F.23/167-83 (SR-II) of 17.1.84) to A.K.D.G.

Farmer, J.L. and D.J. Fairbanks, Brigham Young University, Provo, Utah USNA. Interaction of the bw and w loci in D. melanogaster.

The allele wco2 is suppressed by a gene on 2R (Farmer 1977). The experiments reported here show that the allele which was formerly called Su(wco2) is bw+ and the Su*(wco2) is a previously undescribed allele which we have named bw6. This is the second reported interaction of these loci. Rifenburgh & Sutfin (1935) reported that w;bw flies were "slight light buff or cream", definitely darker than w alone. There have been reports of interaction between other eye color loci (e.g., Nolte 1958; Reedy & Cavalier 1971).

Materials and methods. Stocks were obtained from Mid-America Drosophila Stock Center, Bowling Green State University; the Division of Biology, California Institute of Technology; J.A. Beadmore, University College of Swansea; and P.T. Ives, Amherst College. Most of the w and bw alleles which were tested were in multiply marked stocks. Only the relevant alleles have been shown for simplicity. A full listing of genotypes is available on request. Wildtype flies from various populations were obtained from R.S. Singh, McMaster University.

Flies were maintained on a cornmeal-molasses-yeast-agar medium or on Carolina Instant Drosophila Medium.

The drosoppterin content of single heads was measured using the acidic ethanol extraction method described previously (Farmer 1977).

Results and discussion. If bw and Su(wco2) are allelic, crossovers between them should be rare. In an attempt to find crossovers between bw and Su(wco2), females of genotype wco2 v; px bw sp/+ + + were crossed with px bw sp/+ + + males. Male progeny which had crossed over described previously (Farmer 1977). The experiments reported here show that the allele which was formerly called Su(wco2) is bw+ and the Su*(wco2) is a previously undescribed allele which we have named bw6. This is the second reported interaction of these loci. Rifenburgh & Sutfin (1935) reported that w;bw flies were "slight light buff or cream", definitely darker than w alone. There have been reports of interaction between other eye color loci (e.g., Nolte 1958; Reedy & Cavalier 1971).

The nature of this cross made it possible to exclude the possibility that Su(wco2) might be outside the px-sp interval.

Combining the data from the two crosses yielded the following map distances: px-bw = 4.65; bw-sp = 2.72; maximum bw-Su(wco2) = 0.016 at the 95% confidence level.

The results of the mapping crosses were consistent with the hypothesis that Su(wco2) was identical with bw+. To test the hypothesis further, a large number of bw alleles were tested to see if any of them might be linked to a gene which could suppress wco2 in a male fly whose genotype was wco2 v;bw+/Su(wco2), where bw* is any one of the bw alleles tested. None of the chromosomes carrying bw alleles which were tested acted as a suppressor of wco2. The bw alleles which were tested were: bw2b, bw4, bw5, bw381, bw45A, bw49h, bw72, bw80, bw81, bwD, and bwV1.

We concluded that Su(wco2) was identical with bw* since no crossing over was detected between them and since all chromosomes which carry bw* suppress wco2 while all chromosomes which carry a non-wild-type allele of bw do not suppress. The allele which was formerly called Su*(wco2) must therefore be a mutant allele of bw. We have named it bw6.

No interaction between bw6 and alleles of w other than wco2 had been noticed in previous experiments. We looked for enhancement of a number of w alleles by bw6 in flies whose genotypes were w*bw6. Flies whose genotypes were w*bw6/bw6 served as controls. The symbol w* represents any one of the w alleles tested. None of the w alleles which were tested was enhanced by bw6. The w alleles which were tested were wa, wa3, wbf, wbf2, wbf3, wbfw, wcf, wch, wco, wcol, we, wec3, wh, wmr7Ah1, wsr, wsp, and wt.

The major properties of the bw6 and wco2 alleles can be summarized as follows. Flies which were homozgyous or hemizygous wco2 and homozgyous bw6 or heterozygous bw6 with any other mutant bw allele had very dark eyes due to the near absence of drosoppterins. Flies which were homozgyous bw6 and which had at least one w+ allele were wildtype. Flies which were heterozygous bw6 with any other recessive bw allele and which had at least one w+ allele were wildtype in appearance, although the drosoppterin level of some genotypes was above or below the control value (Table 1). The one exception to this was wco2/w+ bw3/bw6 whose eyes were noticeably darker than controls (Table 2). The bw5 allele interacts with wco2 also, as shown by the very low drosoppterin concentration in wco2 Y;bw5/bw+ flies. Flies which were homozgyous or hemizygous wco2 and homozgyous or heterozygous bw+ were wildtype in appearance, although the heterozygotes had a lower concentration of drosoppterins, perhaps due to gene dosage effect.
Table 1. Drosopterin content of wco2 v/+ + flies heterozygous for bw6 and other bw alleles.

<table>
<thead>
<tr>
<th>genotype</th>
<th>mean absorbance at 480 nm</th>
<th>standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>bw+/bw6</td>
<td>0.050b,e</td>
<td>0.005</td>
</tr>
<tr>
<td>bw/bw6</td>
<td>0.041</td>
<td>0.008</td>
</tr>
<tr>
<td>bw4/bw6</td>
<td>0.072d</td>
<td>0.009</td>
</tr>
<tr>
<td>bw5/bw6</td>
<td>0.030d,e</td>
<td>0.009</td>
</tr>
<tr>
<td>bw38j/bw4</td>
<td>0.048</td>
<td>0.007</td>
</tr>
<tr>
<td>bw4s/bw6</td>
<td>0.043c</td>
<td>0.002</td>
</tr>
<tr>
<td>bw49j/bw6</td>
<td>0.064c</td>
<td>0.010</td>
</tr>
</tbody>
</table>

a N is the number of measurements.
b control value used for t tests.
c 0.01 < p < 0.05 using t test.
d p < 0.005 using t test.
e also in Table 2.

Table 2. Drosopterin content of flies containing the bw5 and/or bw6 alleles.

<table>
<thead>
<tr>
<th>genotype</th>
<th>mean absorbance at 480 nm</th>
<th>standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td>wco2/w+;bw+bw+</td>
<td>0.065</td>
<td>0.010</td>
</tr>
<tr>
<td>wco2/w+;bwbw6</td>
<td>0.055</td>
<td>0.009</td>
</tr>
<tr>
<td>wco2/w+;bw5bw6</td>
<td>0.050b</td>
<td>0.009</td>
</tr>
<tr>
<td>wco2/w+;bw5bw6</td>
<td>0.030b</td>
<td>0.006</td>
</tr>
<tr>
<td>w+/w+;bw5bw6</td>
<td>0.062</td>
<td>0.008</td>
</tr>
<tr>
<td>wco2 Y;bw+/bw+</td>
<td>0.045</td>
<td>0.001</td>
</tr>
<tr>
<td>wco2 Y;bw5bw+</td>
<td>0.014</td>
<td>0.000</td>
</tr>
<tr>
<td>wco2 Y;bw5bw+</td>
<td>0.037c</td>
<td>0.004</td>
</tr>
<tr>
<td>w+ Y;bw+/bw+</td>
<td>0.061</td>
<td>0.008</td>
</tr>
<tr>
<td>w+ Y;bw5bw+</td>
<td>0.058</td>
<td>0.006</td>
</tr>
<tr>
<td>w+ Y;bw6bw+</td>
<td>0.063</td>
<td>0.006</td>
</tr>
<tr>
<td>w+ Y;bw5bw6</td>
<td>0.057</td>
<td>0.014</td>
</tr>
</tbody>
</table>

a N is the number of measurements.
b also in Table 1. c from Farmer 1977.

(Farmer 1977). Flies which were heterozygous wco2 with any other recessive w allele and homozygous bw6 had very dark eyes due to the nearly absence of drosopterins, while flies which had the same genotype except for being heterozygous bw6/bw+ had a nearly wildtype eye color, the drosopterins being reduced, perhaps due to gene dosage effect (Farmer 1977; this paper).

If one considers all of the possible combinations of wco2, bw6, and their wildtype alleles, only flies which were homozygous or hemizygous wco2 and homozygous bw6 had the mutant (dark brown) eye color. Thus w+ suppressed homozygous bw6, and bw+ suppressed homozygous or hemizygous wco2. It is possible to construct at least two kinds of models which would account for this kind of interaction.

In the first model, it is presumed that gene products of w+ and bw+ join together to form a multimer which is the active protein. The alleles wco2 and bw6 are assumed to be mutated at sites which prevent formation of the quaternary structure between their gene products. However, it is assumed that the mutations allow weak but effective bonding between the gene products of wco2 and bw+ or between the gene products of w+ and bw6.

The second model assumes that the w and bw loci each code for at least two activities. This model requires that one of the activities coded for by the bw locus, deficient in bw6, is identical to one of the activities coded for by the w locus, deficient in wco2. Then only the double mutant wco2,bw6 would produce the phenotype which is caused by the loss of the common activity. This model predicts that most, if not all, previously described w and bw mutants would be deficient for one of the unique activities.

Like bw6, the bw4 allele is wildtype when homozygous. However, unlike bw6, it produces dark eyes when heterozygous with the bw5 allele. It is possible that bw6 and bw5 represent an unusual class of mutations at the bw locus.

A preliminary survey of wildtype stocks from various parts of the world indicates that some populations carry an allele which acts like bw6 (fails to suppress hemizygous wco2) (Fairbanks, unpubl. data). No frequency data are available. Several laboratory stocks also carry a non-suppressing allele (Farmer 1977). Although the suppressing allele was found to be more common in both laboratory and wildtype socks, the non-suppressing allele was not rare.

Acknowledgements: We thank M.M. Green for helpful suggestions and C.A. Istock for providing laboratory facilities to DJF. This research was supported by a research grant from Brigham Young University. DJF was supported by an undergraduate research fellowship from the Honors Program of Brigham Young University.


Although Drosophila melanogaster females elicit vigorous courtship from Drosophila affinis males, the flies never mate (S.P. McRobert & L. Tompkins, in prep.). This observation suggested the possibility that the external genitalia of D.affinis males or D.melanogaster females might be structurally dissimilar to the corresponding parts of conspecific individuals, making intromission physically impossible. Accordingly, I examined the genitalia of males and females from these two species by scanning electron microscopy.

D.melanogaster males and females were collected from a Canton-S laboratory stock, while D.affinis males and females were collected from isofemale lines that had recently been established from local populations. Newly eclosed males and females were separated by sex and aged for five days, then anesthetized with CO₂ and affixed to pin mounts with SEM silver paint. The flies were then sputter coated with gold, examined with a Phillips 501B scanning electron microscope, and photographed.

There are two vaginal plates on the ventral surface of the eighth segment of a Drosophila female. Each of the plates bears a row of thorn bristles, between which is located the vulva (Hodgkin & Bryant 1978). A comparison of the external genitalia of a D.affinis female (Fig. 1) and a D.melanogaster female (Fig. 2) reveals no conspicuous differences.

The penis of a Drosophila male is located between the posterior lobes of the genital arch, which is surrounded by the two halves of the eighth tergite (Hodgkin & Bryant 1978). A comparison of a D.affinis male (Fig. 3) and a D.melanogaster male (Fig. 4) reveals that the males' eighth tergites and genital arches are similar, but the D.affinis male's penis is much larger and is shaped differently from that of the D.melanogaster male. Ten males of each species were examined and these differences were consistently observed.

If living D.affinis males also have larger penises than living D.melanogaster males, this anatomical difference may be at least partially responsible for the fact that D.melanogaster females do not copulate with D.affinis males.

**Figure 1.** Posterior view of the external genitalia of a D.affinis female. P, vaginal plates; TB, thorn bristles; V, vulva; A, anus. Magnified 160X.

**Figure 2.** Posterior view of the external genitalia of a D.melanogaster female. See Fig. 1 for abbreviations. Magnified 160X.
Figure 3. Posterior view of the external genitalia of a D. affinis male. T, eighth tergite; GA, genital arch; P, penis. Magnified 160X.

Figure 4. Posterior view of the external genitalia of a D. melanogaster male. See Fig. 3 for abbreviations. Magnified 160X.

Acknowledgements: I am grateful to Joel Sheffield for help with the SEM. I also thank Laurie Tompkins, Lisa Napolitano and Scott McRobert for advice, unpublished material, and Drosophila stocks. This research was supported by a Biomedical Research Support Grant from Temple University.


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Consequences on the serotonin level in Drosophila melanogaster of an alimentary chronic pharmaceutical treatment.

We have previously described (Marenco et al. 1984) the fact that serotonin (5-HT) depletion only applied during larval instars, reduced learning performance of the seven-day imago. The same results were obtained when the depletion was carried out during the first week of the imago life. To obtain a noticeable effect on the learning performance, the usual amount of the inhibitor of serotonin synthesis: para-chlorophenylalanine (p-CPA), added to the synthetic chemically defined nutritive medium (S), has been carried out from 0.3g/l to 0.6g/l (Vaysse et al. 1983). Biochemical control of 5-HT level was performed on these flies treated for a week, the question is whether a seven day treatment gives the best depletion.

The present note concerns 5-HT amounts in imagos seven days old before the chemical analysis. They are fed on the S medium for seven, six, ... one or zero days; and, in each case, the seven day period is completed by a breeding on S medium added with a pharmacological agent:
- S + p-CPA medium with 0.6g/l of p-CPA,
- S + 5-HTP medium with 0.6g/l of 5-hydroxytryptophan (5-HTP), which is the immediate precursor of serotonin.

A rapid procedure of analysis by reversed phase high-performance liquid chromatography is applied to the estimation of serotonin from the flies’s extracts, and allows us to follow, sample by sample, the consequences of a p-CPA or 5-HTP over-dose in the nutritive medium. The chromatographic system
Figure 1. Level of 5-HT.

The chromatographic method consists of LiChrosorb RP8 as stationary phase and methanol-sodium acetate gradient as mobile phase. Samples of twenty male flies are collected with an insect aspirator, weighed then frozen in liquid nitrogen at about 4 p.m. (accounting for the 5-HT circadian variation). Each one is lyophilysed for 24 hours, crushed in a micropotter crusher, then suspended in 1 ml HCl 0.1 N and centrifuged for two minutes at 6,000 g. The supernatant is injected directly into the chromatograph (two injections) and the eluted fraction is detected at 275 nm.

Figure 1 gives the results found, expressed as the decimal logarithm of the concentration of 5-HT (median values of the 5 samples used in each case), related to the concentration measured for the control group: for the experimental flies treated (a) by p-CPA; (b) by 5-HTP.

For the p-CPA group, 5-HT level falls from the first day of treatment; this tendency holds until the 5th day, when the strongest effect is seen. Following this, the control level is restored in this experimental group on the 7th day.

On the other hand, the 5-HTP level is markedly increased in the 5-HTP group on the third to the fifth day. If the treatment lasts longer, the curve returns towards the control level. In both cases, after a period of latency (one or two days), the level of the experimental groups differs from the control one, and in spite of the chronicity of the treatment returns towards the initial level. These results strongly suggest the existence of regulatory processes. It could be for each of these pharmacological agents: either an elimination under native form in the excreta; or an increase of the degradation processes by the normal metabonic paths of the 5-HT; or else an enzymatic induction (parametabolic for 5-HTP and xenometabolic for p-CPA).


In a selection experiment, involving four different lines (H1, H2, L1 and L2), for abdominal bristle number performed in D. subobscura (Martínez-Sebastian & Mensua 1986), different responses were noticed in one low selection line (L2). The differences included sudden increase of variability, disturbed abdominal bristle pattern and disturbed sclerotization, which in D. melanogaster have been shown to be due to the bobbed locus (Frankham, Briscoe & Nurthen 1978).
Table 1. Means (X ± e) and coefficients of variability (C.V. ± eC.V.) with standard errors in each high (H1 and H2) and low (L1 and L2) selection lines, in the last generation of selection (A) and three years after the selection was discontinued (B).

<table>
<thead>
<tr>
<th></th>
<th>X ± e</th>
<th>C.V. ± eC.V.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>B</td>
<td>A-B</td>
</tr>
<tr>
<td>H1</td>
<td>74.73±0.34</td>
<td>-0.74</td>
</tr>
<tr>
<td>H2</td>
<td>72.08±0.36</td>
<td>-2.27</td>
</tr>
<tr>
<td>L1</td>
<td>12.47±0.15</td>
<td>5.03</td>
</tr>
<tr>
<td>L2</td>
<td>14.72±0.30</td>
<td>4.54</td>
</tr>
</tbody>
</table>

When selection finished, the four lines were maintained under laboratory conditions without further selection for a three years period (30 generations approx.). At this time, the number of abdominal bristles was measured in each of the four lines.

Table 1 shows the means and coefficients of variability of abdominal bristle number of each line in the last generation of selection (A) and in each line three years after the selection was discontinued (B).

In the high selection lines (H1 and H2), the means as well as the coefficients of variability remained practically constant. These results support the hypothesis that abdominal bristle number is a neutral character for natural selection (Clayton, Morris & Robertson 1957).

However, the increase of the means occurring in the low selection lines (L1 and L2) suggests the existence of a minimum threshold for abdominal bristle number. The coefficient of variability decreased in both low selection lines, with the loss of variability being more extensive in the L2 than in the L1 line.

The behaviour of L2 line seems to suggest that part of the response to selection may be due, in this line, to the accumulation of bobbed alleles. During the three years period following selection the L2 line might then have tended to eliminate bobbed alleles in order to reach the normal level of rRNA. Consequently, the L2 line will have increased the number of abdominal bristles.

It was not possible to map the putative bobbed allele because there is no marker strain in D. subobscura. Work is in progress to demonstrate the presence of bobbed alleles using in situ hybridization with probes of D. melanogaster.


We have canalized through selection the phenotype "2 anterior dorsocentral extra bristles", in two isofemale lines of D. melanogaster: ADC-4 and ADC-7 (Figure 1). The lines do not segregate individuals in classes higher than the selected class 2; their individuals do not have bristles in other positions than the selected one.

Ten males of ADC-7 were mated with 10 females of ADC-4, and vice versa, in two replicas. We analysed 100 individuals of each sex in the progeny. The two replicas of each crossing were homogeneous; by this reason, they were pooled.

After these crossings, we studied the influence of each chromosome in both lines obtained canalization. Experimental design is as the Kearsey & Kojima one (1967). Three independent replicas of each chromosomal combination have been obtained. They were homogeneous; by this reason they have been grouped. We counted 60 males and 60 females in each replica.

Eight homozygotic chromosomal combinations of the three main chromosomes were obtained. Each chromosome may be of the selected line (ADC-4 or ADC-7), or of the Oregon strain (normal phenotype, without extra bristles). We show female data; males behave in the same way.

The obtained distributions of both lines are very similar; but we have selected different genetic factors for extra bristle in each one: crossing both lines, the number of individuals with extra bristles decrease in F1 and F2 (Figure 2). There is not any significant difference among reciprocal crossings. We do not find any discanalization, since there are no individuals with more than 2 extra bristles, and extra bristles are only in the selected position.

Figure 1. Canalized phenotype in ADC-4 and ADC-7.
Table 1. Extra bristles distribution (in % of individuals) of each chromosomal combination, in ADC-4 and ADC-7.

<table>
<thead>
<tr>
<th>Classes of Extra Bristles</th>
<th>ADC-4</th>
<th>ADC-7</th>
</tr>
</thead>
<tbody>
<tr>
<td>BBB</td>
<td>8.9</td>
<td>15.6</td>
</tr>
<tr>
<td>BBA</td>
<td>94.4</td>
<td>92.2</td>
</tr>
<tr>
<td>BAB</td>
<td>55.0</td>
<td>47.8</td>
</tr>
<tr>
<td>ABB</td>
<td>93.9</td>
<td>98.3</td>
</tr>
<tr>
<td>ABA</td>
<td>98.3</td>
<td>97.4</td>
</tr>
<tr>
<td>AAA</td>
<td>79.4</td>
<td>85.6</td>
</tr>
<tr>
<td>Class 0</td>
<td>97.8</td>
<td>92.8</td>
</tr>
<tr>
<td>Class 1</td>
<td>15.6</td>
<td>85.0</td>
</tr>
<tr>
<td>Class 2</td>
<td>47.8</td>
<td>89.4</td>
</tr>
<tr>
<td>Class &gt; 2</td>
<td>95.0</td>
<td>95.0</td>
</tr>
<tr>
<td>Class 2, Sel.p.</td>
<td>33.3</td>
<td>7.2</td>
</tr>
<tr>
<td>Unsel.p.</td>
<td>7.2</td>
<td>8.3</td>
</tr>
<tr>
<td>Classes &gt; 2</td>
<td>3.3</td>
<td></td>
</tr>
</tbody>
</table>

Numerical STP: AAA ABA BBA BAA AAB ABB BAB BBB

Positional STP: BAB AAB BBA BAA ABB BBB AAA

B = line chromosome; A = Oregon chromosome; sel.p. = selected phenotype; unsel.p. = unselected phenotype.

All the lines are carrying in all their chromosomes some genetic factors for bristle number increase (Table 1). The numerical STP analysis shows that the chromosome 3 is the main one responsible for this increase in both lines. AAB do not differ significantly from BBA: chromosome 3 increases the bristle number as chromosome 1 and chromosome 2 do. Chromosome 1 in ADC-4 and chromosome 2 in ADC-7 are the next chromosomes in importance to increase the number of bristles. There are strong interactions between the three chromosomes. These data confirm the results of other authors (i.e., Rubio & Albornoz 1982).

Some chromosomal combinations are discanalized: they have a high proportion of individuals in classes > 2, and they have two extra bristles in unselected positions (Table 1). Positional STP analysis shows that chromosome 2 is essential for the canalization keeping in ADC-4. Chromosome 1 is the main one responsible for this canalization in ADC-7, followed by chromosome 2. Obtained canalization of these lines have a polygenic or multifactorial basis.

As other authors say (Fraser 1967; Rendel 1976), lines have two systems determining the obtained phenotype. One system increases bristle number; another system maintains the canalization. First system is mainly in chromosome 3; the second one is mainly in chromosomes 1 and/or 2. Nevertheless, we cannot find any large effect gene in the two systems, as above cited authors do.

The character extra bristles is studied in this work. These extra bristles, both dorsocentral and scutellar, are normally canalized in nature for the phenotype that we can see in Figure 1. The hidden variation for this character is easily revealed using isofemale lines (Parsons & Hosgood 1967); some individuals with extra bristles appear in most of the lines.

Eleven Asturian natural populations have been examined, analysing the percent of individuals of each line which have extra bristles. Isofemale lines are jointed in three classes: Class I, well canalized (less than 5% of individuals have extra bristles); class II, (5-20% of individuals with extra bristles); and class III (more than 20%).

Likewise the isofemale lines are classified by their positional definition of extra bristles, as DC (more than 75% of extra bristles are dorsocentral), SC (more than 75% of extra bristles are scutellar), and DC/SC (extra bristles in both dorsocentral and scutellar positions).

The sites of capture are in the map of Figure 2. Four populations were captured along the river valley Paramo-Nalon (Proaza, Trubia, Sandiche and Soto del Barco); four along the Asturian coast (Aviles, Somio, Villaviciosa, Celorio); and three are central populations, captured near Oviedo city (Los Areneros A, Los Areneros B, La Granda). In each line we have examined 60 individuals of each sex.
**Table 1.** Percent of isofemale lines of each extra bristles positional class. NL = number of lines in classes II and III. Lines of class I are not positionally classifiables. $X^2_{20}$ df(global)=23.710 N.S.

<table>
<thead>
<tr>
<th>Populations</th>
<th>Class DC</th>
<th>Class SC</th>
<th>Class DC/SC</th>
<th>NL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proaza</td>
<td>69.0</td>
<td>10.3</td>
<td>20.7</td>
<td>29</td>
</tr>
<tr>
<td>Trubia</td>
<td>73.7</td>
<td>17.8</td>
<td>10.5</td>
<td>19</td>
</tr>
<tr>
<td>Sandiche</td>
<td>70.2</td>
<td>14.9</td>
<td>14.9</td>
<td>47</td>
</tr>
<tr>
<td>Soto del Barco</td>
<td>71.4</td>
<td>25.0</td>
<td>3.6</td>
<td>28</td>
</tr>
<tr>
<td>Aviles</td>
<td>68.2</td>
<td>20.4</td>
<td>11.4</td>
<td>44</td>
</tr>
<tr>
<td>Somlo</td>
<td>66.7</td>
<td>23.1</td>
<td>10.2</td>
<td>39</td>
</tr>
<tr>
<td>villaviciosa</td>
<td>80.0</td>
<td>15.0</td>
<td>5.0</td>
<td>20</td>
</tr>
<tr>
<td>Celorio</td>
<td>80.0</td>
<td>3.3</td>
<td>16.7</td>
<td>30</td>
</tr>
<tr>
<td>Los Areneros A</td>
<td>74.0</td>
<td>5.2</td>
<td>20.8</td>
<td>77</td>
</tr>
<tr>
<td>Los Areneros B</td>
<td>71.4</td>
<td>12.1</td>
<td>16.5</td>
<td>91</td>
</tr>
<tr>
<td>La Granda</td>
<td>77.8</td>
<td>13.3</td>
<td>8.9</td>
<td>45</td>
</tr>
</tbody>
</table>

$X^2_{20}$ df(global) = 70.712***

**Table 2.** Percent of isofemale lines of each extra bristles numerical class. NT = number of lines.

<table>
<thead>
<tr>
<th>Populations</th>
<th>Class I</th>
<th>Class II</th>
<th>Class III</th>
<th>NT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proaza</td>
<td>35.5</td>
<td>51.1</td>
<td>13.1</td>
<td>45</td>
</tr>
<tr>
<td>Trubia</td>
<td>26.9</td>
<td>57.7</td>
<td>15.4</td>
<td>26</td>
</tr>
<tr>
<td>Sandiche</td>
<td>36.5</td>
<td>47.3</td>
<td>16.2</td>
<td>74</td>
</tr>
<tr>
<td>Soto del Barco</td>
<td>42.9</td>
<td>40.8</td>
<td>16.3</td>
<td>49</td>
</tr>
<tr>
<td>Aviles</td>
<td>36.2</td>
<td>50.7</td>
<td>13.1</td>
<td>69</td>
</tr>
<tr>
<td>Somlo</td>
<td>61.0</td>
<td>37.0</td>
<td>2.0</td>
<td>100</td>
</tr>
<tr>
<td>Villaviciosa</td>
<td>58.3</td>
<td>35.4</td>
<td>6.3</td>
<td>48</td>
</tr>
<tr>
<td>Celorio</td>
<td>28.6</td>
<td>66.7</td>
<td>4.7</td>
<td>42</td>
</tr>
<tr>
<td>Los Areneros A</td>
<td>29.3</td>
<td>54.1</td>
<td>16.5</td>
<td>109</td>
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<tr>
<td>Los Areneros B</td>
<td>29.4</td>
<td>62.8</td>
<td>7.8</td>
<td>129</td>
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<tr>
<td>La Granda</td>
<td>55.9</td>
<td>40.2</td>
<td>3.9</td>
<td>102</td>
</tr>
</tbody>
</table>

$X^2_{6}$ df=2.521 N.S.

Populations are not different for the extra bristles position (Table 1). Most of the lines have dorsocentral extra bristles; some lines of each population have only scutellar, and some of them, both scutellar and dorsocentral extra bristles. All populations have some variation in their isofemale lines for the extra bristles position, but this variation is the same in every populations, or at least there are not statistical differences.

Populations are significantly different for the extra bristle number of each line (Table 2). Classifying populations by their geographic situation, we find that both coastal and central ones differ significantly to their isofemale line variation; while, the valley populations are homogeneous, because their isofemale lines have the same distribution ($X^2 = 2.52$, N.S.).

There is a different rate of migration between populations, greater in the valley than in the coast or in the central belt of Asturias, because the dispersal along the river valley is favoured by the predominant N-S wind direction, and by the human agricultural transport, through main roads and railway. By this reason, we think that the main factor responsible for the homogeneity of the valley populations is the migration.

Catalase activity was measured in the 3rd instar larvae of trisomy-3L, Compound-3L and Oregon-R+ stocks following the method used by Lubinsky & Bewley (1979). A unit of catalase activity is defined as 1 micromole $H_2O_2$ decomposed/minute assuming the molar extinction coefficient of 70.67 at 230 nm. Total protein was determined following the method of Lowry et al. (1951).

We have observed that the specific activity of catalase enzyme is almost same in trisomy for 3L, Compound-3L and Oregon-R+ stocks of *Drosophila melanogaster* (see Table 1).

From this observation, it is clear that the catalase shows a compensatory level of activity in whole arm trisomic condition with the control Oregon-R+ and compound-3L stocks. The specific activity of catalase in segmental aneuploids is 1.5 times higher than that of euploid level (Lubinsky & Bewley 1979). Whether duplication of the whole left arm of third chromosome is necessary for catalase gene to be compensated or not, awaits further investigation.

**Table 1.** Catalase activity in Trisomy-3L, Compound-3L and Oregon-R+ stocks of *Drosophila melanogaster*.

<table>
<thead>
<tr>
<th>Enzyme</th>
<th>T/D</th>
<th>T/C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trisomy-3L(T) Oregon-R+(D)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Mean ± S.E.</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Catalase</td>
<td>2.889 ± 0.226</td>
<td>2.440 ± 0.194</td>
</tr>
</tbody>
</table>

*Specific activity: micromole $H_2O_2$ decomposed/minute/ug of total protein.

DNA replication in polytene chromosomes of different species of *Drosophila* has been examined autoradiographically by various groups of workers. In *Drosophila* polytene chromosomes a general pattern of synchronous replication among different sites of the same chromosome and between different chromosomes has been reported (Plaut et al. 1966), with the exception of the X-chromosome of the male. However, asynchronous replication is known to exist in *Drosophila* polytene nuclei, viz., under replication of the nucleolus organizer (Laird 1973) and faster replication of male-X (Lakhotia & Mukherjee 1970). In *Drosophila pseudoobscura* there is an asynchrony in replication between the two autosomal arms (2nd and 3rd) though both the autosomes are in diploid condition. Nothing precisely is known about the regulation of these cases of asynchronous replication, although much work has been done on the significance of such asynchrony (Lakhotia & Mukherjee 1970; Laird 1973). In *Drosophila melanogaster* polytene chromosome, in which DNA replication has been studied in detail (Lakhotia & Mukherjee 1970), no asynchrony between nonhomologous chromosomes has been reported.

The aim of the present investigation on replication is to find out:
1. Whether asynchrony of replication exists between trisomic autosomal arms (2L and 3L) and other disomic chromosomal arms (2R and X) where the trisomic arm contains 1.5 times more DNA in comparison with non-duplicated arms of the same nuclei?
2. If there is an asynchrony of replication, when does this occur, i.e., in the initial or terminal phase of the S?

For 3H-thymidine autoradiography, salivary glands were dissected out from late third instar larvae of trisomy 2L and trisomy 3L stocks and processed according to Lakhotia & Mukherjee (1970). To find out whether extra DNA content in the trisomic arms have any influence on its replication behaviour, we

**Table 1a.** 3H-thymidine incorporation. Frequency of labelled sites on 2L arm, 2R and female-X chromosome in trisomy for 2L stock.

<table>
<thead>
<tr>
<th>Chromosomal arms</th>
<th>&gt;50%</th>
<th>&gt;25%</th>
<th>&lt;25%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trisomy-2L</td>
<td>0.36</td>
<td>0.22</td>
<td>0.42</td>
</tr>
<tr>
<td>Disomic-2R</td>
<td>0.32</td>
<td>0.24</td>
<td>0.44</td>
</tr>
<tr>
<td>Female-X</td>
<td>0.29</td>
<td>0.23</td>
<td>0.48</td>
</tr>
</tbody>
</table>

**Table 1b.** Frequency of labelled sites on T3L arm, 2R, and X chromosome (female) in trisomy for 3L stock.

<table>
<thead>
<tr>
<th>Chromosomal arms</th>
<th>&gt;50%</th>
<th>&gt;25%</th>
<th>&lt;25%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trisomy-3L</td>
<td>0.42</td>
<td>0.25</td>
<td>0.32</td>
</tr>
<tr>
<td>Disomic-2R</td>
<td>0.32</td>
<td>0.24</td>
<td>0.44</td>
</tr>
<tr>
<td>Female-X</td>
<td>0.33</td>
<td>0.24</td>
<td>0.43</td>
</tr>
</tbody>
</table>

have studied the patterns of replication (DD-1C-2C-3C-3D = 2D-1D), frequency of labelled sites on trisomic arms and compared with the reference segments of 2R (60A-56F) and female-X (1A-11A).

Data reveal that 36% of the total studied (69 subsegments) sites in T2L have labelling frequency greater than 50%, similarly 32% of the total studied sites (24 subsegments, 56F-60A) in 2R have labelling frequency greater than 50% and the labelling frequency greater than 25% and less than 25% have been presented in Table 1a. In trisomy for 3L nuclei, 42% of the total studied (68 subsegments 61A-80A) sites have labelling frequency greater than 50% whereas 33% of the sites in 2R have labelling frequency greater than 50% (Table 1b).

So there is no significant difference in the frequency of labelled sites in trisomic autosomal arms (2L and 3L) and disomic non-homologous chromosomal arms. Besides, it is evident from our observation that no asynchrony exists in initial or in late phase of replication pattern between the T2L/T3L and disomic arms.

References:


In Drosophila, as also in various tissues of many eukaryote systems, the nucleolus manifests as a round or oval structure organized by a specific region of the genome commonly known as nucleolar organizer. In many systems, as in Drosophila larval salivary glands, the nucleolus is a very prominent body often attached to the chromocentric region through a bundle of thread-like structure. The thread-like connections seem to penetrate into the nucleolar mass and form different patterns of distribution in the nucleolar matrix.

Different morphological conformations of nucleolar chromatin threads in the form of granules, blocks and threads in the nucleoli of Drosophila hydei have been reported earlier (Ghosh & Mukherjee 1982). Such configurations are not unique to the species of D. hydei but are found in other species as well (Barr & Plaut 1966a, b; Rodman 1968, 1969; Ghosh 1984).

In the present investigation an attempt has been made to characterize the nature of the nucleolar chromatin thread (NCT) of D. melanogaster. For this purpose, aceto-carmine aceto-orcein staining, Feulgen staining, Acridine orange staining techniques as well as ³H-thymidine and ³H-uridine autoradiography have been performed.

Figure 1. Photomicrographs showing four different types of nucleolar chromatin threads (NCTs) of D. melanogaster. Types 1, 2, 3 and 4 are represented by a, b, c and d, respectively.
Observations reveal that different configurations of NCTs as are found in *D. hydei* are also present in *D. melanogaster* (Fig. 1). However, the type 4 of *D. melanogaster* is not so much complex as they are in *D. hydei*. All the 4 NCT types of *D. melanogaster* are Feulgen positive (Fig. 2) which remain embedded in the Feulgen negative nucleolar matrix. Fluorescence staining with Acridine orange (AO) also reveals that all 4 NCT types exhibit bright yellow fluorescence while the nucleolar matrix shows red fluorescence (Fig. 3).

Data on $^3$H-uridine and $^3$H-thymidine labelling reveal that all the 4 different NCT types incorporate $^3$H-uridine and $^3$H-TdR. Therefore, all the 4 NCT types of *D. melanogaster* are transcriptionally and replicationally active. But the detailed analysis of the data suggest that the labelling density of $^3$H-uridine and that of $^3$H-thymidine as evident from the actual count of silver grains are not similar for 4 NCT types. This finding suggests that the 4 NCT types may be under different state of activity and the filamentous/branched network thread like nucleolar chromatin structures (that types 3 and 4) are metabolically most active.

The nucleolus of larval salivary glands of Drosophila contains nucleolar chromatin structure (NCT) which have various conformations. It has been suggested that the various conformations are functionally related (Ghosh & Mukherjee 1982). The functional relationship of these conformations have been further examined in different mutational forms in which the number of nucleolar genes is different.

The different stocks of Drosophila hydei used for this purpose were y m ch/Y (attached-X/Y), bb vg P, KOMFP 290/2 and KOMTKS 697/16. The number of nucleolar organizers and rDNA cistrons present in these stocks have been reported by Kunz & Schafer (1976) from their hybridization experiments.

The purpose of this investigation has been to find out whether the configurations of different chromatin thread morphotypes reported earlier by Ghosh & Mukherjee (1982) and their activity in terms of 3H-uridine incorporation are related to the number and/or distribution of the nucleolar organizers or rDNA cistrons.

Results on the observations on the morphology of the nucleolar chromatin types in different mutant strains reveal that except y m ch/Y, all the 4 types of NCTs are present in the other three mutant stocks of D. hydei. Results are presented in Table 1. The females of y m ch/Y, KOMFP 290/2 and KOMTKS 697/16 possess attached-X/Y chromosomes (Kunz & Schafer 1976). As reported the X-chromosomal NORs are absent and only 2 Y-chromosomal NORs are present in the females of these strains. On the other hand, in the mutant strain, bb vg p, certain parts of NOR are deleted in both X and Y chromosomes.

Data on the morphology of NCT types in these mutant strains reveal that there is no direct correlation between the more active NCT types (i.e., types 3 and 4) and the nucleolar organizer(s) (Table 1). It is also evident from the data that the presence of the Y chromosome appears to induce considerably more activity than its absence as the frequencies of NCT types 3 and 4 are higher in both males and females of the mutant stocks possessing Y chromosome compared to the females of bb vg p and wild type where Y chromosome is absent. It is also interesting to note that the frequencies of NCT types 3 and 4 are comparatively lower where the intact Y is absent.

Data on the mean 3H-uridine grain number in the nucleoli with different NCT types for the four different mutant stocks and wild type are presented in Table 2. Data show that there is a clear one-to-one relationship in the mean grain number in the corresponding NCT types of the two sexes, regardless of the dosage of the NOR(s).

When the different intensity classes are plotted histographically, the results seem to indicate that in both male and female of all the genotypes, the frequency of low labelling intensity classes decreases from type 2 to 4 and that of the high labelling intensity classes which appear first in the type 2, increases from type 2 to 4. The pattern of labelling intensity appears to be similar in the two sexes. The pattern of distribution of the intensity classes presented in Figures 1a and b also indicate a differential labelling profile specific for a particular genotype.

It is evident from the results that the source of rDNA cistrons (X chromosomal or Y chromosomal) seems to be important for the frequency of different types of NCT. For example, it has been observed that in the presence of Y chromosome when the number of NOR is equal the differences in the intensity classes of labelling among them is minimum. This observation seems to reiterate the significance of the quality rather than the quantity of the rDNA cistrons. This interpretation is supported by Endow & Glover (1979) who proposed that in the diploid cells one of the two sets of NOR might be metabolically active. This is

### Table 1. Nucleolar chromatin thread (NCT) types and their frequencies (in %) in wild type and different mutant stocks of Drosophila hydei.

<table>
<thead>
<tr>
<th>Stocks</th>
<th>No. of NOR(s)</th>
<th>NCTs in males</th>
<th>NCTs in females</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>1 2 3 4</td>
<td>1 2 3 4</td>
</tr>
<tr>
<td>Wild type</td>
<td>m f</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3 2</td>
<td>(21.68) 242</td>
<td>(21.68)</td>
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<tr>
<td></td>
<td></td>
<td>(58.29) 45</td>
<td>(58.29)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(16.84) 38</td>
<td>(16.84)</td>
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<tr>
<td></td>
<td></td>
<td>(9.15)</td>
<td>(9.15)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>144 129</td>
<td>144 129</td>
</tr>
<tr>
<td></td>
<td></td>
<td>151 22</td>
<td>151 22</td>
</tr>
<tr>
<td>Y m ch/Y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(attached-X/Y)</td>
<td>3 2</td>
<td>--</td>
<td>--</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(43.75) 224</td>
<td>(43.75) 224</td>
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<td></td>
<td></td>
<td>(52.15) 267</td>
<td>(52.15) 267</td>
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<td></td>
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<td>-- 215</td>
<td>-- 215</td>
</tr>
<tr>
<td></td>
<td></td>
<td>330 9</td>
<td>330 9</td>
</tr>
<tr>
<td>bb vg P</td>
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<td>(30.23) 117</td>
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<tr>
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<td>(32.29) 125</td>
<td>(32.29) 125</td>
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<td>(36.43) 141</td>
<td>(36.43) 141</td>
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<td>(3.14)</td>
<td>(3.14)</td>
</tr>
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<td>KOMFP 290/2</td>
<td>2 1</td>
<td>(17.16) 76</td>
<td>(17.16) 76</td>
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<td></td>
<td></td>
<td>(143) 134</td>
<td>(143) 134</td>
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<td></td>
<td></td>
<td>(213) 11</td>
<td>(213) 11</td>
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<td>(2.48)</td>
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<td>(4.53)</td>
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<tr>
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<td></td>
<td>(12.55) 115</td>
<td>(12.55) 115</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(12.79) 100</td>
<td>(12.79) 100</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(12.08) 116</td>
<td>(12.08) 116</td>
</tr>
<tr>
<td>KOMTKS 697/16</td>
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<td>(12.08) 40</td>
<td>(12.08) 40</td>
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<tr>
<td></td>
<td></td>
<td>(46.53) 154</td>
<td>(46.53) 154</td>
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<td>(39.58) 131</td>
<td>(39.58) 131</td>
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<td>(12.55) 115</td>
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<td></td>
<td></td>
<td>(3.14)</td>
<td>(3.14)</td>
</tr>
</tbody>
</table>

(-) indicates some portion deleted in the Nucleolar Organizer Region (NOR). m = male, f = female.
A large number of mutations have been found in <i>Drosophila melanogaster</i> which alter the structure and/or function of the nucleolar organizer (NOR). An investigation has been made to find out the relation between the structural conformations of nucleolar chromatin thread (NCT) and their function(s). Therefore, from this observation it is suggested that the four NCT types are different forms of metabolic expression of the DNA sequences present in the rDNA cistrons.

Table 1. Nucleolar chromatin thread and their frequencies (in percent) in wild type and different mutant stocks of *Drosophila melanogaster*.

<table>
<thead>
<tr>
<th>Stocks</th>
<th>No. of NOR(s)</th>
<th>NCT types 1 and 2 (Broad Group I)</th>
<th>NCT types 3 and 4 (Broad Group II)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>m</td>
<td>f</td>
<td>m</td>
</tr>
<tr>
<td>Oregon</td>
<td>2</td>
<td>2</td>
<td>66</td>
</tr>
<tr>
<td>y sn&lt;sup&gt;3&lt;/sup&gt; bb</td>
<td>2(-)</td>
<td>2(-)</td>
<td>85</td>
</tr>
<tr>
<td>y w&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1</td>
<td>1</td>
<td>184</td>
</tr>
<tr>
<td>y</td>
<td>2</td>
<td>2</td>
<td>180</td>
</tr>
<tr>
<td>w&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2</td>
<td>2</td>
<td>144</td>
</tr>
<tr>
<td>Normal</td>
<td>3</td>
<td>3</td>
<td>240</td>
</tr>
<tr>
<td>yf:=/Y(attached-X/Y) and XY</td>
<td>2</td>
<td>3</td>
<td>132</td>
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<tr>
<td>T(X; y&lt;sup&gt;y&lt;/sup&gt; Y&lt;sup&gt;L&lt;/sup&gt;) 1la and yf:=/Y</td>
<td>3</td>
<td>3</td>
<td>291</td>
</tr>
</tbody>
</table>

(-) indicates some portion deleted in the Nucleolar organizer region (NOR). m = male, f = female.

When the distribution of the frequencies for different NCT types in mutant strains of *D. melanogaster* containing different numbers of NOR(s) is compared, it is evident that the increase in the number of NOR is not concurrent with an increase in the frequency of the NCT types 1 and 2 (Group I) in either sex. However, there appears to be a threshold level in the activity of the NOR. This is borne out by the fact that the number of NOR is slightly less than 2, e.g., in y sn<sup>3</sup> bb which has a partial deletion of NOR, the frequency of NCT types is also not changed.

From the results it seems that the number of NOR does not have any direct influence on the variation of NCT types among the wild type and different mutant strains of *D. melanogaster*. The difference in the frequency of NCT morphotypes between male and female may be due to difference either in number of rDNA cistrons in the X and Y NOR, or may be due to differential activation of the cistrons.

The above mentioned findings on the activity of the rDNA lead to suggest that all the cistrons are not functionally active in the diploid cells. As pointed out by Endow & Glover (1979), only half the number of rDNA cistrons are active in producing the rRNA in the polytene nuclei. Data presented here on the distribution of the four morphotypes in genetically different strains of *D. melanogaster* also corroborate these findings. The data show that as long as a minimum of one complete set of rDNA cistrons is present, i.e., 130 cistrons in *D. melanogaster*, the activity is maintained at a normal level. This level is maintained and does not change considerably with the increase in the dosage of the NOR or rDNA cistrons. This implies, therefore, that the ribosomal genes maintain dosage compensation in both sexes.

The wing somatic mutation and recombination test (SMART) for the detection of genotoxic activity of chemicals makes use of the two wing cell markers mwh (multiple wing hairs, 3-0.0) and flr (flare, 3-39.0). The trans-heterozygous configuration of these markers allows the registration of somatic mutations (gene mutations, deletions, etc.) and of mitotic recombination. Mitotic recombination between the proximal marker and the centromere leads to the formation of twin spots on the wings of adult flies after treatment of larvae with a given compound. Single spots may be the consequence of somatic mutation or mitotic recombination between the two markers. Depending on the time of clone induction during larval development, smaller or larger spots are produced. The size of each spot can be determined by counting the number of cells expressing the mutant phenotype. The classification of small mwh spots consisting of only one or two cells poses some problems. In our routine scoring of wings only those spots are classified as "true" mwh spots in which at least one wing cell shows three or more hairs instead of only one hair as in wild type. However, quite frequently single cells are encountered with two hairs of more or less equal size. These "false" spots—which are probably mainly due to developmental disturbances in trichome pattern formation—are not included in our scoring. The terms "true" and "false" used here refer only to the phenotype of the mwh spots.

In the course of our investigations, we discovered that the frequency of the mwh spots, especially the false ones, depends on the temperature at which the larvae and pupae are reared. Thus, an experiment was performed as follows. Two different crosses were set up: (1) Cross A: y; mwh jv females and y; Dp(1;3)sc4, flr/TM1, Mri s6d males, and (2) Cross B: mwh females and flr3/TM3, Ser males.

From each cross eggs were collected for 8 h on standard cornmeal-sugar-agar-yeast medium in two bottles at 25°C. After the egg collection period one bottle from each cross was kept at 25°C, the other one at 29.5°C. No further treatment was given to the larvae. All the eclosing flies were collected and stored in 70% ethanol. Approximately 40 wings from each series were mounted and scored under a compound microscope at 400x magnification. All the spontaneous spots were counted and classified according to the two categories true and false as explained above. The true spots were mainly small mwh single spots (1 or 2 cells) with a few larger single and twin spots. The false spots were almost exclusively single cells with two hairs. The data are given in Table 1.

<table>
<thead>
<tr>
<th>Cross</th>
<th>Progeny</th>
<th>Temp. (°C)</th>
<th>Wings (n)</th>
<th>True spots</th>
<th>False spots</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>n</td>
<td>f</td>
<td>n</td>
<td>f</td>
</tr>
<tr>
<td>A</td>
<td>mwh x</td>
<td>25</td>
<td>40</td>
<td>19</td>
<td>0.48</td>
</tr>
<tr>
<td></td>
<td>flr/TM1</td>
<td>29.5</td>
<td>40</td>
<td>98</td>
<td>2.45</td>
</tr>
<tr>
<td></td>
<td>mwh/TM1</td>
<td>25</td>
<td>36</td>
<td>10</td>
<td>0.28</td>
</tr>
<tr>
<td></td>
<td>flr3/TM3</td>
<td>29.5</td>
<td>44</td>
<td>10</td>
<td>0.23</td>
</tr>
<tr>
<td>B</td>
<td>mwh x</td>
<td>25</td>
<td>40</td>
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<td>0.25</td>
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<tr>
<td></td>
<td>flr3/TM3</td>
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<td>40</td>
<td>35</td>
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</tbody>
</table>

The frequencies of both categories of spots are increased with the higher temperature in all three genotypes studied. This shows that higher temperature affects the expression of mwh spots quite drastically. However, in general this effect is far more pronounced for the false spots as compared with the true spots. More important, the increase is most pronounced in the trans-heterozygous mwh+/+ flr progeny of cross A. These flies carry the scJ4 duplication (which contains a y+ gene) at the tip of the flr chromosome in the immediate proximity of the mwh+ locus. This is not the case for the trans-heterozygous progeny of cross B. Apparently, the scJ4 duplication has an influence on mwh expression. This is further corroborated by the fact that no such influence is seen in the inversion-heterozygous mwh/TM1 flies. Furthermore, a comparison of the two crosses shows that flies derived from cross B have much lower frequencies of false spots. Although these false spots are not included in our scoring data in standard SMART experiments, they can be quite disturbing in the screening of the wings. Therefore we now prefer to use larvae derived from cross B for our experiments. In addition, the data presented above demonstrate clearly that strict temperature control is essential in wing SMART experiments in order to obtain reproducible and quantitatively comparable results.

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Individual differences in conditionability.

Recently we presented evidence for the classical conditioning of the proboscis extension reflex based on the average performance of a group of *D. melanogaster* (Holliday & Hirsch 1984, 1986). Now we describe an experiment to measure individual differences (IDs) in conditionability.

If, as is now widely believed, the contingency between the conditioned stimulus (CS) and the unconditioned stimulus (US) is necessary for the development of a conditioned response (CR), then a reduced CS-US contingency (CS-US presentation randomized or unpaired) will retard or prevent acquisition of the CR (Rescorla 1967; Ricker, Hirsch, Holliday & Vargo 1986). Traditional experiments in the contingency framework test this hypothesis by presenting to one group of subjects a conditioning procedure with a positive contingency, and comparing the results with those of a control group that received noncontingent pairings of the CS and US. By showing that the average CR is greater in the experimental group than in the control group, it can be inferred that: (1) Conditioning has occurred, and (2) the contingency is important for the development of the CR (e.g., Holliday & Hirsch 1984, 1986). Because no individual receives both contingent and noncontingent stimuli, however, this method precludes demonstrating the effectiveness of the contingency analysis to measure conditioning in identified individuals. To measure conditioning in individuals, we presented each subject with nine trials of an automated conditioning procedure (Vargo, Holliday & Hirsch 1983; Holliday, Vargo & Hirsch 1983) followed by six trials of unpaired stimuli. The conditioning procedure consisted of a 5 second tarsal stimulation of 0.5 M NaCl (the CS) followed 0.5 seconds later by 5 seconds of tarsal stimulation (available to the proboscis for 2-3 seconds) with 0.25 M sucrose solution (the US). In addition, to discharge sucrose-induced CES (Vargo & Hirsch 1982a, b), a 5 second tarsal stimulation of distilled water (the intertrial stimulus) was presented 2 minutes 55 seconds after the US (Figure 1). For the unpaired extinction procedure, the CS and US were separated by approximately 90 seconds.

Thirty-four subjects (18 females, 16 males) were tested with this procedure. Figure 2 depicts the average CR as a function of paired and unpaired trials. The characteristic conditioning curve increases over the first nine trials (Holliday & Hirsch 1984, 1986), followed by the hypothesized decrease in responding to the unpaired CS. The evidence thus supports our interpretation that the CS-US contingency is important for conditioning to occur in this species (Holliday & Hirsch 1984, 1986). And, the procedure allows us to look at the same effect in individuals.

Individual cumulative response patterns have been classified into four categories, with individual variation within categories. Examples appear in Figure 3: (a) acquisition followed by extinction, 24%; (b) acquisition without extinction, 15%; (c) responding, but failure to show either acquisition or extinction, 34%; and not shown is (d) no response to the CS, 26%.

![Figure 1. Diagram of the stimulus schedule (note the gap between the CS and US creating approximately 0.5 s trace [where the rubber band prevents contamination between stimuli]).](image-url)
The ability reliably to identify IDs in conditionability now allows us to attempt divergent selection for high and low conditionability in *D. melanogaster*. Individuals in category (a) (above) would be classified as good conditioners, while those in category (c) would be poor or non-conditioners. It is not immediately certain why individuals in category (b) continue to respond when the CS and US are unpaired. Perhaps visual stimuli, in addition to the (NaCl) CS, serve as US predictors that are not affected by the unpaired procedure. Also, because sucrose is still present during extinction, CES may influence responding in these individuals.

It has been shown in the blow fly *Phormia regina* that breeding selectively for conditionability, using acquisition scores only to select individuals for breeding, resulted in lines divergent in fact for CS-induced sensitization, not conditioning (Ricker et al. 1986). Our ability now to distinguish between conditioned responding and that due to nonassociative excitation (e.g., between individuals in categories a and b) makes possible controls lacking in previous research.
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Islam, M.S., M.A.R. Khan, P.C. Barman and S.I. Ali. University of Rajshahi, Rajshahi, Bangladesh. Effect of copper and ferrous sulphates on offspring production in Drosophila melanogaster. Attempts have been made to understand the developmental effect of gene action on Drosophila by subjecting the eggs, larvae or pupae of this fly to various stress conditions, such as chemicals, temperature-shock, radiations of various wave-lengths, ether-shocks and so forth. The present work deals with the effect of addition of copper and ferrous sulphates to food on the offspring production in Drosophila melanogaster. The wild type D.melanogaster was used in the present investigation. The flies were reared on the cornmeal-agar-molasses food medium. Traces of Nipagin were added as mould inhibitor. The doses of copper sulphate used were 0(control), 0.05, 0.10, 0.15, 0.20 and 0.25%, and those of ferrous sulphate were 0, 0.10, 0.15, 0.20, 0.25 and 0.30%. The experiments were started with 25 pairs of flies, equally shared by virgin females and unmated males, per population jar (size 15 cm x 6 cm). The experiments were replicated six times and five times for copper sulphate and ferrous sulphate, respectively, per dose per generation. Counting of flies on the control and treated foods was continued for five successive generations. Experiments were conducted at 25±0.5°C.

The results are given in Tables 1 and 2. Both copper and ferrous sulphates reduced the number of offspring of the fly (P<0.001). It was found that with the increase of doses the offspring production decreased. Copper sulphate produced a more drastic effect on Drosophila than did ferrous sulphate. Dobzhansky & Spassky (1967) reared D.melanogaster and D.pseudoobscura on sodium chloride added food medium and found that the latter was more sensitive to the salt than the former species. Islam (1981) reported that the Curly, an autosomal dominant mutant of D.melanogaster, had an advantage over its normal competitor in such an abnormal environment. Shakoori & Butt (1980) observed that at stronger doses of thioacetamide the larva of D.melanogaster had an advantage over its normal competitor in such an abnormal environment. Shakoori & Butt (1980) observed that at stronger doses of thioacetamide the larva of Musca domestica failed to metamorphose into adults. A recent study by Shakooodi & Parveen (1983) showed that no adult emerged from the eggs of M.domestica placed in culture media containing 0.56 and 0.64% thioacetamide. These findings are similar to that observed in the present work. In the present study, it was also observed that flies reared at stronger doses of the salts showed certain morphological abnormalities, e.g., collapsed abdomen with blackened and stubby body. However, whether these characters were due to phenocopic effects or not was not investigated.

Both copper and ferrous sulphates was found to reduce the number of offspring of D.melanogaster significantly, and therefore every care should be taken against the contamination of food of D.melanogaster with these salts to avoid any shortage in the number of this fly for experimental purposes.

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| Table 1. Offspring production in D.melanogaster at different conc. of copper sulphate added to food. |
|--------------------------------------------------------|--------------------------------------------------------|
| Generations | 0.00% | 0.05% | 0.10% | 0.15% | 0.20% | 0.25% |
| 0(parental) | 50 | 50 | 50 | 50 | 50 | 50 |
| 1 | 517 | 321 | 267 | 162 | 97 | 62 |
| 2 | 892 | 394 | 265 | 153 | 81 | 52 |
| 3 | 1337 | 486 | 291 | 164 | 70 | 41 |
| 4 | 1772 | 531 | 334 | 186 | 62 | 38 |
| 5 | 1958 | 558 | 354 | 198 | 51 | 37 |

| Table 2. Offspring production in D.melanogaster at different conc. of ferrous sulphate added to food. |
|--------------------------------------------------------|--------------------------------------------------------|
| Generations | 0.00% | 0.10% | 0.15% | 0.20% | 0.25% | 0.30% |
| 0(parental) | 50 | 50 | 50 | 50 | 50 | 50 |
| 1 | 438 | 314 | 198 | 112 | 98 | 78 |
| 2 | 807 | 529 | 367 | 286 | 198 | 108 |
| 3 | 1148 | 749 | 465 | 312 | 285 | 97 |
| 4 | 1852 | 1135 | 872 | 498 | 313 | 114 |
| 5 | 2022 | 1618 | 996 | 618 | 348 | 118 |
An estimation of the "rare male advantage" can be done using the so called "Wattiaux's equation" (1964), which changes, by logarithmic transformation, the sigmoid curves of mating successes into straight lines. These straight lines, which can differ in slope or in mean elevation, can be compared by covariance analysis (Snedecor 1956). The "Wattiaux's formula" has been used, e.g., to study the competition between the mutant rucuca flies and the wild type Canton S or the blind mutant ora flies (Kaningini et al. 1986, this issue; Schuermans et al. 1986, this issue). The same formula is applied here with certain modifications. In the original formula, In(dx/N-x, In is the naperian logarithm, dx the number of individuals of a given type (e.g., wild type males) mating during this time interval (15 min), N the total number of individuals of the same type, and x the number of these individuals having already mated before the considered interval of time. Wattiaux's equation was based on the assumption that males could mate only once in the total observation time (3 hr). Present experiments seem to indicate that they had obviously mated more than once, the original formula had to be modified by us. If x (the number of individuals having copulated before the considered interval of time) becomes greater than N (the total number of these types of individuals), the denominator of the equation dx/N-x becomes negative. Consequently, it seemed preferable to introduce "10. x/N" (a mating value) in place of x. The formula thus becomes:

\[ \ln(dx/10) \frac{N}{N-x} \cdot 10/N \]

In the present experiments, we have studied the sexual activity of wild type Canton S flies when they are in the presence of mutant white-ebony flies, which are certainly less active (Grossfield 1975). The method of observation has been previously described (Elens 1957, 1958; Elens & Wattiaux 1964). A total of 30 pairs of virgin flies, 4-5 days old, were introduced together in the mating chamber, but the relative frequencies of both types were varied (for males and females simultaneously). All the observations were done at 25°C and 40-60% relative humidity, but at two different light intensities (50 and 1000 lux), and at two different hours (8 a.m. and 5 p.m.), with 10 repetitions for each relative frequency. The differences between homogamic and heterogamic mating frequencies were never significant. But significant differences were often found between the sexual activity of males or of females when they were "rare" (relative frequency = 5/30) than at other relative frequencies. Such differences were evident not only for the wild type flies but sometimes even for the mutant ones.

At a high intensity of light (1000 lux), in the morning, the wild type males were obviously more active when they were "rare" (Fig. 1, A) than at all the other relative frequencies that were tried (P<0.001). Similarly, the white-ebony males displayed their highest activity when they were rare (Fig. 1, D and E).

**Figures:** Relationships between the competing types wild Canton S and white-ebony, when their relative frequencies differ: columns, from left to right: 5+/25we; 10+/20we; 15+/15we; 20+/10we; 25+/5we. Abscissa: time in minutes. Ordinate: In(dx/10)/N - x) In is the naperian logarithm, N the total number of flies of one sex and one type, dx the number of these flies mating in the considered interval of time dt (15 min) and x the number of these flies having already mated before.

Figure 1. morning, 1000 lux.
In such conditions, they were even more active than the wild type males, which at other frequencies mated more often than the white-ebony ones (P<0.001). The activity level of the females of both types was also higher when they were "rare" (Fig. 1, F and J): at the frequency "5+/25we" the wild type females were more active at frequency "25+/5we" (P<0.001). In darkness (50 lux) the results were quite similar (Fig. 2). The white-ebony males were more active when rare (P<0.001), even more than the wild type ones (Fig. 1, E). At the relative frequencies "5+/25we", "10+/20we", "15+/15we", the wild type males activity was
always higher. A "rare female advantage" was also observed, for the white-ebony flies (Fig. 2, J) as well as for the wild type ones (Fig. 2, F). At the intermediate relative frequencies, the differences between mutant and wild type females were not significant (Fig. 2, G, H, I).

In the evening, the white-ebony males activity level was lower than in the morning, at least in light (Fig. 3). They were always much less active than the wild males (P<0.001), and when they were the rarest they did not mate at all (Fig. 3, E). In darkness, one observed the same "rare male advantage" as in the morning experiments (Fig. 4, A and E). In light and in darkness, the females behaved as they did in the morning.

Knoppien (1985) has recently excellently reviewed the whole matter of the "rare male mating advantage", first observed by Petit (1951) and as a result often called "Petit effect". Its causes are discussed, even stronger its existence has sometimes been doubted. Knoppien concludes, however, that this phenomenon is very widespread in insects, at least under laboratory conditions, and perhaps even in nature (1985). Our present observations are in good agreement with this view. Besides, thus, we consider that equations such as the so called "Wattiaux's formula" are interesting tools for visualizing the differences in activity level of the competing types.

Kalisch, W.-E., Ruhr-Universität Bochum, FR Germany. The EM band-interband pattern of SSP chromosomes in D. subobscura: Division 100A-D.

The D (Dot) chromosome of D. subobscura was observed electron microscopically (EM) using the surface-spread polytene (SSP) chromosome preparation technique (Kalisch et al. 1984, 1985a,b; Kalisch & Böhm 1985). Salivary glands were used from late 3rd instar larvae of a wild-type strain. Electron micrographs of SSP chromosomes were prepared with a final magnification of x 3,200 (Fig. 1C and D). Cytological data were calculated from eight electron micrographs and used for a computer plot (for methodological details, see Kalisch et al. 1984, 1986a,b) of an EM chromosome map (Fig. 1E). EM chromosome cytology was compared with the original chromosome map (Fig. 1A) based on LM analyses of squash preparations (Kunze-Mühl & Müller 1958) and with a single light micrograph of a chromosome squash preparation (Fig. 1B).

A total number of 27 chromosome bands was observed. This is about a 93% increase of additional bands compared with the 14 bands of the LM chromosome map (100C1 in Fig. 1A indicates a very tight doublet in the original map of Kunze-Mühl & Müller 1958). Earlier studies have already shown (Whitmore et al. 1984) that the dot chromosomes in Drosophila are very difficult to analyze, because they are usually attached to or embedded in the chromocenter along with the proximal ends of the other chromosomes. Furthermore, they are usually not well-extended in chromosome squash preparations or not well-spread in SSP chromosomes. The latter case is probably a methodological problem based on the two different spreading effects involved (Kalisch et al. 1985b). Due to this, it has not been possible to depict so far all of the polytene structures of this chromosome in an individual photo map. Therefore, two examples are shown of which Fig. 1C shows more structural details of the proximal and Fig. 1D more of the distal part of the dot chromosome.

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Figure 1. D (Dot) chromosome of D. subobscura. (A) LM chromosome map based on squash preparations of salivary glands (Kunze-Mühl & Müller 1958). (B) Light micrograph, squash preparation, Orcein-staining, Neofluar 100/1.30 Ph objective, x3,200. (C-D) Electron micrographs of SSP chromosome preparations, x3,200. (E) EM chromosome map based on the cytological data of SSP chromosomes.
Kaningini, M., J. Lechien and A. Elens.  
FNDP, Namur, Belgium. Blind flies and normal flies tested for phototactism.

Visual mutants with abnormal electroretinograms have been described by Hotta & Benzer (1969). Being blind, they are normally not attracted by light; and one may expect that their behavior in phototactical tests will differ from that of normal flies. Consequently, they would be a very good material for students' practical exercises in behavior genetics or ethology, comparing visual mutants and wild controls.

In the present experiments, blind ora flies (kindly given by L. Craymer, of the California Institute of Technology) have been compared with wild Canton S flies, using the Kekic test (1981), and the Benzer test (1967) modified by Tompkins et al. (1978). The flies had been raised and maintained at 25°C and 60-80% R.H. Males and females had been separated from birth, and the flies were 5 days old when tested. The phototactical tests were done at 25°C and 60-80% relative humidity, with 5 repetitions, and at least 100 flies of same sex and age for each test. As a preliminary, the locomotor activity of flies of both strains had been compared in a square arena (30 flies of each sex, 5 days old, for each strain) according to Manning (1961), at 25°C and 60-80% R.H.; the differences are not significant.

Figure 1 shows the results of the Kekic test for males (right) and females (left) of both strains. For the same strain, the differences between males and females are highly significant; and the differences between the males and between the females of the two strains are highly significant as well (as shown by a chi-square test). Both Figure 2 and Figure 3 show the results of the Benzer test, but in the second case the experiment has been done "inside out": the flies have been tested first for negative phototactism (from light), and after that for positive phototactism (toward light). The differences between sexes are always highly significant for the wild Canton S flies, but never for the ora flies. In both methods (right side out and inside out), the differences between ora and wild Canton S flies are always highly significant (p < 0.001), but the behavioral differences are more evident with the "inside out" method, especially for the male flies.

Kaningini, M., J. Lechien, M. Lichtenberger and A. Elens. FNDP, Namur, Belgium. The measure of the "rare male mating advantage".

It appears that the "rare male mating advantage" was first observed by Petit (1951). This phenomenon has held the fascination of population geneticists for a long time and has given rise to a tremendous amount of controversy, with regards to its causes and even to its mere existence. Various approaches have been attempted to find its best mathematical expression, as seen in the recent, excellent review by Knoppien (1985). As a global expression of the phenomenon, the first formula, proposed by Petit, seems the best one. Petit's equation, also known as the "cross product ratio" (Knoppien 1985), can be expressed as

$$ K_M = \left( \frac{q_M}{p_M} \right) \left( \frac{P_M}{Q_M} \right) $$

where $p_M$ and $q_M$ are the respective frequencies of both types among males ($p_F$ and $q_F$ being the...
frequencies among females), and $P_M$ and $Q_M$ are the probabilities for the performance of any given mating by an A type male or by a B type male, respectively (Knoppien 1985). This equation expresses the male sexual fitness of one type relative to the other.

However, $K_M$ is only a global expression of the rare male advantage; and does not allow one to follow its effect during the course of time. It must be remembered that the course of mating successes with time in a sexual competition experiment has been expressed as a composite curve starting as an exponential but ending as a sigmoid. Different competing genotypes are characterized by a variety of curves in which different "plateaux" are reached at different rates. Accordingly, comparisons between various competing types may be biased when the length of the observation period is kept constant from one experiment to the other. In the case of sexual competitions where both types are in equal number, Wattiaux (1964) suggested the use of a logarithmic transformation which changes the curves of mating successes into straight lines. In Wattiaux's formula

$$\frac{dx_A}{n_A - x_A} dt$$

$n_A$ is the total number of individuals of one sex and one type, $dx_A$ the number of these flies mating in the interval of time $dt$, and $x_A$ the number of these individuals having already mated before. After logarithmic transformation the equation can be written as:

$$\ln \frac{dx_A}{n_A - x_A} dt = \ln b - kt = a - kt$$

The two constants $a$ and $k$ are easily estimated from the regression line $Y = A - k (X - X)$ where $Y$, (the value of $Y$ is estimated from regression), is the naperian logarithm of $x_A$ and $X$ is the mean of the independent variable $X$ (i.e., the time in minutes). The value of the method is based on the biological meaning of $A$ and $k$. The first coefficient provides information on the general level of sexual activity, while the second gives an insight on the variation of sexual activity with time.

For sexual competitions where both types are in equal number, Wattiaux suggested comparing the following regression lines: (1) homogamic and heterogamic matings, (2) matings of the males of both strains in competition, (3) matings of the females of both strains in competition, analogous to the "isolation estimate", "female mating ratio" and "male mating ratio" of Merrell (1950). This, indeed, has already been done for a sexual competition experiment where both types were in equal number (Elens et al. 1974).

The purpose of the present paper is to apply the same formula to the male sexual activities of the two types in competition, when their respective frequencies differ. For each interval of time (15 min), one has to calculate the quantity $\ln dx/(N - x)$, $dx$ being the number of males of a given type having mated during this interval, $N$ the total number of males of the same type, and $x$ the number of males which have already mated before the considered interval of time.
The laboratory strains used are the well known mutant strain "rucuca" and the Canton S "wild" strain. The sexual activity was determined by our "multiple choice direct observation method" fully described elsewhere (Elens 1958; Elens & Wattiaux 1964), and which has been widely used by Ehrman and others (Ehrman 1964, 1966, 1967, 1968; Ehrman et al. 1965; Petit & Ehrman 1968). Matings were directly observed. Individual males may mate more than once. Frequencies of types are varied among males and females simultaneously. In the present experiments, a total of 30 pairs of virgin flies, 4-5 days old, are used for each test. But the relative frequencies of both genotypes differed: 30/30, 15/30., or 20/30 of the pairs were wild, the other flies being "rucuca". The three "copulating chambers" were observed simultaneously. All the tests have been done at 25°C and 40-60% relative humidity. The copulating pairs were observed at time intervals less than the duration of copulation and the observations were recorded every 15 minutes. Five repetitions have been made.

The results are shown in Figure 1. The "wild" males are obviously advantaged when rare: the regression line at $q_{M/P}\cdot PM = 20/10$ (C) differs in slope (probability lower than 1/1000) and in elevation (probability lower than 1/100)from the regression line at frequency $q_{W/P}\cdot PM = 10/20$ (A); $PM$ and $q_{M}$ being the relative frequencies of "wild" and "rucuca". The regression line at $q_{M+/P}\cdot PM = 20/10$ (C) even differs in elevation (probability lower than 1%) from the regression line at equal frequencies (B). Obviously, many "wild" males have mated more than once, when they were rare. For the "rucuca" males, the regression lines are never significantly different. It seems that the sexual activity of the "wild" males is really stimulated by the presence of many "rucuca" flies. The factors acting in such a stimulation remain to be determined more precisely.

It has been emphasized, in one of our previous publications (Elens et al. 1964), that such a method may throw more light on the causes of the rare male advantage. It has been suggested, by Faugeres et al. (1964), that one has to distinguish between the "vigor factors" --or "metabolic factors"--, and the "behavioral factors" responsible for the rare male advantage. It is possible that a genotype is always sexually more active than the other one (this sexual vigor is higher); but it could be attributed to the fact that the presence of the second type is actually a stimulant for the first one (as previously said, it appears to be applicable to the "wild" males in the present case).


Knoppien, P. University of Groningen, Haren, Netherlands. Low density storage enhances mating speed in Drosophila melanogaster.

It has been known for some time that virgin Drosophila males stored singly are superior in mating compared to males stored in groups (Ellis & Kessler 1975; Knoppien 1985a; Knoppien, in press). It is conceivable that this effect results in rare male mating advantage in nature, since it may confer a mating advantage to males derived from a low-density population at the time that flies of different populations meet. Rare male mating advantage may in turn play a role in maintaining genetic variability (Knoppien 1985b). However, flies of both sexes are found together in nature, providing a situation different from the storage of virgin males at different densities in the laboratory.

The experimental approach to answer this question was as follows. Flies of D. melanogaster from an outbred Bogota strain were reared on standard agar medium (Knoppien, in press). They were etherized once within 8 hr from eclosion to collect virgin males and females. These flies were stored in vials, both sexes combined, at two different densities: at low density (L): 10 and 1° per vial, and at high density (H): 50° and 50° per vial, and then aged for 3-5 days. Low-density and high-density males were alternately marked by wing clipping for identification. Probably most of the females had mated after that time (this prediction was easily tested for the low-density vials by scoring the presence of progeny: only one of the fifty low-density females failed to produce progeny). Additionally, in order to test male mating success, virgin tester (T) females were stored without males (50° per vial), and aged for 3-5 days. These females were marked throughout the experiment by wing clipping. To test male mating success, 25 flies (5 L 1°, 5 L 1°, 5 H 1°, 5 H 1° and 5 T 1°) were combined in a mating chamber of the Elens-Wattiaux (1964) type of reduced size (all dimensions halved; Knoppien, in press), and mating was observed for one hour. An excess of males over non-fertilized females was used here, since this has been shown to intensify inter-male competition (Sharp 1982). Ten replicates of this experiment were made.
Only T females were found to mate in this experiment, which does not surprise, because probably nearly all L and H females had mated before. Mating success of low-density and high-density males was found to be strikingly different: 38 L \(d'\) and only 2 H \(d'\) mated \((X^2=30.63, P<0.001)\). Such a result is not entirely unexpected, since it is known that males can be discouraged to mate as a result of encounters with fertilized females (Siegel et al. 1984). These encounters will be more numerous at high density. The present results suggest that the density adult flies have experienced is an important agent in determining subsequent male mating speed. It is conceivable that this effect may also play a role in nature.


Kortier, M.G. and M.H. Gromko. Bowling Green State University, Ohio USNA. The effect of esterase-6 genotype on productivity.

The esterase-6 (EST-6) enzyme of D.melanogaster is polymorphic for two forms, S and F, in all natural populations studied (Oakeshott et al. 1981). Considerable effort has been devoted to the study of the possible adaptive significance of the two allozymes. Because the enzyme is concentrated in the male reproductive system and is transferred in the seminal fluid to females during copulation (Richmond et al. 1980), the focus of most investigations has been on the effects of the male enzyme type on reproduction by females. Here we present data that suggest the female EST-6 allozymes may be having effects on progeny production.

Lines homozygous at the locus coding for EST-6 were derived by pair matings of virgin flies from a wild stock of D.melanogaster (the same stock used by Gromko & Pyle 1978). After completion of copulation, males were removed from the vials and individual females allowed to oviposit for six days on cornmeal-molasses agar. Starch gel electrophoresis of the males and females was used to determine if a mating was homogametic or heterogametic, with the progeny from homogametic matings used to start homozygous lines. Since the S allele was more common in the wild stock, 10 SS lines were derived within a month but six more months were required to derive 10 FF lines. The lines were maintained in 8 dram food vials for approximately seven months before all lines of the same genotype were chain-crossed to start a large cage population. Flies from the two large cages created were used to start small one-generation cages from which virgin flies were collected under ether anesthesia for use in this experiment.

To collect the productivity data, SS and FF virgins were pair-mated with SS or FF virgin males in individual food vials, resulting in four groups of 30 mated females each. Males were removed within 30 minutes of completion of copulation. Females were transferred to fresh food every other day for eighteen days. All progeny emerging by 20 days at 23°C were counted.

Two-way analysis of variance revealed that FF females produced many more progeny than did SS females \((F=52.12, p<0.0001, \text{Table 1})\). There was no effect of male genotype \((F=0.14, p=0.7066)\) nor a significant interaction between male and female genotypes \((F=0.95, p=0.3321)\). Thus the female genotype appears to be responsible for the large differences in productivity. Although the direction of this difference agrees with Kojima & Yarbrough's (1967) finding of a slight but insignificant superiority of FF over SS females, the magnitude of the difference reported here is much greater. Our data do not agree with Birley & Beardmore's (1977) finding of no difference in fecundity of Est-6 homozygotes. We acknowledge the limitations of the chain cross as a means to eliminate linkage disequilibrium. Thus, within the framework of the inherent limitations of this sort of approach, we conclude that the region of chromosome marked by Est-6 has an effect on female function.


| Table 1. Average number of progeny produced by each Est-6 genotype mating combination (+ s.e.). Sample sizes are reported in square brackets. |
|---|---|---|
| | SS | Male |
| Female | | |
| SS | 58.6 | 18.6 |
| (+ 20.8) | (+ 5.1) |
| [25] | [26] |
| 243.4 | 261.0 |
| FF | 243.4 | 261.0 |
| (+ 20.8) | (+ 5.1) |
| [25] | [26] |

Drosophila nasuta, a member of the nasuta subgroup of the immigrans species group, was originally described by Lamb (1914) from the Seychelles Islands in the Indian Ocean. Besides this type locality, it has been also recorded from Kenya, Madagascar, Mauritius, Sri Lanka and India (Kitagawa et al. 1982). In India, the species is widespread and commonly seen particularly during colder months of the year.

In recent years, some attempts have been made to analyse naturally occurring inversion polymorphism in the Indian populations (Sajjan & Krishnamurthy 1974; Ranganath & Krishnamurthy 1975, 1978a, b; Rajasekaraksetty et al. 1979) as well as in the populations obtained from Sri Lanka, Mauritius, Kenya and Madagascar (Wakahama & Kitagawa 1980), but no efforts have been made to correlate their findings. During the last few years our studies on several populations of this species inhabiting the northern parts of the Indian subcontinent, in particular, have yielded altogether 22 paracentric inversions. In this report, we wish to provide a comprehensive review of the information on inversion polymorphism in this species. The final picture emerged from the results of these studies suggests this species to be highly polymorphic, carrying 85 distinct inversions (Table 1).

Acknowledgements: The financial assistance from the DST (Govt. of India) is thankfully acknowledged.

Table 1. Summary of naturally occurring inversions detected in Drosophila nasuta Lamb.

<table>
<thead>
<tr>
<th>Inversion</th>
<th>Photographic Ref.</th>
<th>Description</th>
<th>Breakpoints</th>
<th>Locality</th>
</tr>
</thead>
<tbody>
<tr>
<td>X-Chromosome:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>X-2</td>
<td>[7] as X-B; [8] as X-C</td>
<td>subterminal, overlaps with X-1</td>
<td>4-6</td>
<td>Ind, Sey</td>
</tr>
<tr>
<td>X-3</td>
<td>[4] as X-C</td>
<td>median</td>
<td>9-11</td>
<td>Ind</td>
</tr>
<tr>
<td>X-4</td>
<td>[8] as X-D</td>
<td>submedian</td>
<td>17-19</td>
<td>Sey</td>
</tr>
<tr>
<td>X-5</td>
<td>Present study</td>
<td>median</td>
<td>11-14</td>
<td>Ind</td>
</tr>
<tr>
<td>X-6</td>
<td>[8] as X-B</td>
<td>subterminal</td>
<td>4-8</td>
<td>Sey</td>
</tr>
<tr>
<td>X-7</td>
<td>[8] as LX-A</td>
<td>subterminal</td>
<td>4-9</td>
<td>SL</td>
</tr>
<tr>
<td>X-8</td>
<td></td>
<td>subterminal</td>
<td>3-9</td>
<td>Mrt</td>
</tr>
<tr>
<td>II-L-Chromosome:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>II-L-2*</td>
<td>[4] as 2L-B</td>
<td>median</td>
<td>34-49</td>
<td>Ind</td>
</tr>
<tr>
<td>II-L-3</td>
<td></td>
<td>as 2L-C</td>
<td>subterminal, overlaps with 2L-D</td>
<td>31-39</td>
</tr>
<tr>
<td>II-L-4</td>
<td></td>
<td>as 2L-D</td>
<td>subterminal, overlaps with 2L-3</td>
<td>34-43</td>
</tr>
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<td>II-L-5</td>
<td>[8] as 2L-A</td>
<td>subterminal</td>
<td>30-34</td>
<td>Sey</td>
</tr>
<tr>
<td>II-L-6</td>
<td>[8] as 2L-C</td>
<td>submedian</td>
<td>34-39</td>
<td>Sey, SL</td>
</tr>
<tr>
<td>II-L-7</td>
<td></td>
<td>as 2L-D</td>
<td>median</td>
<td>38-43</td>
</tr>
<tr>
<td>II-L-8</td>
<td></td>
<td>as 2L-E</td>
<td>med-4n</td>
<td>39-43</td>
</tr>
<tr>
<td>II-L-9</td>
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<td>as 2L-F</td>
<td>submedian</td>
<td>39</td>
</tr>
<tr>
<td>II-L-10</td>
<td></td>
<td>as 2L-G</td>
<td>submedian</td>
<td>40-49</td>
</tr>
<tr>
<td>II-L-11*</td>
<td>Present study</td>
<td>Complex</td>
<td>--</td>
<td>Ind</td>
</tr>
<tr>
<td>IIR-Chromosome:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IIR-1</td>
<td>[8] as 2R-A; [4] as 2R-G</td>
<td>subterminal</td>
<td>82-77</td>
<td>Ind, Sey</td>
</tr>
<tr>
<td>IIR-2*</td>
<td>[8] as 2R-B</td>
<td>submedian</td>
<td>74-71</td>
<td>Sey</td>
</tr>
<tr>
<td>IIR-3</td>
<td></td>
<td>as 2R-C</td>
<td>submedian</td>
<td>74-68</td>
</tr>
<tr>
<td>IIR-4</td>
<td>[7] as 2R-A</td>
<td>subterminal</td>
<td>82-74</td>
<td>Ind</td>
</tr>
<tr>
<td>IIR-5</td>
<td></td>
<td>as 2R-B</td>
<td>subterminal</td>
<td>81-76</td>
</tr>
<tr>
<td>IIR-6</td>
<td></td>
<td>as 2R-C</td>
<td>subterminal</td>
<td>81-74</td>
</tr>
<tr>
<td>IIR-7</td>
<td></td>
<td>as 2R-D</td>
<td>submedian</td>
<td>75-61</td>
</tr>
<tr>
<td>IIR-8</td>
<td>[4] as 2R-E</td>
<td>submedian, overlaps with 2R-7</td>
<td>73-59</td>
<td>Ind</td>
</tr>
<tr>
<td>IIR-9</td>
<td></td>
<td>as 2R-F</td>
<td>submedian, included within 2R-7</td>
<td>71-69</td>
</tr>
<tr>
<td>IIR-10*</td>
<td>Present study</td>
<td>subterminal</td>
<td>81-73</td>
<td>Ind</td>
</tr>
<tr>
<td>IIR-11*</td>
<td></td>
<td></td>
<td>submedian, included within 2R-12</td>
<td>77-60</td>
</tr>
<tr>
<td>IIR-12*</td>
<td></td>
<td></td>
<td>submedian, included within 2R-11</td>
<td>77-70</td>
</tr>
<tr>
<td>(IIR+II-L)-1</td>
<td>[7] as (2L-2R)-A</td>
<td>pericentric inversion between basal regions of 2L &amp; 2R</td>
<td></td>
<td>Ind</td>
</tr>
<tr>
<td>Inversion</td>
<td>Photographic Ref.</td>
<td>Description</td>
<td>Breakpoints</td>
<td>Locality</td>
</tr>
<tr>
<td>----------</td>
<td>------------------</td>
<td>--------------------------------------------------</td>
<td>-------------</td>
<td>-----------</td>
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<tr>
<td>III-Chromosome:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>III-1A</td>
<td>[8] as 3A</td>
<td>terminal</td>
<td>84-85</td>
<td>Sey, SL</td>
</tr>
<tr>
<td>III-1B</td>
<td>[3B as 3B</td>
<td>subterminal</td>
<td>84-85</td>
<td>Sey</td>
</tr>
<tr>
<td>III-2*</td>
<td>[4] as 3V</td>
<td>subterminal</td>
<td>85-87</td>
<td>Ind</td>
</tr>
<tr>
<td>III-3</td>
<td>[8] as 3C</td>
<td>submedian</td>
<td>86-89</td>
<td>Sey, SL</td>
</tr>
<tr>
<td>III-4</td>
<td></td>
<td>as 3D; [7] as 3B subterminal</td>
<td>89-94</td>
<td>Ind, Sey</td>
</tr>
<tr>
<td>III-5</td>
<td>[8] as 3E</td>
<td>median</td>
<td>97-112</td>
<td>Sey</td>
</tr>
<tr>
<td>III-7</td>
<td>[8] as 3G</td>
<td>submedian, included within III-6</td>
<td>97-109</td>
<td>Sey, SL</td>
</tr>
<tr>
<td>III-8</td>
<td>[8] as 3H</td>
<td>median</td>
<td>104-106</td>
<td>Sey</td>
</tr>
<tr>
<td>III-9</td>
<td></td>
<td>as 3I median</td>
<td>109-118</td>
<td>Sey</td>
</tr>
<tr>
<td>III-10</td>
<td></td>
<td>as 3J; [7] as 3P basal, overlaps with III-23</td>
<td>118-134</td>
<td>Ind, Sey, SL, Mad</td>
</tr>
<tr>
<td>III-11</td>
<td>[8] as 3K</td>
<td>subterminal</td>
<td>126-130</td>
<td>Sey, Mad</td>
</tr>
<tr>
<td>III-12</td>
<td>[7] as 3A</td>
<td>subterminal</td>
<td>85-87</td>
<td>Ind</td>
</tr>
<tr>
<td>III-13</td>
<td></td>
<td>as 3C subterminal</td>
<td>91-96</td>
<td>Ind</td>
</tr>
<tr>
<td>III-14*</td>
<td></td>
<td>as 3E basal</td>
<td>112-122</td>
<td>Ind</td>
</tr>
<tr>
<td>III-15*</td>
<td></td>
<td>as 3H basal, overlaps with III-14 &amp; III-19</td>
<td>121-133</td>
<td>Ind</td>
</tr>
<tr>
<td>III-16</td>
<td></td>
<td>as 3I subterminal</td>
<td>132-135</td>
<td>Ind</td>
</tr>
<tr>
<td>III-17</td>
<td></td>
<td>as 3J basal overlaps with III-14 &amp; III-17</td>
<td>128-136</td>
<td>Ind</td>
</tr>
<tr>
<td>III-18</td>
<td>[8] as 3G</td>
<td>subterminal, tandem inversion with III-33</td>
<td>132-133</td>
<td>Ind</td>
</tr>
<tr>
<td>III-19</td>
<td></td>
<td>as 3K basal, overlaps with III-15</td>
<td>127-134</td>
<td>Ind</td>
</tr>
<tr>
<td>III-20</td>
<td>[8] as 3L</td>
<td>basal, overlaps with III-14</td>
<td>119-137</td>
<td>Ind</td>
</tr>
<tr>
<td>III-21</td>
<td></td>
<td>as 3M basal, overlaps with III-14</td>
<td>122-136</td>
<td>Ind</td>
</tr>
<tr>
<td>III-22</td>
<td>[8] as 3N</td>
<td>basal, overlaps with III-10</td>
<td>122-134</td>
<td>Ind</td>
</tr>
<tr>
<td>III-23</td>
<td></td>
<td>as 3O basal, overlaps with III-10</td>
<td>119-136</td>
<td>Ind</td>
</tr>
<tr>
<td>III-24</td>
<td>[4] as 3S</td>
<td>basal</td>
<td>127-131</td>
<td>Ind</td>
</tr>
<tr>
<td>III-25</td>
<td></td>
<td>as 3T basal</td>
<td>132-124</td>
<td>Ind</td>
</tr>
<tr>
<td>III-26</td>
<td></td>
<td>as 3U submedian, associated with III-13</td>
<td>90-91</td>
<td>Ind</td>
</tr>
<tr>
<td>III-27</td>
<td></td>
<td>as 3W submedian, included within III-28</td>
<td>121-124</td>
<td>Ind</td>
</tr>
<tr>
<td>III-28</td>
<td></td>
<td>as 3X basal</td>
<td>120-134</td>
<td>Ind</td>
</tr>
<tr>
<td>III-29</td>
<td></td>
<td>as 3Q submedian, included within overlapping</td>
<td>116-121</td>
<td>Ind</td>
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<tr>
<td>III-30*</td>
<td></td>
<td>inversions III-14 &amp; III-15</td>
<td>116-121</td>
<td>Ind</td>
</tr>
<tr>
<td>III-31</td>
<td>[3] as 3Y</td>
<td>submedian, overlaps with III-32</td>
<td>112-127</td>
<td>Ind</td>
</tr>
<tr>
<td>III-32</td>
<td></td>
<td>as 3Z submedian, overlaps with III-31</td>
<td>119-136</td>
<td>Ind</td>
</tr>
<tr>
<td>III-33</td>
<td>[7] as 3F</td>
<td>basal, tandem inversion with III-18</td>
<td>126-132</td>
<td>Ind</td>
</tr>
<tr>
<td>III-34*</td>
<td>Present study</td>
<td>submedian</td>
<td>97-110</td>
<td>Ind</td>
</tr>
<tr>
<td>III-35*</td>
<td></td>
<td>median</td>
<td>98-123</td>
<td>Ind</td>
</tr>
<tr>
<td>III-36*</td>
<td></td>
<td>basal</td>
<td>124-133</td>
<td>Ind</td>
</tr>
<tr>
<td>III-37*</td>
<td></td>
<td>basal, includes III-30</td>
<td>119-133</td>
<td>Ind</td>
</tr>
<tr>
<td>III-38*</td>
<td></td>
<td>basal</td>
<td>117-133</td>
<td>Ind</td>
</tr>
<tr>
<td>III-39*</td>
<td></td>
<td>submedian</td>
<td>105-118</td>
<td>Ind</td>
</tr>
<tr>
<td>III-40*</td>
<td></td>
<td>submedian</td>
<td>114-122</td>
<td>Ind</td>
</tr>
<tr>
<td>III-41*</td>
<td></td>
<td>median</td>
<td>98-127</td>
<td>Ind</td>
</tr>
<tr>
<td>III-42*</td>
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<td>112-122</td>
<td>Ind</td>
</tr>
<tr>
<td>III-43*</td>
<td></td>
<td>submedian, tandem inversion with III-42</td>
<td>122-132</td>
<td>Ind</td>
</tr>
<tr>
<td>III-44</td>
<td>[4] as Fig. 5B</td>
<td>basal, complex</td>
<td>--</td>
<td>Ind</td>
</tr>
<tr>
<td>III-45</td>
<td></td>
<td>as Fig. 5C basal, complex</td>
<td>--</td>
<td>Ind</td>
</tr>
<tr>
<td>III-46</td>
<td></td>
<td>as Fig. 5D basal, complex</td>
<td>--</td>
<td>Ind</td>
</tr>
<tr>
<td>III-47</td>
<td></td>
<td>as Fig. 5E basal, complex</td>
<td>--</td>
<td>Ind</td>
</tr>
<tr>
<td>III-48</td>
<td></td>
<td>as Fig. 5F basal, complex</td>
<td>--</td>
<td>Ind</td>
</tr>
<tr>
<td>III-49*</td>
<td>Present study</td>
<td>basal, complex</td>
<td>--</td>
<td>Ind</td>
</tr>
<tr>
<td>III-50</td>
<td>[8] as M3-A</td>
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<td>86-88</td>
<td>KY</td>
</tr>
<tr>
<td>III-51</td>
<td></td>
<td>as M3-B submedian</td>
<td>94-96</td>
<td>KY</td>
</tr>
<tr>
<td>III-52</td>
<td></td>
<td>as L3-A basal</td>
<td>129-131</td>
<td>SL</td>
</tr>
</tbody>
</table>

*Inversions encountered during present study. Ind=India; Sey=Seychelles; Mrt=Mauritius; SL=Sri Lanka; KY=Kenya; Mad=Madagascar.

Two populations of *D.melanogaster* from Iraq were analyzed to determine the maintenance of genetic variation at Esterase-6 (Est-6 map position 36.8) and Phosphoglucomutase (PGM map position 43.6), both located on chromosome 3. This work is a continuation of our previous investigation (Lamooza et al. 1985). Flies were collected from the two localities: Basrah (B) and Tuwaitha (T); the nature of collections and collecting procedures were the same as that described in our previous investigation. Collections were continued over a period of 3 years for about the same time each year. Electrophoretic separations were performed as specified for each reaction mixture for the two enzymes, where phenotypes are similar to those which were observed in our previous studies. Chi-square tests for heterogeneity were performed to demonstrate the variation between populations. Allezyme allele frequency estimates are given in Table 1. The fast allele in all collections ranged from 0.271 to 0.693. The allele frequencies were significantly different between T & B populations among collections at PC 0.001. The slow allele in T population was the most dominant allele through 1981-1983. Previous results indicated that the fast allele acquired the most adaptive value 0.72, 0.732 for 1979, 1980, respectively, and this pattern of genetic variation may be due to chance and/or to the result of external causative forces. In B population the fast allele frequency fluctuated and showed a dominant value in 1982. The results show that configuration of allelic frequencies at Est-6 locus was different between the two localities during the years of collection. Variation in allele frequencies could be ascribed to the geographic and environmental variability (Rockwood-Sluss 1973; DE Albuquerque et al. 1981). On the other hand, Bryant (1974) considers that the genetic variation seems to be associated with temporal variation in the environment.

As PGM locus the fast allele in T population shows the most dominant values as shown in Table 2, confirming the previous results in 1979, 1980 (0.910, 0.912, respectively). The PGM<sup>f</sup> allele shows an apparent increase and seems to become monomorphic in 1983 (frequency of fast allele: 1.000). Although it is impossible to predict whether PGM<sup>s</sup> allele would be eventually eliminated, this requires further and more extensive sampling of this population to have a clear picture of this situation.

Acknowledgements: We wish to thank Dr. Mohammed S.H. Ahmed for critical reading of the manuscript.


---

**Table 1.** Est-6 allele frequencies in Tuwaitha and Basrah populations of *D.melanogaster*, where the results for the faster (F) allele (out of two alleles) are shown.<sup>a</sup>

<table>
<thead>
<tr>
<th>Year of collection</th>
<th>Tuwaitha allele frequency ± S.D.</th>
<th>Basrah allele frequency ± S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>0.26±0.033</td>
<td>0.34±0.033***</td>
</tr>
<tr>
<td></td>
<td>(181)</td>
<td>(200)</td>
</tr>
<tr>
<td>1982</td>
<td>0.35±0.053</td>
<td>0.69±0.043***</td>
</tr>
<tr>
<td></td>
<td>(81)</td>
<td>(114)</td>
</tr>
<tr>
<td>1983</td>
<td>0.27±0.037</td>
<td>0.29±0.046***</td>
</tr>
<tr>
<td></td>
<td>(164)</td>
<td>(79)</td>
</tr>
</tbody>
</table>

<sup>a</sup> figures in () show sample size. 
*** H.S. difference from Tuwaitha data.

---

**Table 2.** PGM allele frequencies for Tuwaitha population.<sup>a</sup>

<table>
<thead>
<tr>
<th>Year of collection</th>
<th>PGM allele frequency ± S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1981</td>
<td>0.96±0.019</td>
</tr>
<tr>
<td></td>
<td>(79)</td>
</tr>
<tr>
<td>1982</td>
<td>0.84±0.039</td>
</tr>
<tr>
<td></td>
<td>(83)</td>
</tr>
<tr>
<td>1983</td>
<td>1.000</td>
</tr>
<tr>
<td></td>
<td>(164)</td>
</tr>
</tbody>
</table>

<sup>a</sup> Figures in () show sample size.
Phylogenetic relationships among five sibling species, D.auraria, D.biauraria, D.triauraria, D.quadraria and D.subauraria were investigated by SDS polyacrylamide gel electrophoresis (SDS PAGE) and two-dimensional electrophoresis (TDE).

Water soluble protein patterns of the five species were compared by SDS PAGE. Similarities among D.auraria, D.triauraria and D.quadraria were found though there was a slight difference in density, while the pattern of D.biauraria was similar to D.subauraria.

### Table 1. Estimates of genetic distance between D.aurantia complex obtained by TDE.

<table>
<thead>
<tr>
<th>Species</th>
<th>D.aurantia</th>
<th>D.biauraria</th>
<th>D.triauraria</th>
<th>D.quadraria</th>
</tr>
</thead>
<tbody>
<tr>
<td>D.aurantia</td>
<td>0.204</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D.biauraria</td>
<td>0.130</td>
<td>0.124</td>
<td></td>
<td></td>
</tr>
<tr>
<td>D.quadraria</td>
<td>0.110</td>
<td>0.163</td>
<td>0.070</td>
<td></td>
</tr>
<tr>
<td>D.subauraria</td>
<td>0.213</td>
<td>0.052</td>
<td>0.090</td>
<td>0.170</td>
</tr>
</tbody>
</table>

In consequence, it could be found that D.biauraria was more closely related with D.subauraria, and D.aurantia was distantly related with D.subauraria. The direction of speciation in these sibling species appeared to be made from D.quadraria to D.aurantia and D.triauraria, and from D.triauraria to D.biauraria and D.subauraria, respectively.


Pupal, larval or adult testes were isolated from seven different species of Drosophila representing the four species groups melanogaster, repleta, immigrans and virilis. To isolate single cysts the testes were punctured with fine tungsten needles. Routinely around 50 cysts would flow undamaged through the punctured testis membrane. They were cultured as described elsewhere (Liebrich 1981), except that 1 mm thick (rather than 2.2 mm) culture chambers were used.

In general, cysts were isolated from the testes of late pupae and newly emerged adults where a wide range of stages in spermatogenesis is present. Exceptions were D.melanogaster where cysts isolated from late larval or young pupal testes have already commenced to differentiate and are relatively easy to isolate and D.bifurca where only cysts of adult tests could be successfully cultured.

The cysts of all six species (D.melanogaster, D.neohydei, D.bifurca, D.fulvimacula, D.virilis, D.simulans) continue to differentiate in culture and pass through meiosis and early spermiogenesis. A time-table of duration of phases of meiosis and spermiogenesis was constructed for five species. Although in all 5 species investigated the differentiation processes and the duration of the different stages are similar, there are particular species-specific differences in morphological details.

Especially in D.melanogaster during elongation phase a few spermatid nuclei can often be detected in the "tail region" of the bundle (Fig. 1d, arrows). Since this phenomenon is also seen in freshly explanted cysts, it cannot be caused by any culturing effect. The lagging behind of the nuclei may be a genuine failure of the nuclei to migrate to the head, but could also be due to an improper orientation of the whole spermatid.

In detail there are differences between the cysts of all investigated species of Drosophila such as the morphology of the Y-chromosomal loops appearing in prophase I (Hess 1967), the number of germ cells per cyst (Hanna et al. 1982; Liebrich et al. 1982) or the number of Nebenkern derivatives (Hess & Meyer 1968). On the other hand, differentiating cysts of species of the same group or subgroup (according to Patterson & Stone 1952) may have the same morphological appearance during meiosis and spermiogenesis in vitro: cysts isolated from D.melanogaster and D.simulans (melanogaster group) are of very similar appearance. It is nearly impossible to discriminate between cysts of D.hydei, D.neohydei, and D.eohydei (repleta group, hydei subgroup). Surprisingly, males of D.virilis, though this species belongs to a different group, have cysts with a morphology similar to cysts of D.hydei, D.bifurca and D.fulvimacula, on the other hand, are an exception in that they belong to the same subgroup (melanapalpa of repleta group), but their
Figure 1a-d. Single isolated cyst of different species of Drosophila differentiating in vitro.

1d. **D. melanogaster.** Spermatid bundle elongation. Spermatid nuclei (n) aggregate at the head end, a few at the "wrong" tail end of the bundle (Arrows). DIC, x480.

1a. **D. fulvivaculata.** Meiosis I. Arrow indicates chromosomes that can be seen between the mitochondria of a spermatocyte. Phaco, x770.

1c. **D. bifurca.** Meiosis I (m: mitochondria of a spermatocyte). DIC, x480.

1b. **D. immigrans.** Interphase between meiosis I and II (m: aggregated mitochondria of a spermatocyte). Phaco x480.

In cysts of **D. immigrans** (subgroup melanogaster) the germ cell mitochondria aggregate by the end of the first meiotic division (Fig. 1b). During the second meiotic division they remain more or less aggregated. Thus, it is difficult to discriminate between metaphase I and the phase of Nebenkern formation at the end of meiosis II.

In cysts of **D. melanogaster** during prophase I the Y-chromosomal loops first disintegrate into granular material after which the spindle apparatus is formed. In cysts of **D. hydei** the reverse occurs. Spermatids
Table 1. Time of duration of the single phases of meiosis and spermiogenesis of 5 Drosophila species at (22-23)°C. Time in hours, + = not exactly determined: in all cases more than 7 hr.

<table>
<thead>
<tr>
<th>phase</th>
<th>D.melanogaster</th>
<th>D.hydei macula</th>
<th>D.fulvi ilis</th>
<th>D.vir grans</th>
</tr>
</thead>
<tbody>
<tr>
<td>metaphase I to telophase I</td>
<td>0.7-1.5</td>
<td>2</td>
<td>1.5-2</td>
<td>2</td>
</tr>
<tr>
<td>interphase</td>
<td>0.8-1</td>
<td>0.3-5</td>
<td>1</td>
<td>1.5</td>
</tr>
<tr>
<td>meiosis II</td>
<td>0.7-2</td>
<td>0.3-1-1.5</td>
<td>2.5</td>
<td>1.5</td>
</tr>
<tr>
<td>Nebenkern formation</td>
<td>0.8-2</td>
<td>2-3</td>
<td>1.7</td>
<td>1-1.5</td>
</tr>
<tr>
<td>onion phase</td>
<td>6.5-7</td>
<td>8</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

Spermatid elongation and nuclear movement

2-3          5     5     4     5

The results show that the general course of meiosis and spermiogenesis is the same in all Drosophila species investigated so far. However, the events within the single phases are not strictly determined. It seems unimportant, e.g., whether there exist one or two Nebenkern derivatives and 16 or 21 spermatogonia. The spindle apparatus may appear earlier or later in prophase I, or the germ cell mitochondria aggregate during meiosis or after meiosis. For the correct course of spermatogenesis, it is only important that these events take place. The findings may serve as an additional example that general norms exist for distinct differentiation processes. The way, however, to reach this norm is not determined exactly.

Acknowledgement. The author wishes to thank Mrs. Ursula Glos-Mettbach for her helpful technical assistance. The work was supported by the Deutsche Forschungsgemeinschaft.


HCl, pH 8.5 and containing 10 mg NADP, 20 mg glucose-6-phosphate dehydrogenase, 30 units phosphoglucomutase, 2 units phosphoglucoisomerase, 2 mg Nitro Blue Tetrazolium and 1 mg Phenazine Methosulfate.

To localize MPI we used the net strain (net: a recessive mutant located on chromosome U), the ch cu strain (cherry eyes and curled wings: both recessive mutants located on chromosome O), the p pl strain (poppy eyes and plexus wings: both recessive mutants located on chromosome E), the enzyme locus PGM (phosphoglucomutase located on chromosome J), and finally the enzyme locus XDH (xanthine dehydrogenase located on chromosome O). The following crosses demonstrated that MPI is located on chromosome O.

For chromosome U: Males of the net strain, homozygous for the allele MPI1.00 were crossed with females of a wild type strain homozygous for the allele MPI1.07. F1 males were then crossed with females of the net strain. Half of the wild and half of the net progeny of this backcross were heterozygous (MPI1.07/MPI1.00). Those of the other half of both phenotypes were homozygous (MPI1.00/MPI1.00).

For chromosome J: Males of a strain homozygous for allele PGM1.00 and for allele MPI1.07 were crossed with females homozygous for the alleles PGM1.10 and MPI1.00. F1 males were then backcrossed with one of the parents. Half of the homzygous and half of the heterozygous for the PGM locus progeny were homozygous for the MPI locus. Those of the other half of both electrophoretic phenotypes for the PGM locus were heterozygous for MPI.

For chromosomnes E and O: Males of the ch cu strain, homozygous for allele MPI1.11, were crossed with females of the p pl strain, homozygous for the allele MPI1.07. Some of the F1 males were crossed with females of the p pl strain and some others with females of the ch cu strain. Half of the wild and half of the p pl progeny of the first backcross were heterozygous (MPI1.11/MPI1.07) while those of the other half of both phenotypes were homozygous (MPI1.07/MPI1.07). All the wild type progeny of the second backcross were heterozygous (MPI1.11/MPI1.07) and all the ch cu homozygous (MPI1.11/MPI1.11).
Table 1.

<table>
<thead>
<tr>
<th>Genotype of female parent</th>
<th>Markers</th>
<th>Crossovers</th>
<th>Sample size</th>
<th>Recombination value</th>
<th>Map distance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. 034+, cu, MPI1.11, ch</td>
<td>cu-MPI</td>
<td>102</td>
<td>276</td>
<td>0.370±0.029</td>
<td>0.461</td>
</tr>
<tr>
<td>034+, + MPI1.00, +</td>
<td>MPI-ch</td>
<td>3</td>
<td>276</td>
<td>0.011±0.006</td>
<td>0.011</td>
</tr>
<tr>
<td>2. 034+, cu, XDH1.00, MPI1.11, ch</td>
<td>cu-XDH</td>
<td>53</td>
<td>185</td>
<td>0.286±0.033</td>
<td>0.327</td>
</tr>
<tr>
<td>034+, +, XDH0.94, MPI1.00, +</td>
<td>XDH-MPI</td>
<td>20</td>
<td>185</td>
<td>0.108±0.023</td>
<td>0.112</td>
</tr>
<tr>
<td></td>
<td>MPI-ch</td>
<td>5</td>
<td>185</td>
<td>0.027±0.012</td>
<td>0.027</td>
</tr>
</tbody>
</table>

Therefore, the enzyme locus MPI is located on chromosome 0.

In order to construct a genetic map of the region of chromosome 0 in which the MPI is located, we analyzed the progeny of two relative crosses: For each cross we give all the data pertaining to the estimation of the recombination distances (shown in Table 1).

The male parent of each cross was cytologically and genically homozygous. The map distance was corrected for multiple crossovers using the formula proposed by Haldane (1919): \( x=0.7y-0.15\log(1-2y) \), where \( y \) is the percentage of crossovers. Pooling the data of crosses 1 and 2 (that gave homogenous results) and taking into account map distances from previous reports (Loukas et al. 1979; Zouros & Krimbas 1973), we have for the chromosome 0 of *D. subobscura*:

\[
\begin{array}{c|c|c|c}
\text{centr.} & 52.6 & 86.0 & 97.2 \\
\text{cu} & & XDH & ch \\
\end{array}
\]

In order to study the genetic differentiation of MPI-locus in Drosophila species of the obscura group, we used eight different isofemale strains for *D. subobscura*; one for *D. madeirensis*; one for *D. guanche*; eleven for *D. obscura*; three for *D. tristis*; seven for *D. ambigua*; two for *D. subsilvestris*; three for *D. bifasciata*; one for *D. pseudoobscura* and one for *D. helvetica*. MPI classified the ten species into six different electrophoretic phenotypes. *D. madeirensis*, *D. obscura* and *D. subsilvestris* displayed the allele 1.00. Four strains of *D. subobscura* were homozygous for allele 1.00, two for allele 1.07 and two for allele 1.11. *D. guanche* and *D. tristis* displayed the allele 1.03. Four strains of *D. ambigua* were homozygous for allele 1.03 and three for allele 0.98. Finally, *D. bifasciata*, *D. pseudoobscura* and *D. helvetica* displayed the alleles 1.09, 1.11 and 1.16, respectively.


Lovering, R. Sheffield University, Sheffield, England. Morphology of the proximal region of 3R in salivary gland polytene nuclei of *D. melanogaster*.

In the polytene chromosome 3R of *D. melanogaster* the region proximal to 81F was described by Bridges (1935-1940) and by Lefevre (1976) as heterochromatic. Heterochromatin is characteristically unbanded and granular in appearance, but the Bridges's maps, and electronmicrographs by Saura (pers. comm. 1979), suggest that some heterochromatic bands can be identified. In Lefevre's photographic representation of chromosome 3R the most proximal band illustrated is the doublet 81F1-2. However, Lefevre also suggests that heterochromatic material is present between 81F and 80F.

Electron micrographs produced by Saura suggest that bands proximal to 81F do exist, and these they have labelled 80B-F. Nevertheless, the bands are heterochromatic, maintaining some of the granular appearance characteristic of this region. The morphology of 81F in the electron micrographs suggests that it may contain heterochromatin, as the bands are more dense than euchromatic bands and, like the chromocentre, it is strongly fluorescent when stained with quinacrine; this suggests that it contains a high frequency of A-T base pairs (Barr & Ellison 1972). This region cannot, therefore, be considered as strictly euchromatic.

I have been investigating the DNA content of the proximal region of 3R by Feulgen staining of salivary gland nuclei, and have observed that the morphology of the region proximal to 81F varies from cell to cell. Four different morphological categories can be detected (see Fig. 1).

(a) The doublet in 81F is closely associated with the heterochromatic chromocentre; it is not possible to define the proximal boundary of the band.
Figure 1. The proximal region of 3R; (e) Bridges's map 1941; the arrow indicates an extra band proximal to 81F1-2. The bar corresponds to 5μ.

Table 1. The effect of larval incubation temperature on the morphology of the region proximal to 81F.

<table>
<thead>
<tr>
<th>Temp.</th>
<th>Morphology of the proximal region of 3R</th>
<th>Total nuclei</th>
<th>No. slides</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>a</td>
<td>b</td>
<td>c</td>
</tr>
<tr>
<td>15°C</td>
<td>23</td>
<td>12</td>
<td>11</td>
</tr>
<tr>
<td>25°C</td>
<td>10</td>
<td>6</td>
<td>18</td>
</tr>
<tr>
<td>Total</td>
<td>33</td>
<td>18</td>
<td>29</td>
</tr>
</tbody>
</table>

\[ X^2 = 16.01; D.O.F. = 3; p < 0.01. \]

(b) An interband is visible between the doublet and the heterochromatin; the proximal boundary of the band can be defined.

(c) 81F1-2 is attached to the chromocentre by a thin thread-like structure, as seen in Lefevre's photographs (1976). The doublet itself is not usually distorted by this condition.

(d) One or two bands are visible proximal to 81F1-2 and distinct from the heterochromatin. It is also possible that these bands are 81F4-5 and that 81F1-2 is very closely associated with the chromocentre.

The morphology of the proximal region of 3R was studied in larvae reared at different incubation temperatures. The distribution of nuclei among the morphological classes was significantly affected by the temperature at which the larvae were reared (p<0.01, see Table 1). In larvae reared at 15°C the double band 81F1-2 was closely associated with the chromocenter (type a) in almost half of the nuclei, whereas only a fifth of the nuclei from 25°C-reared larvae fell into this class. In addition at 25°C four times more class d nuclei were observed, with bands proximal to 81F, than at 15°C.

Using the light microscope it is not possible to determine whether the extra bands observed proximal to 81F originate from 81F or from the chromocentre. As the larval incubation temperature has a significant effect on the morphology of this region, it seems unlikely that the presence or absence of bands proximal to 81F is an artifact.

The heterochromatic chromocentre usually replicates twice, without cell division (Rudkin 1969), whereas the euchromatin may initiate between 8 and 11 S-phases. In chromosome 3R the bands 81F1-2 are in, or very close to, a region where the transition from euchromatin to heterochromatin occurs. At the junction between the two different types of chromatin, a large number of replication forks are thought to be present (Laird 1973) because of the substantial change in polyteny.

Studies on the Feulgen-DNA content of the polytene nuclei (Hartmann-Goldstein & Goldstein 1979, and unpubl.) have shown that an increase in larval incubation temperature causes a decrease in polyteny. In larvae reared at 15°C the euchromatin is usually at a higher polytene level than in 25°C reared larvae. Thus the effect of temperature observed on the morphology of 3R may be due to the effect of temperature on the polytene levels in the nucleus. If the increase in the DNA content occurred only in the euchromatin, and not the heterochromatin, then at the proximal regions of the chromosomes there would be an increase in the number of replication forks. The alignment of chromomeres might be disrupted in a region where many replication forks occur, so causing the banding pattern to be disorganised; these bands might then appear granular.

In nuclei of 25°C-reared larvae, bands are observed proximal to 81F more frequently than in 15°C larvae. An increase in euchromatin polyteny, due to a decrease in the larval incubation temperature, could cause the loss of banding pattern proximal to 81F by increasing the stress imposed on the structure by the large numbers of replication forks. Alternatively, the structural proteins themselves might be affected by the temperature.
Acknowledgement: This work was supported during tenure of an SERC (C.B.) postgraduate award.


Loverre, A. D.A. Hickey* & G. Carmody.§
Universita di Roma, Italy; *University of Ottawa, Canada; §Carleton University, Ottawa, Canada.
[1] Present address: Biology Dept., University of Ottawa, Ont. K1N 6N5 Canada. A test of the hypothesis that the Segregation Distortion phenomenon in Drosophila melanogaster is characterized by the preferential recovery of Distorter (SD) second chromosomes in the progeny of heterozygous males (Sandler, Hiraizumi & Sandler 1959; see also Hartl & Hiraizumi 1976; Sandler & Golic 1985, for reviews).

The Segregation Distorter phenomenon in Drosophila melanogaster is characterized by the preferential recovery of Distorter (SD) second chromosomes in the progeny of heterozygous males (Sandler, Hiraizumi & Sandler 1959; see also Hartl & Hiraizumi 1976; Sandler & Golic 1985, for reviews).

A new model of Segregation Distortion in Drosophila has been proposed (Hickey, Loverre & Carmody 1986) and experimental results are presented here as a test of the proposed model. According to the model, the Segregation Distorter (Sd) locus is the residence site of a transposable element, while the responder (Rsp) locus is a target site for insertion of copies of this element. Insertion of the transposon causes disruption of the sensitive chromosomes which, in turn, leads to the dysfunction of non-SD-bearing sperm. The experimental work is aimed at finding evidence of chromosomal contamination by the Sd element of non-Sd chromosomes which had been combined with SD chromosomes in heterozygotes. The non-SD chromosomes were then tested for the acquired ability to induce distortion, a phenotype which would indicate the presence of an acquired Sd element.

The one major complication in this test is the following. According to the model, the non-SD homologue is normally contaminated at the Rsp site and this event leads to sperm dysfunction. Therefore we can only hope to pick up transposition events at secondary sites. Secondly, because we use SD-activity as a measure of chromosome contamination, any transposition events which involved rearrangement of transposon sequences or insertion in chromosomal regions which did not allow further high-frequency transposition, would not be detected. Moreover, Sd elements on naturally-occurring SD chromosomes are surrounded by a co-adapted series of elements that allow a high degree of distortion against sensitive chromosomes. Thus, although a positive result would strongly support the genetic transposition model, a negative result would not rule it out.

Most experiments followed the scheme outlined in Figure 1. SD chromosomes were made heterozygous with non-SD "target" chromosomes. These non-SD chromosomes, which had been exposed to SD activity, were then made heterozygous with other non-SD chromosomes ("tester" chromosomes) and the heterozygous males mated to appropriately marked females to calculate a k-value, i.e., the ratio of target chromosome-bearing progeny to total progeny.

The SD chromosomes used in this study were SD-5, SD-72 and SD-Madison. All three chromosomes display high levels of distortion (k>0.98) when heterozygous with sensitive chromosomes.

Figure 1. Scheme of crosses to recover SD+ chromosomes after exposure to SD, and for testing them for distorting ability.
June 1986  

**Table 1. Distribution of k-values for potentially contaminated and control chromosomes.** Each cross in this case was between one bw/lt bw male and two lt bw/lt bw females.

<table>
<thead>
<tr>
<th>SD/Target</th>
<th>Target/Tester</th>
<th>no. males tested</th>
<th>Total progeny</th>
<th>k±st.dev.</th>
<th>Males with k&gt;0.70</th>
</tr>
</thead>
<tbody>
<tr>
<td>SD-5/bw</td>
<td>bw/lt bw</td>
<td>8</td>
<td>629</td>
<td>0.65±0.10</td>
<td>3</td>
</tr>
<tr>
<td>SD-72/bw</td>
<td>bw/lt bw</td>
<td>9</td>
<td>877</td>
<td>0.59±0.12</td>
<td>2</td>
</tr>
<tr>
<td>Control</td>
<td>bw/lt bw</td>
<td>14</td>
<td>948</td>
<td>0.58±0.08</td>
<td>1</td>
</tr>
</tbody>
</table>

**Table 2. Average k-values for insensitive target chromosomes when tested in heterozygosis with sensitive tester chromosomes.** For estimating k values, males heterozygous for cn bw were crossed with cn bw/cn bw females, while males heterozygous for lt bw were crossed with lt bw/lt bw females.

<table>
<thead>
<tr>
<th>Target/Tester Combination</th>
<th>Average k-value (±st.dev.)</th>
<th>Range (min-max)</th>
<th>No. Chromosomes tested</th>
<th>Total progeny scored</th>
<th>Males with k&gt;0.65</th>
</tr>
</thead>
<tbody>
<tr>
<td>pr cn/cn bw</td>
<td>0.49±0.06</td>
<td>0.36-0.63</td>
<td>37</td>
<td>2558</td>
<td>0</td>
</tr>
<tr>
<td>Df(2R)14C/lt bw</td>
<td>0.50±0.05</td>
<td>0.37-0.61</td>
<td>20</td>
<td>1372</td>
<td>0</td>
</tr>
<tr>
<td>Df(2R)2J/lt bw</td>
<td>0.53±0.07</td>
<td>0.38-0.76</td>
<td>40</td>
<td>2543</td>
<td>3</td>
</tr>
<tr>
<td>R(Cy-40)/cn bw</td>
<td>0.52±0.07</td>
<td>0.37-0.75</td>
<td>122</td>
<td>7833</td>
<td>4</td>
</tr>
<tr>
<td>Control*</td>
<td>0.49±0.08</td>
<td>0.33-0.63</td>
<td>19</td>
<td>1752</td>
<td>0</td>
</tr>
</tbody>
</table>

*The "Control" genotype is identical to the preceding R(Cy)-40/cn bw combination. In the case of the control experiments, however, the R(Cy)-40 chromosome was not heterozygous with an SD chromosome in the previous generation (See Fig. 1).*

Single crosses (1 male X 2 females) were carried out for measuring k values.

All stocks and crosses were maintained at 25±1°C.

For a full description of the chromosomes and markers used, see Lindsley & Grell (1968).

I. Testing for chromosomal contamination in rare escaper males. The first set of experiments tested for contamination of Sd+ Rsp+ chromosomes in SD/SD+ males showing a k value greater than 0.9. The bw chromosome, isolated from a bw stock, allows a high degree of distortion by SD-5 or SD-72 (k=0.98). Aged males of the genotype SD-5/bw (k=0.96) and SD-72/bw (k=0.93) were crossed to lt bw homozygous tester females. The use of older males results in an increased recovery of bw chromosomes. From among the progeny, bw/lt bw males were recovered and examined. In this case the bw chromosome represents the target chromosome and the lt bw is a more sensitive tester chromosome. The results are presented in Table 1. The prediction of the model is that contaminated target chromosomes might distort ratios in their own favor when heterozygous with the very sensitive lt bw chromosome. As can be seen from Table 1, some bw chromosomes do indeed show evidence of an ability to distort against lt bw (k>0.7) and this is reflected in the average k value of 0.61 which is slightly higher than the average for the control cross (k=0.58). The control in this case is an identical cross using bw chromosomes which had not been previously exposed to an SD chromosome. At first glance, the results indicate that some but not all of the bw chromosomes show some evidence in favor of contamination by Sd and that the distorting ability of these chromosomes is low relative to that of the original SD-5 or SD-72. The potentially contaminated chromosomes were retested for the heritability of this effect in subsequent generations. Three bw chromosomes showing high k-values (k>0.7) and one with a low k-value (k≈0.4) were tested in subsequent generations. There was a significant heritability of low and high k-values among the F1 progeny but the difference between the high and low lines tended to disappear in subsequent generations. The results indicate that the abnormal k-values, although heritable are not stably inherited.

A parallel set of experiments which tested the ability of target cn bw chromosomes, descended from SD-72/cn bw aged males, to induce self-distortion when heterozygous with the pr cn insensitive chromosome resulted in k-values that were close to 0.5 (data not shown). Contaminated chromosomes would show suicide behaviour when heterozygous with the insensitive pr cn tester chromosome. This result might reflect a lack of chromosomal contamination or it might be due to the presence of suppressors of SD on the pr cn tester chromosome.

II. Testing for chromosomal contamination in females. There is the formal possibility that the Sd-Rsp interaction occurs in heterozygous females as well as in heterozygous males, but that there is no detectable phenotypic effect in the case of females. If this were the case, the chromosomes which were heritably changed in the heterozygous females might show the phenotypic effects of this change in the male offspring of these females. From SD-72/cn bw females, cn bw (Rsp sensitive) chromosomes were recovered and tested for the ability to induce self-distortion when heterozygous, in progeny males, with an Sd+ insensitive chromosome (pr cn or Df(2R)14C). Among a total of 36 chromosomes examined, no unusually low k-values were observed (data not shown). The lack of self-distortion in this case is best explained by the assumption that the lack of observed phenotypic effect in female gametes does reflect the absence of Sd activity in heterozygous females.

III. Testing for chromosomal contamination using insensitive target chromosomes. Sd+ second chromosomes insensitive to SD were tested for the acquired ability to distort a sensitive homolog in males after having been in heterozygosis with an SD chromosome. Our prediction was that if transposition of
Sd occurred in the heterozygous parental males, this could be expressed phenotypically as a distorting ability of the contaminated chromosome in the offspring males. The use of insensitive target chromosomes has two advantages. First, since the Rsp site is "blocked" all of the non-SD chromosomes can be recovered. Secondly, if these chromosomes acquire an Sd element at another site, they will have the genotype Sd Rsp\(^1\) and so can be used directly to test for distortion against sensitive tester chromosomes.

The insensitive target chromosomes used were pr cn, a laboratory insensitive chromosome, and two deficiencies for the heterochromatic 2R centric region where Rsp has been localized. In addition, the R(Cy)-40 chromosome was used because it carries a Rsp\(^1\) allele which originated from an SD chromosome, and it also includes E(SD) and M(SD), two major enhancers of SD activity. The results are summarized in Table 2. Of the 37 pr cn chromosomes tested, after having been in heterozygosis with SD-5, none showed k-values greater than 0.65. Generally, the values were normally distributed about a mean value of 0.49. Those chromosomes which gave the most extreme values (highest or lowest) were re-tested in the following generation; there was no evidence for heritability of the more extreme values. These negative results could again be explained by postulating the presence of suppressors of SD on the pr cn chromosome. The problem of possible suppressors on the target chromosome can be circumvented by using the deficiencies of the Rsp region, Df(2R)14C, bw and Df(2R)2J, bw. The two deficiencies used have been shown to induce a significant degree of suicide behaviour in Sd Rsp\(^5\) chromosomes (Ganetsky 1977; Sharp et al. 1985).

Target Df(2R) chromosomes were recovered from Df(2R)/SD-5 heterozygotes and tested for their ability to distort the sensitive It bw tester chromosome. Again, there is no evidence that the majority of these chromosomes acquired a distorting ability subsequent to the exposure to SD chromosomes. There were however three chromosomes among the sixty tested which gave k-values greater than 0.65. When these three chromosomes, which gave an average k-value of 0.72 were re-tested in the following generation the average k-value for eight progeny lines was 0.57 and the range was from 0.50 to 0.67. This shows that there is some degree of heritability of these elevated k-values. The final test for chromosomal contamination in heterozygous males was performed using the R(Cy)-40 chromosome (Hartl 1980). This is an Sd\(^+\) chromosome that through recombination has acquired the centromeric region and right arm from the SD-36 chromosome. The presence of E(SD) and M(SD) on this chromosome should enhance their distorting ability if they acquire an Sd element from the SD chromosome. The mean k-value, after exposure to SD-5 and SD-MAD, of the 122 chromosomes tested, 0.52±0.57 was not significantly higher than the controls. Among this set, however, were several chromosomes with k-values greater than 0.65. The maximum k-value in the control is 0.63 whereas three experimental chromosomes showed values greater than 0.65. The chromosome which showed the highest k-value (0.75) was tested again in nine heterozygous F\(^1\) offspring males; there was a large variation in k-values among these progeny (range: 0.32-0.71) and one of the nine had a k-value that was again greater than 0.7. One possible explanation for this proportion of partially heritable high k-values would be the presence of an acquired but unstable Sd element. Because of the possibility that E(SD) might show some residual SD (or meiotic drive) activity (Sharp et al. 1985), we compared our results to a control carrying an identical R(Cy)-40 E(SD) chromosome, which had not been exposed to SD activity in the previous generation.

Our proposal for an underlying mechanism for segregation distortion suggests that the Sd element was originally inserted into SD chromosomes by a genetic transposition event in the past, and, moreover, that the distortion we observe is caused by high-frequency site-specific transposition of copies of this element into sensitive homologous chromosomes.

In our experiments, a few combinations gave evidence of having acquired distorting ability. In no case did we succeed in isolating a line which repeatedly, over many generations, gave high levels of distortion. These results indicate to us that the chromosome contamination effect we were looking for may occur, but at low frequency and that the altered chromosomes are genetically unstable.

The major difficulty in demonstrating chromosomal contamination by copies of an Sd element is analogous to the problem of isolating dominant lethal mutations. That is, the event of interest may take place at detectable frequencies but one cannot recover the mutant products. In the case of the SD system, if normal transposition is into the Rsp site, we can only hope, in these experiments, to recover secondary transpositions into other chromosomal sites. Although the model predicts very high levels of transposition and insertion into the Rsp\(^5\) site, we have no way of predicting the rate of insertion at other sites. Secondly, because of the nature of our biological assay for the presence of Sd, many possible insertions would go undetected. Sd insertions which did not subsequently lead to high levels of transposition into the Rsp site of the tester chromosome would go undetected. In addition, it is not unreasonable to suspect that the levels of SD activity may be subject to position effects. Given these considerations, the results we obtained here are encouraging.
Acknowledgements: We are grateful to Drs. D. Hartl, A. Hilliker and R. Temin and to the Mid-American Drosophila Stock Center for providing Drosophila strains. This work was supported by NSERC Canada and by the Minister della Pubblica Istruzione Italiano. The experimental work was carried out at Carleton University while the senior author was on a leave of absence from the Dipartimento di Genetica e Biologia Molecolare, Università di Roma.


Maiti, A.K. and A.K. Ghosh. University of Calcutta, India. Hybrid dysgenesis in Drosophila ananassae. Hybrid dysgenesis is a syndrome of correlated genetic traits that is spontaneously induced in hybrids between certain mutually interacting strains, usually in one direction only (Kidwell 1979). Dysgenic traits are: mutation, chromosomal aberration, distorted segregation, sterility, etc. Using sterility as an indicator of hybrid dysgenesis, we have tested some long established laboratory strains and some newly caught wild type strains of D. ananassae to see whether or not similar type of dysgenesis occurs in the hybrids of these strains like that of D. melanogaster. Twelve newly collected strains (from different regions of India) were tested within which two strains behaved like inducer or P-strain.

<table>
<thead>
<tr>
<th>Table 1. Hybrid sterility test.</th>
</tr>
</thead>
<tbody>
<tr>
<td>F1-Hybrid</td>
</tr>
<tr>
<td>-----------</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>0085 ♂ X Belur ♀</td>
</tr>
<tr>
<td>0085 ♀ X Belur ♂</td>
</tr>
<tr>
<td>a-Cal ♂ X Garia ♀</td>
</tr>
<tr>
<td>a-Cal ♀ X Garia ♂</td>
</tr>
</tbody>
</table>

On examining the gonads of the complete sterile female were observed that they were of dysgenic form. If such dysgenesis is due to the presence of some P-element like factors, like that of D. melanogaster, then it can be concluded that D. ananassae must conserve the P-element in its genome in the course of evolution.


Manousis, T.H. and C.D. Kastritis. University of Thessaloniki, Greece. Electrophoretic analysis of polypeptide and mucopolsaccharide content of several tissues and organs of Drosophila auraria larvae. In the course of an investigation dealing with gene activities in Drosophila auraria, we have included the study of the polypeptides, and PAS-positive polypeptides of several late 3rd instar and prepupal tissues and organs. The study was carried out by SDS polyacrylamide gel electrophoresis followed by PAS and C.B. staining. PAS-positive polypeptides are tissue specific and are different from those of the salivary glands of the same animal at the developmental stages studied (Manousis & Kastritis, submitted). Some quantitative and qualitative changes of the polypeptide content can be observed during stages expected to coincide with a high titre of ecdysterone in the hemolymph. Hemolymph contains a group of polypeptides (Fig. 1. Ha) present in the fat tissue (Fig. 1, Fb) which in the case of other diptera produces the dominant polypeptides of the hemolymph.

Acknowledgements: This work was supported by a grant from Volkswagenwerk Stiftung to C.D.K.
Figure 1. Electrophoretic profiles of polypeptides of tissues and organs of 20 individuals per sample, after PAS-staining followed by C.B. staining. The arrows indicate the position of the PAS-positive polypeptides which in the case of the guts are shown, as an example, on the PAS stained gel (Gb). The numbers indicate the developmental stages as defined by Scouras & Kastritsis, L. whole larva, F. food extract, H. Hemolymph, FB. Fat Body, BW. Body Wall, B. Brains, MT. Malpighian Tubules, Ga. Guts after C.B. staining, Gb. Guts after PAS-staining of the same gel.

Mather, W.B. and R. Casu. University of Queensland, St. Lucia, Australia. Inversions from Phuket, Thailand. 8th Report.

In January 1984 twenty-five isolines of D.s.albostrigata and two isolines and two isolines of D.albomicans were established from Phuket, Thailand. The inversions from the region were last reported on from a collection made in July 1984 (Mather & Pope, DIS 60:143).

(a) D.s.albostrigata. Six simple and one complex inversion were detected (Table). All inversions had previously been detected from Southeast Asia but X2 was new to Phuket. The heterozygosity frequency of all inversions detected is given in the Table.

(b) D.albomicans. Five simple inversions, E on chromosome II L and B6, C1, L3 on chromosome III were detected. All had previously been recorded from Phuket.

The material was collected and the isolines established by W.B.M. The laboratory work was carried out by R.C.

Mather, W.B. and R. Casu. University of Queensland, St. Lucia, Australia. Inversions from Phuket, Thailand. 9th Report.

In July 1984 twenty-seven isolines of D.s.albostrigata, eight isolines of D.albomicans and one isolate of D.kohkoa were established from Phuket, Thailand. The inversions from this region were last reported on from a collection made in January 1984 (Mather & Casu, DIS this issue).

(a) D.s.albostrigata. Five simple and two complex inversions were detected (Table 1).

Six of the seven inversions had previously been detected from Phuket. A photograph of a new inversion (D7) is presented with breakpoints 26.7-38.0 assigned in relation to the standard photographic map (Thongmeearkom 1977, DIS 52:154).

The heterozygosity frequency of all inversions detected is given in Table 1.
Table 1.

<table>
<thead>
<tr>
<th>Inversion</th>
<th>Chromosome</th>
<th>Simple Complex</th>
<th>Het. Freq.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A5</td>
<td>II L</td>
<td>x</td>
<td>66.7</td>
</tr>
<tr>
<td>D5</td>
<td>II L</td>
<td>x</td>
<td>3.7</td>
</tr>
<tr>
<td>C5</td>
<td>II R</td>
<td>x</td>
<td>66.7</td>
</tr>
<tr>
<td>C1</td>
<td>III</td>
<td>x</td>
<td>51.5</td>
</tr>
<tr>
<td>F3</td>
<td>III</td>
<td>x</td>
<td>3.7</td>
</tr>
<tr>
<td>W2</td>
<td>III</td>
<td>x</td>
<td>63.0</td>
</tr>
<tr>
<td>D7</td>
<td>III</td>
<td>x</td>
<td>3.7</td>
</tr>
</tbody>
</table>

Table 2.

<table>
<thead>
<tr>
<th>Inversion</th>
<th>Chromosome</th>
<th>Simple Complex</th>
</tr>
</thead>
<tbody>
<tr>
<td>E'</td>
<td>II L</td>
<td>x</td>
</tr>
<tr>
<td>C1</td>
<td>III</td>
<td>x</td>
</tr>
<tr>
<td>E3</td>
<td>III</td>
<td>x</td>
</tr>
<tr>
<td>E6</td>
<td>III</td>
<td>x</td>
</tr>
<tr>
<td>L3</td>
<td>III</td>
<td>x</td>
</tr>
<tr>
<td>T4</td>
<td>III</td>
<td>x</td>
</tr>
</tbody>
</table>

**Figure.** The free end of the chromosome is to the right.

(b) *D. albomicans.* Four simple and two complex inversions were detected (Table 2). All had been detected from southeast Asia but F3 was new to Phuket.

(c) *D. kohkoa.* The one isolate analyzed proved to be inversion free.

The material was collected and the isolines established by W.B.M. The laboratory work was carried out by R.C.

Mather, W.B. and R. Casu. University of Queensland, St. Lucia, Australia. Inversions from Phuket, Thailand. 10th Report.

In January 1985 twenty isolines of *D. albostrigata* and nineteen isolines of *D. albomicans* were established from Phuket, Thailand. The inversions from this region were last reported on from a collection made in July 1984 (Mather & Casu, DIS 63 this issue).

(a) *D. albostrigata.* Seven simple and two complex inversions were detected (Table 1). All the inversions had previously been detected from Southeast Asia but G and Q5 were new to Phuket. The heterozygosity frequency of all inversions detected is given in Table 1.

(b) *D. albomicans.* Seven simple and two complex inversions were detected (Table 2). Of these J6 and W5 were new to Phuket. A photograph of a new inversion (E7) is presented with breakouts 19.7-24.2 assigned in relation to the standard photographic map (Mather & Thongmeearkom 1980, DIS 55:101). The heterozygosity frequency of all inversions detected is given in Table 2.
In July 1985 twenty-seven isolines of *D. albostrigata* and three isolines of *D. albomicans* were established from Chiang Mai, Thailand. Inversions in these species were last reported on from Chiang Mai in July 1984 (Mather & Pope, DIS 61:116).

(a) *D. albostrigata*. Five simple and two complex inversions were detected (Table). All inversions had previously been detected from Chiang Mai. The heterozygosity frequency of all inversions detected is given in the Table.

(b) *D. albomicans*. Three simple inversion (E’, S5 and A7) on chromosome II L were detected. All had previously been detected from Chiang Mai.

The material was collected and the isolines established by W.B.M. The laboratory work was carried out by A.K.P.

Table 1.

<table>
<thead>
<tr>
<th>Inversion</th>
<th>Chromosome</th>
<th>Simple Complex</th>
<th>Het. Freq.</th>
</tr>
</thead>
<tbody>
<tr>
<td>A5</td>
<td>II L</td>
<td>x</td>
<td>48.1</td>
</tr>
<tr>
<td>E</td>
<td>II L</td>
<td>x</td>
<td>14.8</td>
</tr>
<tr>
<td>C7</td>
<td>II L</td>
<td>x</td>
<td>3.7</td>
</tr>
<tr>
<td>D5</td>
<td>II L</td>
<td>x</td>
<td>3.7</td>
</tr>
<tr>
<td>C5</td>
<td>II R</td>
<td>x</td>
<td>7.4</td>
</tr>
<tr>
<td>C1</td>
<td>III</td>
<td>x</td>
<td>3.7</td>
</tr>
<tr>
<td>B5</td>
<td>III</td>
<td>x</td>
<td>14.6</td>
</tr>
</tbody>
</table>

In July 1985 nineteen isolines of *D. albostrigata* and twenty-three isolines of *D. albomicans* were established from the River Kwai region of Thailand. The inversions from the region were last reported on from a collection made in July 1983 (Mather & Pope, DIS 60:143).

(a) *D. albostrigata*. Five simple and one complex inversion were detected. All inversions have been previously found at the collection site. The heterozygosity frequency of the inversions detected is given in Table 1.

(b) *D. albomicans*. Five simple and one complex inversion were detected. All of the inversions had previously been found at the River Kwai. The heterozygosity frequency of the inversions detected is given in Table 2.

The material was collected and the isolines established by W.B.M. The laboratory work was carried out by A.K.P.

Table 2.

<table>
<thead>
<tr>
<th>Inversion</th>
<th>Chromosome</th>
<th>Simple Complex</th>
<th>Het. Freq.</th>
</tr>
</thead>
<tbody>
<tr>
<td>R5</td>
<td>I</td>
<td>x</td>
<td>8.7</td>
</tr>
<tr>
<td>E’</td>
<td>II L</td>
<td>x</td>
<td>8.7</td>
</tr>
<tr>
<td>S5</td>
<td>II L</td>
<td>x</td>
<td>39.1</td>
</tr>
<tr>
<td>C1</td>
<td>III</td>
<td>x</td>
<td>17.4</td>
</tr>
<tr>
<td>L3</td>
<td>III</td>
<td>x</td>
<td>13.0</td>
</tr>
<tr>
<td>E6</td>
<td>III</td>
<td>x</td>
<td>67.6</td>
</tr>
</tbody>
</table>
Recombination in males of *D. ananassae* has been proved to be meiotic in origin (Matsuda et al. 1983) and to be controlled by a variety of genetic factors (Moriwaki et al. 1970; Hinton 1970, 1974, 1983; Moriwaki & Tobari 1975; Matsuda & Tobari 1983; Tobari et al. 1983). Two marker stocks, b pea; bri ru and b se; bri ru, and 3 wild stocks, TNG, L8 and HW, were used in the present experiment, because each stock carries different genetic factors controlling recombination in males (Matsuda & Tobari 1983). The F1 heterozygous males of 8 different genotypes were obtained by crossing marker females with wild males or vice versa, as shown in Fig. 1 legend. F1 heterozygotes were denoted as markers/wild or wild/markers according to whether the marked chromosomes were derived from their mothers or fathers, respectively. F1 males exposed to four different temperatures, 18°C, 22°C, 25°C and 29°C during the entire life cycle were obtained to test the effect of temperature on recombination in males.

The 10-25 F1 males from each cross at each temperature were mated to 3-day old marker females individually at 22°C, and, after 4 days, the flies were transferred to new vials to let them lay eggs for another 4 days. Progeny from each vial were scored for recombination up to 20 days after egg laying.

Average recombination frequency in a given genotype is plotted at a given temperature in Fig. 1. Map distances for b-pea and b-se in chromosome 2 and bri-ru in chromosome 3 are about 15, 60, and 100, respectively. Only a few of F1 males emerged from the cross b pea; bri ru x TNG cf at 18°C, and very rare progeny were obtained from F1 males of b pea/+(HW); bri ru/+(HW), +(HW)/b pea; +(HW)/bri ru and +(TNG)/b pea; +(TNG)/bri ru at 29°C.

As shown in Fig. 1, elevated temperature enhanced meiotic crossing-over in males of *D. ananassae*. However, the effects of temperature were quite different between reciprocal F1 males.

These results have led us to the experiment to localize the time of crossing-over in spermatocytes.

**Acknowledgements:** This study, supported by the Yamada Science Foundation, was done largely in the laboratory of Prof. James F. Crow whose generosity in giving help to the author is sincerely appreciated.


Matsuda, M. Tokyo Metropolitan University, Tokyo, Japan. The effect of temperature on recombination in males of *D. ananassae*.
As part of an ongoing study of the reproductive behavior of Drosophila, we have collected flies at various locations in the Philadelphia area. These surveys were conducted during the spring and summer of 1983 and the fall of 1985, in several suburban locations approximately 17 miles west of Philadelphia.

Our traps were clear plastic cups, baited with banana and live yeast or pumpkin and live yeast, that were suspended from trees or, in one case, an apartment balcony (see McRobert & Tompkins 1983, for a description of the trapping procedure). Flies were identified immediately after capture except for some females that appeared to be from species that could only be identified by keying males. In these cases, the females were maintained individually on cornmeal-molasses medium until their offspring emerged and the males could be identified. After identification, the flies were either killed, used to start laboratory cultures or preserved in alcohol.

Table 1. Species trapped in the Philadelphia area.

<table>
<thead>
<tr>
<th>Species</th>
<th>Spring-Summer</th>
<th>Fall</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>D. affinis</td>
<td>203</td>
<td>3</td>
<td>206</td>
</tr>
<tr>
<td>D. melanogaster</td>
<td>71</td>
<td>0</td>
<td>71</td>
</tr>
<tr>
<td>D. immigrans</td>
<td>1</td>
<td>69</td>
<td>70</td>
</tr>
<tr>
<td>D. duncanii</td>
<td>8</td>
<td>32</td>
<td>40</td>
</tr>
<tr>
<td>D. robusta</td>
<td>19</td>
<td>6</td>
<td>25</td>
</tr>
<tr>
<td>D. tripunctata</td>
<td>14</td>
<td>2</td>
<td>16</td>
</tr>
<tr>
<td>D. quinaria</td>
<td>14</td>
<td>0</td>
<td>14</td>
</tr>
<tr>
<td>D. melanica</td>
<td>11</td>
<td>0</td>
<td>11</td>
</tr>
<tr>
<td>D. busckii</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

Two aspects of these data are of interest. First, for all species except D. busckii, in which only a single male was caught, the numbers of males and females collected were approximately equal. Second, the distribution of species was seasonal. D. affinis and D. melanogaster were the most common species in the spring and summer months, while D. immigrans was the most common species in the fall.

Acknowledgement: This research was supported by NIH Grant GM 33511 awarded to L.T.


Although biological research has been conducted in Woods Hole for many years, we know of no descriptions of Drosophila species that are indigenous to this area. Therefore, during a recent stay, we surveyed the drosophilids at various sites on the grounds of the Marine Biological Laboratory (MBL).

Flies were trapped in plastic cups that were baited with banana and live yeast, then suspended from trees (McRobert & Tompkins 1983). Twenty collections were made between July 22 and August 7, 1985. In most cases, the flies were immediately keyed. However, since D. affinis and D. algonquin females cannot be distinguished, females that were from one of these two species were maintained individually on Carolina Instant Medium until their offspring emerged, at which time the males were keyed.

Surprisingly, in light of the fact that all of the traps were in windy areas within 300 yards of the ocean, all but one of the seven traps that we set attracted flies. Most of the flies that we collected from these traps are listed in Table 1. We also caught approximately 50 D. melanogaster males and females. However, since students in one of the MBL courses released laboratory flies into the environment, we do not know whether this species is indigenous.

Acknowledgement: This research was supported by NIH Grant GM33511 awarded to L.T.


Table 1. Species trapped in Woods Hole, Massachusetts.

<table>
<thead>
<tr>
<th>Species</th>
<th>No. Individuals Collected</th>
</tr>
</thead>
<tbody>
<tr>
<td>D. affinis</td>
<td>68</td>
</tr>
<tr>
<td>D. busckii</td>
<td>5</td>
</tr>
<tr>
<td>D. immigrans</td>
<td>4</td>
</tr>
<tr>
<td>D. algonquin</td>
<td>3</td>
</tr>
<tr>
<td>D. robusta</td>
<td>3</td>
</tr>
<tr>
<td>D. quinaria</td>
<td>1</td>
</tr>
<tr>
<td>D. melanica</td>
<td>1</td>
</tr>
</tbody>
</table>


Acknowledgement: This research was supported by NIH Grant GM33511 awarded to L.T.

The base of the X chromosome from maroonlike to suppressor of forked has been particularly well characterized in terms of contiguous lethal complementation groups, most of which have been unambiguously ordered (Schalet & Lefevre 1976; Lefevre 1981; Kramers et al. 1983). There are, however, some pairs of complementation groups in region 19F in which the proximal-distal orientation of lethals or visibles remains unresolved. These are pairs [if1/B214], [sol/slg] and [A112/LB20] (Fig. 1).

Small optic lobes (sol) and sluggish (slg) are two visible complementation groups (Fischbach & Heisenberg 1981; Gysen & Benzer, unpubl.) which have recently been mapped into this region (Miklos, Kelly, Coombe, Leeds & Lefevre, unpubl.).

The major part of the mal-su(f) region has now also been microcloned, and a large number of clones have been assigned to single complementation groups (Miklos, Davies, Yamamoto & Pirrotta, unpubl.). However, those clones which have fallen into the pairs of unordered complementation groups remain unassigned at present. Finer localization of such clones would be possible if the order of complementation groups within a pair could be determined by chromosomal rearrangements.

During their studies on hycanthone methanesulphonate (HMS)-induced recessive lethal mutations, Kramers et al. (1983) found that HMS was very effective in inducing multilocus deficiencies in the proximal part of the X chromosome.

We have tested a number of these HMS-induced deficiencies to further refine their genetic breakpoints and report that a large deficiency, Df(1)HM44, has a proximal breakpoint between lethal complementation groups A112 and LB20, thus unambiguously orienting these two loci. The previous tentative order of LB20-A112 was based on deficiencies whose breakpoints required additional characterization (Lefevre 1981). When tested for allelism with various deficiency-bearing chromosomes as well as reference lethals and visibles, Df(1)HM44 yielded the following results (Fig. 1). It was allelic to Df(1)mal6, Df(1)mal8, Df(1)mal10, Df(1)16-3-35, and Df(1)T2-14A. It complemented with Df(1)JC4, Df(1)sulf4B, Df(1)JA27, and was covered by both y*Ymah+ and y*Ymah106. Furthermore, Df(1)HM44 did not complement alleles at the following loci: K(1)17-234, K(1)17-457, mel, K(1)16-398, mel, leg, K(1)R-9-29, K(1)R-9-28, if1, I(1)B214, I(1)W-2, sol and I(1)A112. It did however complement alleles of K(1)34, K(1)LB20, eo and K(1)114. Thus its proximal breakpoint separates K(1)A112 and K(1)LB20, whereas its distal breakpoint has yet to be more precisely localized.

We have also examined a number of alleles of the complementation groups near to the proximal breakpoint and these have behaved as expected. lethals 11P1 and 17-62, which are alleles of K(1)A112, were included within Df(1)HM44, whereas lethal DA618, an allele of K(1)LB20, was excluded from Df(1)HM44. Df(1)HM44 has already been most useful to us in mapping clones deep in region 19F. Its proximal breakpoint is

![Figure 1](image)

**Figure 1.** The genetic boundaries of Df(1)HM44 as determined by complementation tests involving the y*Ymah106 duplication, and reference lethals and visibles in divisions 19 and 20. Alleles of the tested lethal and/or visible complementation groups which fall into Df(1)HM44 are indicated by closed circles, those which complement this deficiency are shown as open circles. It should be noted that Df(1)JA27 is a double deficiency, with another deletion in the 1B45; 1D3-2 area.
breakpoint should also provide a landmark for future chromosomal walks in this area.

Finally, two further points need to be made. First, some additional visible and lethal complementation groups (or complex loci) may still need to be added to the 19-20 region as more data become available. \(1(1)EC235\) is a new lethal complementation group in 19E (Fig. 1) and there may well be an additional lethal between EC235 and If (Miklos, unpubl.). Tumorous head (Pyati 1976) and \(1(1)EA41\) (Lefevre, unpubl.) are two further mutants awaiting accurate assignations. Second, it should be remembered that \(su(f)Pb\) and \(su(f)\) are not a pair of complementation groups. While \(1(1)R-9-18\) and \(1(1)3DES\) are indeed two lethal complementation groups in the \(su(f)\) complex, their situation is very probably an example of allelic complementation.

Acknowledgements: We thank Professors G. Lefevre, E. Munoz, S. Benzer and K. Fischbach for the provision of stocks.


Moltó, M.D. and M.J. Martínez-Sebastián. University of Valencia, Spain. Gene arrangements in polytene chromosomes of Drosophila guanche differing from standard arrangements in Drosophila subobscura. Drosophila guanche, which is endemic to Laurel forest of the Canary Island, is a member of the Obscura group. The karyotype of Drosophila guanche resembles that of Drosophila subobscura which is thought to have the karyotype most closely

Figure 1. Chromosomes of Drosophila guanche showing characteristic inversions. A = chromosome A; B = chromosome J; C = chromosome E; D = chromosome O.
resembling the ancestral form of the genus Drosophila. Both have five pairs of acrocentric or subacrocentric chromosomes (Prevosti 1976) and a pair of point chromosomes (Krimbas & Loukas 1984).

Compared to the standard gene arrangements in D. subobscura, D. guanche shows characteristic inversions in most of its chromosomes.

**CHROMOSOME A** (Fig. 1A). In the distal part of this chromosome, D. guanche has two inversions. The first is a small and is located in the section 16BCD (Krimbas & Loukas 1984). The second is larger and involves the region between 10C and 13A/B subsections.

The proximal part of this sex chromosome (see Fig. 1A labeled by ), also shows many differences with that in D. subobscura. Because there are so many differences in this region, it is difficult to make correlations relative to this area between the two species. One of the changes which probably occurred between the two species is the inversion A1, an inversion characteristic of D. subobscura.

**CHROMOSOME J** (Fig. 1B). This chromosome shows one inversion involving the region between 30A and 34E subsections. Krimbas & Loukas (1984) also indicate the existence of an inversion in this part of the J chromosome but give the boundaries as subsections 31 and 34A.

**CHROMOSOME U**. In agreement with Krimbas & Loukas (1984), the U chromosome of D. guanche carries the U1+2 gene arrangement of D. subobscura.

**CHROMOSOME E** (Fig. 1C). Figure 1C shows that the E chromosome of D. guanche has two inversions. The first one consists of the region between 59D and 66C/D subdivisions. The second one includes the region between 67C and 72B/C.

**CHROMOSOME O** (Fig. 1D). This chromosome carries two overlapping inversions: the O3 arrangement, also present in D. subobscura, and another which includes the region between 84D/85A and 91AB+94. These inversions have been detected by Krimbas & Loukas (1984), though they give different boundaries for this second inversion.

In conclusion, D. guanche has the following altered gene arrangements compared to those of the standard in D. subobscura: A(A1 + several specific inversions), (inv. 10C-13A/B) and (inv. 16BCD); J (inv. 30A-34E); U(U1+2); E (inv. 59D-66C/D) and (inv. 67C-72B/C); O (O3) and (inv. 84D/85A-91AB+94).


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The polymorphism of Aldox in Mediterranean populations of Drosophila simulans.

Tris-citrate II buffer system of Selander (1971), thus allowing us to separate more segregating alleles than the two previously observed in European and African populations by Cabrera et al. (1982) and Hyytia et al. (1985). The staining procedure was adapted from Ayala et al. (1972).

Figure 1 shows zymograms of single-fly homogenates. Alleles are numbered according to their relative mobility referring to that of the more common allele (100).

The allelic frequency pattern is rather close in our three samples (Table 1): three common alleles (100, 112, 118) are present in the three populations; the 106 and nul alleles are rare but detected in all populations.

![Figure 1. Aldox variants](image_url)
samples; the 76 and 124 rare alleles were only detected in the French sample. However, Chi-squared tests performed between pairs of samples show that allelic frequencies are significantly different between populations (Table 2).

Unfortunately, a direct comparison with the Aldox variants, described by Steiner et al. (1976) in Hawaiian populations and by Kojima et al. (1970) in a Texas population, is impossible at the moment; nevertheless, the frequency distribution pattern of alleles in these populations is very different from the Mediterranean pattern.

References:

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**Table 1.** Allelic frequencies at the Aldox locus in the investigated populations (± 1.96 s.e.). (n = no. of gametes assayed, for rare alleles limits of the 5% confidence interval are given in brackets.)

<table>
<thead>
<tr>
<th>Allele</th>
<th>La Sire 82</th>
<th>Barcelone 82</th>
<th>Nasrallah 83</th>
</tr>
</thead>
<tbody>
<tr>
<td>76</td>
<td>0.001(0-0.009)</td>
<td>0.000</td>
<td>0.000</td>
</tr>
<tr>
<td>100</td>
<td>0.499 ± 0.038</td>
<td>0.572 ± 0.033</td>
<td>0.601 ± 0.038</td>
</tr>
<tr>
<td>106</td>
<td>0.022 ± 0.011</td>
<td>0.053 ± 0.015</td>
<td>0.008(0.003-0.018)</td>
</tr>
<tr>
<td>112</td>
<td>0.233 ± 0.032</td>
<td>0.121 ± 0.022</td>
<td>0.166 ± 0.029</td>
</tr>
<tr>
<td>118</td>
<td>0.239 ± 0.032</td>
<td>0.252 ± 0.029</td>
<td>0.220 ± 0.032</td>
</tr>
<tr>
<td>124</td>
<td>0.003(0-0.012)</td>
<td>0.000</td>
<td>0.00</td>
</tr>
<tr>
<td>NUL</td>
<td>0.003(0-0.012)</td>
<td>0.001(0-0.007)</td>
<td>0.003(0-0.012)</td>
</tr>
</tbody>
</table>

**Table 2.** Chi 2 tests (3 d.f.) for allelic frequency homogeneity between populations.

<table>
<thead>
<tr>
<th>Ls 82/</th>
<th>Ls 82/</th>
<th>Barcelone 82/</th>
<th>Nasrallah 83/</th>
<th>Nasrallah 83/</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chi 2</td>
<td>40.383***</td>
<td>18.309***</td>
<td>29.758***</td>
<td></td>
</tr>
</tbody>
</table>

*** sig. 0.01%

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**Najera, C.** University of Valencia, Spain.

Study of eye colour mutant variability in natural populations of *D. melanogaster.*

III. Pine-Wood.

Two samples of *D. melanogaster* were captured in a pine-wood at La Canada (Valencia, Spain), in two different seasons of the year: autumn and spring. The purpose was to search for eye colour mutations by inbreeding through F\textsubscript{1} pair matings from the collected females, as in previous works made in cellar (Najera & Mensa 1985) and vineyard (Najera 1985) populations.

The number of heterozygous females for eye colour mutation was 17/45 (37.7\%) in autumn and 24/67 (35.8\%) in spring. The mean number of mutations per fly was 0.40 in both populations. Adding the results of both captures, 36.61\% of the females were heterozygous and there were 0.40 mutations per fly in the pine-wood.

The distribution of mutations was the following: females with 1 mutation (autumn: 16, spring: 21), and females with 2 mutations (autumn: 1, spring: 3). Both fit a Poisson distribution ($X^2 = 0.469$ ns; $X^2 = 0.319$ ns).

The percentage of heterozygous loci for eye colour mutants was 12.50 (autumn population) and 18.75 (spring population).

The overall frequency of allelism was 10.9±3.9 (7.64) for the autumn population; 6.0±2.0 (9/151) for the spring population and 9.2±2.0 (20/217) interpopulational.

The distribution of alleles in both populations was at random ($X^2 = 0.895$ ns; $X^2 = 0.052$ ns).

It seems that the pine-wood population is more stable since the proportion of eye colour mutations as well as the proportion of sexes (Najera '85) found in both captures (autumn and spring) were similar.


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**Najera, C.** University of Valencia, Spain.

Study of eye colour mutant variability in natural populations of *D. melanogaster.*

IV. Comparison between populations and allelisms.

The percentages of heterozygous females for eye colour mutations and the total number of mutations found in the populations subject of preceding works were compared by means of the ANOVA test. Table 1 shows that the number of mutations and of heterozygous females are higher in cellar populations than in the others; with significant differences between the habitats, but on the contrary, no significant differences appear between the two seasons of the year.
Table 1. Percentage of heterozygous females and number of mutations for eye colour in natural populations.

<table>
<thead>
<tr>
<th></th>
<th>CELLAR Spring</th>
<th>CELLAR Autumn</th>
<th>VINEYARD Spring</th>
<th>VINEYARD Autumn</th>
<th>PIN-WOOD Spring</th>
<th>PIN-WOOD Autumn</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANALYZED FEMALES</td>
<td>68</td>
<td>51</td>
<td>40</td>
<td>70</td>
<td>45</td>
<td>40</td>
</tr>
<tr>
<td>Heterozygous females</td>
<td>36 (52.94%)</td>
<td>13 (25.49%)</td>
<td>23 (32.85%)</td>
<td>17 (37.77%)</td>
<td>24 (35.82%)</td>
<td></td>
</tr>
<tr>
<td>Mutations</td>
<td>42 (62%)</td>
<td>13 (25%)</td>
<td>27 (39%)</td>
<td>16 (40%)</td>
<td>27 (40%)</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Percentage of heterozygous females and number of morphological eye mutations in natural populations.

<table>
<thead>
<tr>
<th></th>
<th>CELLAR Spring</th>
<th>CELLAR Autumn</th>
<th>VINEYARD Spring</th>
<th>VINEYARD Autumn</th>
<th>PIN-WOOD Spring</th>
<th>PIN-WOOD Autumn</th>
</tr>
</thead>
<tbody>
<tr>
<td>ANALYZED FEMALES</td>
<td>68</td>
<td>51</td>
<td>40</td>
<td>70</td>
<td>45</td>
<td>40</td>
</tr>
<tr>
<td>Heterozygous females</td>
<td>6 (8.82%)</td>
<td>3 (5.88%)</td>
<td>3 (4.29%)</td>
<td>1 (2.22%)</td>
<td>4 (5.97%)</td>
<td></td>
</tr>
<tr>
<td>Total mutations</td>
<td>48</td>
<td>16</td>
<td>30</td>
<td>19</td>
<td>31</td>
<td></td>
</tr>
<tr>
<td>Morphol. eye mut.</td>
<td>6 (12.50%)</td>
<td>3 (18.75%)</td>
<td>3 (10.00%)</td>
<td>1 (5.26%)</td>
<td>4 (12.90%)</td>
<td></td>
</tr>
</tbody>
</table>

Table 3. Frequency of intra- and interpopulational allelic crosses.

<table>
<thead>
<tr>
<th></th>
<th>CA</th>
<th>CS</th>
<th>VA</th>
<th>VS</th>
<th>PA</th>
<th>PS</th>
</tr>
</thead>
<tbody>
<tr>
<td>CA</td>
<td>29/381</td>
<td>66/497</td>
<td>90/902</td>
<td>18/230</td>
<td>59/558</td>
<td>34/342</td>
</tr>
<tr>
<td></td>
<td>0.076±0.014</td>
<td>0.133±0.014</td>
<td>0.100±0.010</td>
<td>0.078±0.018</td>
<td>0.099±0.014</td>
<td>0.099±0.016</td>
</tr>
<tr>
<td></td>
<td>0.076±0.014</td>
<td>0.078±0.018</td>
<td>0.1066±0.014</td>
<td>0.099±0.016</td>
<td>0.063±0.010</td>
<td>0.063±0.010</td>
</tr>
<tr>
<td></td>
<td>0.078±0.018</td>
<td>0.1066±0.014</td>
<td>0.099±0.016</td>
<td>0.063±0.010</td>
<td>0.063±0.010</td>
<td></td>
</tr>
<tr>
<td></td>
<td>0.1066±0.014</td>
<td>0.099±0.016</td>
<td>0.063±0.010</td>
<td>0.063±0.010</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Both, from a Student-Newman-Keuls multiple range test and from a "t" test (cellar-vineyard t = 3.95***; cellar-pine-wood t = 2.57**; vineyard-pine-wood t = 1.24 ns), it can be concluded that the differences between habitats are due to a higher number of heterozygous females in the cellar populations. Morphological eye mutations were also detected, and there are no significant differences as regards habitats or seasons for this type of mutation (Table 2).

To carry out inter- and intrapopulational allelism tests, the crosses were made using phenotypes (so that the total number of crosses is reduced from 15931 to 6909) on the basis that dark eyes and light eyes possibly block different metabolic pathways and that it is difficult to find alleles between them, and on the other hand that caramel eyes normally affect both pathways at the same time and even the deposition of pigment granules.

Although the estimated frequency of allelic crosses is not the actual populational frequency but a higher value, the results are useful for comparing the different populations, because in all cases the same method was used (Table 3). The populations showing the highest number of allelic crosses between them
were CS (cellar spring), VS (vineyard spring) and PA (pine-wood autumn). These populations also showed the highest intrapopulational allelisms. On the contrary, CA (cellar autumn) and VA (vineyard autumn), compared to PS (pine-wood spring), were the populations which showed the lowest allelic crosses and intrapopulational allelisms. Both inter- and intrapopulational allelic crosses had the same range.

Considering the total number of different mutations in each population in relation to the total number of analyzed females in such populations as a more exact reflect of the variability, and performing an ANOVA test, differences between habitats, although not significant, can be observed. It can be suggested, therefore, that the allelism rate in the cellar is higher than in the other habitats, so that the variability decreases in the population in relation to different mutations. Altogether, these results seem to suggest that either we deal with a more endogamic and closer related population or selection is favouring a greater accumulation of mutations in this population.

By means of a factorial analysis of correspondances which reflects the 65% of the total variance, and drawing a tridimensional graph of the points corresponding to the six populations (Fig. 1), it can be observed that the closest populations are the cellar ones, the vineyard and the pine-wood populations remaining approximately at the same distance.

Najera, C. University of Valencia, Spain. Study of eye colour mutant variability in natural populations of D. melanogaster. V. Effective sizes and distances.

From a total of the six populations belonging to captures performed in three different habitats (cellar, vineyard and pine-wood), and in two seasons of the year (autumn and spring) subject of preceding works, a series of parameters were estimated. The effective sizes of these populations were estimated, using the "temporal method" of Krimbas & Tsakas (1971) and applying the estimator of Pollack (1983). The population sizes are rather big in the three populations, but slightly smaller in the cellar population (cellar-Ne = 12000; vineyard-Ne = 15000; pine-wood-Ne = 175000) which was predictable from the peculiar characteristics of this habitat.

### Table 1. Average heterozygosity in the six natural populations.

<table>
<thead>
<tr>
<th>Population</th>
<th>Lewontin &amp; Hubby</th>
<th>Nei</th>
</tr>
</thead>
<tbody>
<tr>
<td>CELLAR AUTUMN</td>
<td>0.010553 ± 0.004179</td>
<td>0.010502 ± 0.000003</td>
</tr>
<tr>
<td>CELLAR SPRING</td>
<td>0.011479 ± 0.003973</td>
<td>0.011505 ± 0.000004</td>
</tr>
<tr>
<td>VINEYARD AUTUMN</td>
<td>0.0111512 ± 0.005267</td>
<td>0.011602 ± 0.000001</td>
</tr>
<tr>
<td>VINEYARD SPRING</td>
<td>0.010590 ± 0.004112</td>
<td>0.010615 ± 0.000007</td>
</tr>
<tr>
<td>PINE-WOOD AUTUMN</td>
<td>0.014143 ± 0.006115</td>
<td>0.014272 ± 0.000006</td>
</tr>
<tr>
<td>PINE-WOOD SPRING</td>
<td>0.009539 ± 0.004137</td>
<td>0.009187 ± 0.000001</td>
</tr>
</tbody>
</table>

### Table 2. Genetic distances between natural populations.

<table>
<thead>
<tr>
<th>Distances</th>
<th>Cavalli Sneath</th>
<th>Nei</th>
<th>Rogers Prevosti</th>
</tr>
</thead>
<tbody>
<tr>
<td>CA-CS</td>
<td>0.2674</td>
<td>0.0066</td>
<td>0.00002</td>
</tr>
<tr>
<td>VA-VS</td>
<td>0.2113</td>
<td>0.0061</td>
<td>0.00003</td>
</tr>
<tr>
<td>PA-PS</td>
<td>0.2435</td>
<td>0.0061</td>
<td>0.00002</td>
</tr>
<tr>
<td>CA-VA</td>
<td>0.2567</td>
<td>0.0059</td>
<td>0.00003</td>
</tr>
<tr>
<td>CA-PA</td>
<td>0.2571</td>
<td>0.0067</td>
<td>0.00002</td>
</tr>
<tr>
<td>VA-PA</td>
<td>0.2145</td>
<td>0.0074</td>
<td>0.00004</td>
</tr>
<tr>
<td>CS-VS</td>
<td>0.2603</td>
<td>0.0061</td>
<td>0.00002</td>
</tr>
<tr>
<td>CS-PS</td>
<td>0.2516</td>
<td>0.0062</td>
<td>0.00002</td>
</tr>
<tr>
<td>VS-PS</td>
<td>0.2305</td>
<td>0.0056</td>
<td>0.00002</td>
</tr>
</tbody>
</table>
The average heterozygosity in the six populations was calculated following the estimations of Lewontin & Hubby (1966) and Nei (1978). The results are reflected in Table 1. The values are low, identical in both estimations and rather similar in all populations. The pine-wood populations were the most different; the autumn pine-wood registered the higher heterozygosity and the spring pine-wood populations the lowest.

To quantify the variation of the gene frequencies, the genetic distances between these populations were calculated using five indexes (Table 2). The Cavalli-Sforza index gave the largest distances. In general distances between populations are small and a few are larger in autumn than in spring. Nevertheless, this kind of variability does not seem to be adequate to calculate genetic distances between populations.


Nowak, J. and M.J. Piechowska. Institute of Biochemistry and Biophysics, Warsaw, Poland. Effect of environmental factors on glutamate dehydrogenase activity in D. melanogaster larvae. Since in some invertebrates, including insects, glutamate dehydrogenase (GDH) seems to be involved in the energy-linked metabolism (Bursell et al. 1976; Hansford et al. 1975; Storey et al. 1978), D. melanogaster larvae were subjected to such environmental stress conditions impairing this metabolism within the cells as: anaerobiosis, recovery from anaerobiosis, heat-shock, and starvation.

Materials and Methods. D. melanogaster, wild strain, was reared on the medium of Mitchell et al. (1968) supplemented with dried yeast (214 g per 1 liter of water), at 25°C, in a diurnal cycle of 12 hr light - 12 hr night photoperiod. Egg-laying periods of 4 hr were used to synchronize of larvae. Larvae of the third instar, i.e., at the stage when GDH activity is the highest (Nowak & Piechowska 1980), were used for experiments.

The larvae were subjected to the following treatments: (1) anaerobiosis under N2, for up to 2 hr; (2) recovery from 2 hr anaerobiosis for up to 60 min; (3) heat-shock at 37°C for up to 2 hr; (4) starvation for up to 3 hr. Larvae reared under standard conditions served as controls.

Mitochondria were isolated in 10 mM Tris buffer, pH 7.4, containing 60 mM sucrose, 240 mM mannitol, and 0.2 mM EGTA (Storey et al. 1978a), supplemented with 0.1% 2-mercaptoethanol. Nuclei and cell debris were removed by centrifugation of the homogenate at 2200 g for 6 min. The supernatant obtained was centrifuged at 7000 g for 10 min. The sedimented mitochondria were washed with the isolation buffer and 0.1 M potassium phosphate buffer, pH 7.8. Mitochondria were solubilized with 1% Lubrol PX to liberate the enzyme (GDH) from the matrix. Mitochondrial membranes were removed by centrifugation at 100,000 g for 30 min, and the supernatant obtained was used for the assays. All the isolation procedure was performed at 4°C.

GDH activity was determined both for the deamination and amination reactions at room temperature, by automatic recording of absorbance at 340 nm on a Varian-Cary 118C spectrophotometer, in 1 ml quartz cuvettes, at 1 cm light path. The reaction mixture contained substrates and coenzymes at optimum concentrations (for deamination 6 mM NAD, 10 mM L-glutamate; for amination 0.2 mM NADH, 6 mM 2-oxoglutarate, 300 mM NH4Cl) in 0.1 M potassium phosphate buffer, pH 7.8, and 50 μl of the enzyme preparation, corresponding to 50-70 μg protein (as determined according to Lowry et al. 1951).

Specific activity of the enzyme was expressed in micromoles of NAD or NADH transformed during 1 min of the reaction by 1 mg of the extract protein. Km glu was estimated from the Lineweaver-Burke plot (Segel 1975) for that range of the substrate concentration (1-10 mM) at which the plot was linear. The results were analyzed by Student's t test, assuming that the difference is statistically significant when p < 0.05.

Results and Discussion. In the reaction catalysed by glutamate dehydrogenase isolated from the D. melanogaster larvae subjected to anaerobiosis, i.e., when cellular metabolism is shifted to the glycolytic pathway, the level of deamination was lowered by 30% as compared with controls. This statistically significant decrease was first observed after 1 hr of anaerobiosis and remained at a practically unchanged level also after 2 hr (Table 1).

The decrease in GDH activity could be due to lowered level of L-glutamate in the cell as observed by Meyer (1980) for Callitroga macellaria larvae subjected to 4 hr anaerobiosis. Moreover, under conditions of anaerobiosis, there is an increase in the NADH:NAD ratio in the cell (Meyer 1980), and the appearing excess of NADH could inhibit GDH, an enzyme the activity of which is known to be regulated by reaction products (Bursell 1975; Storey et al. 1978).

Under these conditions, accumulation of the anaerobic metabolism products (alanine, pyruvate, lactate) observed in insects (Price 1963; Meyer 1978) could also lead to lowering of GDH activity.
Table 1. Effect of anaerobiosis on GDH activity in the deamination reaction.

<table>
<thead>
<tr>
<th>time of anaerobiosis (hours)</th>
<th>GDH activity (umoles/min/mg/protein)</th>
<th>number of exp.</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>0.356 ± 0.082</td>
<td>10</td>
<td>--</td>
</tr>
<tr>
<td>1/2</td>
<td>0.348 ± 0.067</td>
<td>5</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>1</td>
<td>0.234 ± 0.050</td>
<td>4</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>2</td>
<td>0.242 ± 0.069</td>
<td>12</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Table 2. Effect of recovery from anaerobiosis on GDH activity in deamination reaction.

<table>
<thead>
<tr>
<th>time of release from anaerobiosis (minutes)</th>
<th>GDH activity (umoles/min/mg/protein)</th>
<th>number of exp.</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>0.356 ± 0.082</td>
<td>10</td>
<td>--</td>
</tr>
<tr>
<td>0</td>
<td>0.242 ± 0.069</td>
<td>10</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>15</td>
<td>0.349 ± 0.051</td>
<td>3</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>30</td>
<td>0.271 ± 0.089</td>
<td>4</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>60</td>
<td>0.291 ± 0.033</td>
<td>4</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

Francesconi et al. (1969) demonstrated that pyruvate and alanine inhibited GDH activity in rat liver slices. Thus it can be expected that GDH from *D. melanogaster*, which in many respects resembles the enzyme of higher organisms (Bond & Sang 1968) could also be inhibited by these metabolites.

Thus, the pattern of anaerobiosis-induced changes in the enzyme activity indicates that the reactivity of GDH in the deamination reaction is dependent on functioning of the aerobic respiratory metabolism.

This dependence was confirmed in experiments on the activity of GDH isolated from larvae recovering after 2 hr anaerobiosis. As early as 75 min after the larvae had been brought back to aerobic conditions the level of deamination returned to control values (Table 2). Thus the resumed activity of the respiratory chain and the Kreb's cycle resulted in the return of the normal capacity of GDH to catalyse of 2-oxoglutarate, NADH and NH₄⁺.

Such a relationship between the level of deamination and functioning of cellular oxidative processes suggests that GDH could cooperate with this metabolic pathway, e.g., by supplying 2-oxoglutarate and/or NADH.

Studies on the activity of GDH isolated from larvae subjected to another type of stress conditions probably also disturbing the functioning of respiratory metabolism - the heat shock, demonstrated that after 30 minutes at 37°C the level of deamination reaction was statistically significantly, by about 25%, decreased as compared with control. In our experiments, on further duration of the shock (1 and 2 hr) the GDH activity ceased to be affected and returned to control value (Table 3). Leenders et al. (1974) suggested that heat-shock activated the respiratory metabolism. However, Meyer (1978) demonstrated that, in *Callitroga macellaria* larvae, both the increased temperature and anaerobiosis led to the appearance in the cells of the same set of compounds. Since in various organisms studied the response to heat-shock is very similar (Ashburner et al. 1979), it can be concluded that the increased temperature inhibits through a still unknown mechanism the aerobic respiratory metabolism. This would lead, in turn, to lowering of GDH activity in the first phase of heat-shock. On further duration of the shock, the energy providing metabolism may become unblocked, resulting in the recovery of the GDH activity observed after 1 and 2 hr of thermal stress.

The changes observed in GDH activity in the deamination reaction were not due to changed enzyme structure, as the $K_m$ glu remained at the control level throughout the time of the experiment (Table 4).

Starvation is another factor known to affect the respiratory activity of the cell. As demonstrated by Bosquet (1976) the respiratory metabolism in *Bombyx mori* larvae starved for 2 hr was decreased by 10%.
Table 7. Effect of various environmental factors on the GDH activity in the deamination reaction.

A. Anaerobiosis

<table>
<thead>
<tr>
<th>time of anaerobiosis (hours)</th>
<th>GDH activity (umoles/min/mg/protein)</th>
<th>number of exp. p</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>1.356 ± 0.331</td>
<td>14 --</td>
</tr>
<tr>
<td>1/2</td>
<td>1.410 ± 0.377</td>
<td>5 &gt;0.05</td>
</tr>
<tr>
<td>1</td>
<td>1.134 ± 0.393</td>
<td>4 &gt;0.05</td>
</tr>
<tr>
<td>2</td>
<td>1.294 ± 0.277</td>
<td>12 &gt;0.05</td>
</tr>
</tbody>
</table>

B. Recovery from anaerobiosis

<table>
<thead>
<tr>
<th>time of release from anaerob. (minutes)</th>
<th>GDH activity (umoles/min/mg/protein)</th>
<th>number of exp. p</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>1.356 ± 0.331</td>
<td>14 --</td>
</tr>
<tr>
<td>0</td>
<td>1.294 ± 0.277</td>
<td>12 &gt;0.05</td>
</tr>
<tr>
<td>15</td>
<td>1.521 ± 0.562</td>
<td>5 &gt;0.05</td>
</tr>
<tr>
<td>30</td>
<td>1.272 ± 0.453</td>
<td>6 &gt;0.05</td>
</tr>
<tr>
<td>60</td>
<td>1.099 ± 0.324</td>
<td>5 &gt;0.05</td>
</tr>
</tbody>
</table>

C. Heat-shock

<table>
<thead>
<tr>
<th>time of heat-shock (hr)</th>
<th>GDH activity (umoles/min/mg/protein)</th>
<th>number of exp. p</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>1.283 ± 0.315</td>
<td>17 --</td>
</tr>
<tr>
<td>1/2</td>
<td>0.933 ± 0.346</td>
<td>4 &gt;0.05</td>
</tr>
<tr>
<td>1</td>
<td>1.212 ± 0.344</td>
<td>7 &gt;0.05</td>
</tr>
<tr>
<td>2</td>
<td>1.082 ± 0.326</td>
<td>6 &gt;0.05</td>
</tr>
</tbody>
</table>

D. Starvation

<table>
<thead>
<tr>
<th>time of starvation (hr)</th>
<th>GDH activity (umoles/min/mg/protein)</th>
<th>number of exp. p</th>
</tr>
</thead>
<tbody>
<tr>
<td>control</td>
<td>1.242 ± 0.351</td>
<td>28 --</td>
</tr>
<tr>
<td>1/2</td>
<td>1.417 ± 0.149</td>
<td>6 0.05</td>
</tr>
<tr>
<td>1</td>
<td>1.303 ± 0.112</td>
<td>6 &gt;0.05</td>
</tr>
<tr>
<td>2</td>
<td>1.455 ± 0.299</td>
<td>5 &gt;0.05</td>
</tr>
<tr>
<td>3</td>
<td>1.368 ± 0.102</td>
<td>5 &gt;0.05</td>
</tr>
</tbody>
</table>

Such a small effect of starvation on mitochondrial metabolism, was not reflected in changes in the activity of GDH in the deamination reaction (Table 5) or in the $K_m$ value (Table 6). On the basis of these experiments as well as the effect of anaerobiosis on GDH activity (a 30% decrease of deamination despite complete blocking of the aerobic metabolism; cf Table 1), it could be concluded that products of glutamate deamination can be utilized not only in respiratory metabolism (by the Krebs cycle or the respiratory chain) but also in other important metabolic pathways.

Simultaneously it should be noted that amination by GDH was not affected by any of the environmental factors applied (Table 7 A, B, C, D) which evidences its independence of the respiratory metabolism, while the lack of its activation during starvation suggests that amination is not involved in the processes supply L-glutamate under conditions when the inflow of the energetic substrates is disturbed.

Summing up, the results presented suggest that, in D.melanogaster larvae GDH is involved in aerobic respiratory metabolism of the cell, most probably supporting it by supplying 2-oxoglutarate and/or NADH; however, the products of the deamination reaction could also be utilized by other metabolic pathways.

References:

Nowak, J. and M.J. Piechowska. Institute of Biochemistry and Biophysics, Warsaw, Poland. Kinetic analysis of glutamate dehydrogenase from D.melanogaster larvae.

The smallest, catalytically active subunit of glutamate dehydrogenase (GDH) of D.melanogaster is an oligomer composed of six subunits (Caggese et al. 1982). In proteins composed of subunits, mutual interactions between subunits can take place (Levitzki 1978). The studies of Engel & Dalziel (1969) point to occurrence of negative cooperativity between the subunits of bovine GDH hexamer. The aim of the present work was to make an initial assessment of the nature of cooperativity between the subunits of GDH from D.melanogaster larvae.

Materials and Methods. Biological material, methods of enzyme isolation, and determination of its activity were the same as in the accompanying paper (Nowak & Piechowska 1986). To assess the type of cooperativity between the subunits of D.melanogaster GDH, kinetic data on the dependence of the deamination rate on coenzyme (NAD) concentration at a fixed, 10 mM L-glutamate, as well as on substrate concentration at fixed, 6 mM NAD, were subjected to graphical analysis. Michaelis-Menten, Lineweaver-Burke & Scatchard plots (Segel 1975) are presented.

Results and Discussion. The Michaelis-Menten plot (Fig. 1) representing the dependence of GDH activity on NAD concentrations is of hyperbolic character. On the other hand, in the Lineweaver-Burke plot (Fig. 2) three linear regions can be discerned, their slope becoming steeper with respect to the ordinate.
The activity of D. melanogaster GDH on NAD concentration: the Michaelis-Menten plot.

Figure 1.

Table 1. Dependence of $K_m$ NAD values of glutamate dehydrogenase on NAD concentration.

<table>
<thead>
<tr>
<th>region range of NAD (mM)</th>
<th>$K_m$ NAD (mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I 0.05 - 0.50</td>
<td>0.06 ± 0.02</td>
</tr>
<tr>
<td>II 0.50 - 3.00</td>
<td>1.22 ± 0.17</td>
</tr>
<tr>
<td>III 3.00 - 10.0</td>
<td>3.31 ± 0.41</td>
</tr>
</tbody>
</table>

Figure 2. Variation of the reciprocal of the activity of D. melanogaster GDH with the reciprocal of NAD concentration: the Lineweaver-Burke plot.

Data on dependence of the reaction rate on L-glutamate concentration indicate that binding of this substrate is also accompanied by negative cooperativity between the subunits: the Michaelis-Menten plot is hyperbolic (Fig. 3) and the corresponding Lineweaver-Burke plot is concave downward (Fig. 4). $K_m$ glu value determined from the linear regions of the plot increase with substrate concentration (Table 2). Moreover, deamination is inhibited by L-glutamate over 10 mM. A similar Lineweaver-Burke plot for GDH from bovine liver was reported by Barton & Fisher (1971).
Figure 3. The activity of *D. melanogaster* GDH on L-glutamate concentration: the Michaelis-Menten plot.

Table 2. Dependence of $K_m^{\text{glu}}$ values of glutamate dehydrogenase on L-glutamate concentration.

<table>
<thead>
<tr>
<th>region concentration range of L-glutamate (mM)</th>
<th>$K_m^{\text{glu}}$ (mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I 0.2 - 1.0</td>
<td>0.68 ± 0.08</td>
</tr>
<tr>
<td>II 1.0 - 10.0</td>
<td>1.35 ± 0.13</td>
</tr>
</tbody>
</table>

Figure 4. Variation of the reciprocal of the activity of *D. melanogaster* GDH with the reciprocal of the L-glutamate concentration: the Lineweaver-Burke plot.

Figure 5. Variation of the ratio of *D. melanogaster* GDH activity to NAD concentration with GDH activity: the Scatchard plot.

Figure 6. Variation of the ratio of *D. melanogaster* GDH activity to L-glutamate concentration with GDH activity: the Scatchard plot.
To determine with greater certainty the model of cooperativity between subunits of *D. melanogaster* GDH, the data obtained on the dependence of the reaction rate on concentration of NAD or L-glutamate were presented also as a Scatchard plot (Fig. 5 and 6). The course of the curves, concave upwards, was consistent with negative interactions between subunits of *D. melanogaster* GDH.

Thus, the kinetic data presented permit us to suggest that the subunits of glutamate dehydrogenase from *D. melanogaster* larvae exhibit negative cooperativity. However, for final assessment of the actual type of subunit interaction, the purified enzyme should be analyzed by isotope exchange experiments or by X-ray crystallography.

References:

O'Dell, K.M.C. and B. Burnet. The University, Sheffield, England. Allelism of the behavioural mutants hypoactive B\(^1\) and inactive in *D. melanogaster*.

Hall (1982) and Lindsley & Zimm (1985) have suggested that the two X-linked behavioural mutants hypoactive B\(^1\) (hypo B\(^1\), Homyk & Sheppard 1977, Homyk 1977) and inactive (iav, Kaplan 1977) are alleles. They are currently mapped at 20.7 and 18.8, respectively (Lindsley & Zimm 1985).

To discover whether hypo B\(^1\) and iav are indeed allelic, we have crossed the two stocks to observe the behaviour of the F\(_1\) heterozygous hypo B\(^1\)/iav females. As a control, a third and independent gene, hypoactive C\(^1\) (hypo C\(^1\), Homyk & Sheppard 1977) mapping at X:c42 was also crossed to hypo B\(^1\) and iav and the behaviour of the F\(_1\) female progeny scored. The mutant hypo C\(^1\) shows a similar phenotype to hypo B\(^1\) and iav, but at more than twenty map units away hypo C\(^1\) is unlikely to be allelic to either hypo B\(^1\) or iav.

The mutants hypo B\(^1\), hypo C\(^1\) and iav are all recessive. Hence the crosses hypo B\(^1\) x hypo C\(^1\) and iav x hypo C\(^1\) should (and do) produce behaviourally wildtype female progeny. The female F\(_1\) progeny of the cross hypo B\(^1\) x iav should show normal locomotor activities if the mutants are at separate loci, but will show marked reductions in locomotor activities if the mutants are allelic.

The technique used to measure locomotor activity of F\(_1\) females was a modified version of that of Connolly (1966). Single flies were observed under a 10 x 10 cm open field grid for a period for 10 minutes and the frequency and duration of key presses made by the observer was recorded using an Apple II Microcomputer as a data logger. This manner scores for amount and speed of movement, distance travelled, time spent preening and frequencies of falling and jumping could immediately be tabulated for each successive minute observed. By this method we can show that these three mutants are characterized by showing similar and extreme reductions in both amount and speed of movement as well as showing deficits in other locomotor activities.

The average scores per minute for the three hybrid classes were calculated using ten replicate individuals per class. The results for speed of movement are shown below.
Clearly the hybrid F₁ females from the cross hypo B¹ x iav show a marked reduction in speed of movement with respect to the hybrid female offspring from the other crosses. In the other behavioural tests a similar result is obtained.

Given the additional information that a stock derived from these hypo B¹/iav hybrids still retains this marked reduction in all activities over twenty generations later, this must lead us to the conclusion that hypo B¹ and iav are allelic.


The frequency of inversion-carrying chromosomes in natural populations of Drosophila melanogaster has recently been reported rather high even in this cosmopolitan species (e.g., Stalker 1976; Mettler et al. 1977; Paik 1979; Knibb et al. 1981), but information on inversion polymorphism of this species is not yet effectively collated. This study was therefore undertaken to measure the year-to-year changes in the frequencies of these polymorphic inversions in a locality. This preliminary communication covers the first four years (1978 - 1981) of the study extending to 1985.

The flies were taken in a large pear-orchard district called Taenung about 16 kilometers N.E. of central Seoul. The population samples were collected once every year in late October when the orchard populations reach their peak size. Haploid sets of chromosomes carried by the wild males were analyzed for their gene arrangements by mating them individually with virgin Oregon-R females homozygous for the standard gene arrangement, and then by examining preparation of smears of the salivary gland cells of one F₁ larva from each mating. The breakpoints of inverted gene arrangements were identified on the basis of the salivary gland chromosome maps provided by Bridges & Breheme (1944) and Lefevre (1976).

A total of 49 types of inversions were detected from the four year collections, of which only six belong to the category of cosmopolitan and polymorphic type, and the rest to that of the type unique to the area. The frequencies of these polymorphic inversions throughout the period of study are given in Table 1 and illustrated in Fig. 1. These table and figure show some points of interest:

1. Directional frequency changes took place at least in three of the cosmopolitan inversions: In(2L)lt, In(2R)NS, In(3R)C. This suggests they are genetically flexible and sensitive to environmental changes whose factor(s) remain to be clarified in the future.

Figure 1. Frequencies of arrangements 2Lt, 2RNS, 3LP, 3RC, 3RP, 3RMo, plotted against date of collection at Taenung.
The reconstituted karyotypes of these Cytoraces are new in composition, the fecundity in the parent and hybrid populations of Drosophila melanogaster.

The estimation of fitness is the first step in understanding the adaptive evolution of any evolutionary lineage. Egg laying capacity or fecundity of a population is one of the important parameters of fitness which can be used as a yardstick for comparisons.

In the present study, fecundity of the parental races (D.n.nasuta and D.n.albomicana), the Cytoraces I and II as well as of the F1 hybrids has been estimated. The aim of this experiment is to know the impact of hybridization and karyotype restructuring on the fecundity of individuals.

After aging the isolated flies of the above said subjects for five days, pair matings were made. These pairs were transferred to fresh vials without etherization once in 24 hr. Then, the number of eggs
in the formation of new Cytoraces which differ from their parents in their karyotypic constitution, quantity of heterochromatin and in their ability to lay different number of eggs.

Authors are grateful to Prof. N.B. Krishnamurthy, Head of the Dept. of Zoology, for his help and encouragement; to the University Grants Commission and the Indian National Science Academy, New Delhi, for financial assistance.


Table 1. Fecundity of Drosophila nasuta nasuta, D.n.alboicana, F1 hybrids and of Cytorace I and II. 60 females of each category were screened for a period of ten days.

<table>
<thead>
<tr>
<th>Strain</th>
<th>Total no.</th>
<th>Eggs/individual</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. D.n.nasuta (2n=8)</td>
<td>12,058</td>
<td>200.96</td>
</tr>
<tr>
<td>2. D.n.alboicana (2n=6)</td>
<td>10,749</td>
<td>179.15</td>
</tr>
<tr>
<td>3. F1 of the cross: (2n=7) D.n.nasuta x D.n.alboicana♂</td>
<td>14,008</td>
<td>233.46</td>
</tr>
<tr>
<td>4. F1 of the cross: (2n=7) D.n.nasuta ♀ x D.n.alboicana♂</td>
<td>15,315</td>
<td>255.25</td>
</tr>
<tr>
<td>5. Cytorace I*: (♂ 2n=7; ♀ 2n=6) a product of hybridization between D.n.nasuta ♀ and D.n.alboicana♂</td>
<td>13,232</td>
<td>220.52</td>
</tr>
<tr>
<td>6. Cytorace II*: (♂ and ♀ 2n=6) a product of hybridization between D.n.nasuta ♀ and D.n.alboicana♂</td>
<td>12,030</td>
<td>200.50</td>
</tr>
</tbody>
</table>

* A continuous inbreeding of the respective hybrid populations for over 20 generations has resulted in the evolution and stabilization of these Cytoraces.

Thus, the hybridizations between D.n.nasuta and D.n.alboicana has resulted in the formation of new Cytoraces which differ from their parents in their karyotypic constitution, quantity of heterochromatin and in their ability to lay different number of eggs.

Acknowledgements: Authors are grateful to Prof. N.B. Krishnamurthy, Head of the Dept. of Zoology, for his help and encouragement; to the University Grants Commission and the Indian National Science Academy, New Delhi, for financial assistance.


Ramachandra, N.B. and H.A. Ranganath.
University of Mysore, India. Resource utilization divergence in two cosmopolitan and sympatric species of Drosophila.

Drosophila melanogaster and D.ananassae of the melanogaster species group of the subgenus Sophophora of the genus Drosophila are a pair of cosmopolitan and sympatric species. They cohabit in the domestic localities and occur in varying frequencies throughout the year. Preliminary studies made by Nirmala & Krishnamurthy (1973) have revealed that under laboratory conditions, D.melanogaster is competitively superior to D.ananassae. Mechanisms of their apparent "coexistence" in nature are not known. Different dimensions of their ecological relationship and their requirements or preferences have not been worked out. Our preliminary report (Ramachandra & Ranganath 1984) on the nutritional requirements of these two species revealed the existence of 'subtle' differences. In view of this, experiments have been initiated in this direction to understand the impact of different ecological regimes on certain aspects of the parameters of fitness in these two ecologically closely placed forms of Drosophila.

Drosophila utilize a variety of sugar sources (Bamberger 1919; Hassett 1948; Taylor & Condra 1983; Jaenike & Grimaldi 1983; Powel & Andgelkovic 1983). Nutritional methodology is a sharper instrument for the dissection of the genotype, particularly through the use of variety of sugars (Sang 1972). In the light of this, we have used four different types of wheat cream agar medium containing either molasses or sucrose or glucose or fructose to study the differences in the ability of D.melanogaster and D.ananassae to utilize these different resources. The impact of differences in the media was assessed by estimating certain parameters of fitness, namely egg to adult rate of development, viability and adaptedness.

The egg to adult rate of development and viability were estimated by adapting the procedure of Ranganath & Krishnamurthy (1974) with little modifications, while the adaptedness was assessed by following the serial transfer technique of Avila (1965). The present findings are as follows:

(1) The egg to adult rate of development is defined as the time between egg laying and emergence of the imago. The statistical comparison revealed that D.melanogaster has the fastest rate of development in the media with molasses and the slowest in the media with glucose, while D.ananassae has achieved the fastest rate of development in the media with sucrose and slowest in the media with fructose. D.melanogaster has better speed of development than D.ananassae in the media with molasses and fructose, while D.ananassae has experienced better rate of development than D.melanogaster in the media with sucrose and glucose. For D.melanogaster the mean rate of developmental time in days in four different media, ranges from 13.57 to 15.33 days and for D.ananassae, the range is 13.79 to 15.34 days.
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(2) The viability is defined as the proportion of eggs reaching the adulthood. Maximum viability for *D. melanogaster* was in the media with molasses (85%) and the least was in the media with sucrose (69%). For *D. ananassae*, the media with molasses has given the highest viability of 64%, while the least was recorded in the media with fructose (51%).

(3) The adaptedness of a population is defined as "the ability of the carriers of a genotype or a class of genotypes to survive and reproduce in a given environment" (Dobzhansky 1968). Population size, productivity, mortality and flies per bottle were used as four different facets of adaptedness. The analysis of variance test revealed that only in the media with fructose *D. ananassae* has more of adaptedness than *D. melanogaster*. While in all the other three types of media, *D. melanogaster* has significantly more values than that of *D. ananassae*. Both for *D. melanogaster* and *D. ananassae*, the media with molasses is more suitable than other media and the sequence is molasses > sucrose > glucose > fructose.

Acknowledgements: Authors are grateful to Prof. N.B. Krishnamurthy, Head of the Dept. of Zoology, for his help and encouragement; to the University Grants Commission, New Delhi, for financial assistance.


Raymond, J.D., T.R. Laverty and M.J. Simmons. University of Minnesota, St. Paul, USNA. Cytogenetic analysis of lethal X chromosomes derived from dysgenic hybrids of *Drosophila melanogaster*. Simmons et al. (1985) measured the frequencies of recessive X-linked lethal mutations occurring in various dysgenic males derived from crosses between pure M females and P-element-containing males. The latter were collected from two pairs of related stocks: 

*π₁₂*, a strong P strain, and C(1)DX, y f/Y/+; π₂₂, an attached-X strain with the π₂ genetic background; and v₆., a Q or weak P strain, and C(1)DX, y f/Y/+; v₆., an attached-X strain with the v₆ genetic background. Through genetic manipulations, Simmons et al. (1985) obtained dysgenesis-induced lethal mutations on both the π₂ and v₆ X chromosomes and on X chromosomes derived from a pure M strain but having been exposed to the autosomes of either of the attached-X stocks. See their paper for experimental details.

We have analyzed 378 of the 1,030 lethal X chromosomes recovered from these P element mutagenesis experiments. In this analysis, a single /FM7 female from each lethal stock was mated to Df(1)Basc/sc²Y males. The Df(1)Basc daughters were then crossed either to y sn² v larvae for cytology, or to duplication/deficiency males in an attempt to locate the lethal mutation through complementation tests. For the latter, we employed six duplication/deficiency stocks; these included the first five stocks listed in Table 1 of Simmons et al. (1984), plus C(1)DX, y w f/Df(1)Basc/2B17-18 + 20A3;20F4. Cytological data on the other five stocks are given in Table 1 of Simmons et al. (1984). Altogether, the deficiencies in these six stocks allowed us to screen approximately 16 percent of the euchromatic portion of the X chromosome for the location of the lethal mutations. The chromosomes having these lethals were maintained in stocks for 20-30 generations before the cytogenetic analysis began. During this time no effort was made to control the cytotype, so secondary dysgenesis-induced lethal mutations and chromosome rearrangements might have occurred.

A summary of complementation tests with the six deficiencies is given in Table 1. In total, 73.5% of the chromosomes tested had a lethal mutation in at least one of the regions uncovered by the six deficiencies. Before discussing the distribution of these lethals in detail, it is important to note that in many instances where a lethal mutation was uncovered by a deficiency, the corresponding duplication did not cover it; this indicated that the tested chromosome had more than one lethal mutation. Sometimes another deficiency uncovered the extra lethal, permitting its localization. Altogether, 109 (39%) of the 278 chromosomes with localized lethals had extra lethal mutations.

The first category of lethal X chromosomes analyzed came from the weak P strain v₆. The vast majority (61.8%) of these had recessive lethal mutations in one of the regions uncovered by the y, r, and mal deficiencies. The other deficiencies (w, ct, and m) uncovered lethals on only four chromosomes—less than 3% of those analyzed. The mal deficiency uncovered lethals on over half the v₆ X chromosomes, the r deficiency on nearly a third and the y deficiency on approximately a sixth. Clearly, these deficiencies reveal hotspots for the occurrence of dysgenesis-induced lethal mutations on the v₆ X chromosome.
By comparison, there were at least 13 independent lethal mutations. The few lethals that were detected may have been due to P element insertions in essential loci, or to other events unconnected with dysgenesis. Significantly, only appreciable numbers of lethal mutations. The few lethals that were detected may have been due to P element insertions rather than by local P element action. These hotspots on the M X chromosome coincide with two of the three hotspots on the V6 X chromosome, suggesting a rather high frequency of mutation therein. Likewise, five independent lethals were uncovered by the y deficiency, pointing to another potential hotspot. Since the M X chromosomes were initially devoid of P elements, these two hotspots could represent sites for preferential P element insertion. These hotspots on the M X chromosome coincide with two of the three hotspots on the V6 X chromosome, suggesting that on the latter chromosome, some of the y and r region mutations might have been caused by P element insertions rather than by local P element action.

Lethal mutations were localized on 67 of the 112 \( \text{v}_6 \) X chromosomes that were analyzed. Nearly 60% of these had more than one lethal mutation, suggesting that many secondary lethal mutations had occurred. The r deficiency uncovered the most lethal mutations, followed by the y, mal, w, ct, and m deficiencies in that order. From in situ hybridization it is clear that the \( \text{v}_6 \) X chromosome possesses P elements in the regions uncovered by the w and mal deficiencies, but not in those uncovered by the y, ct, m and r deficiencies (Engels & Preston 1981). Therefore, although the high frequencies of lethal mutations in the w and mal regions are explicable in terms of local P element action, the high frequencies in the y and r regions are not. It is possible that the abundance of lethals in these two regions is due to frequent P element insertions. Significantly, the m region and, to a lesser extent, the ct region on the \( \text{v}_6 \) X chromosome were unusual mutations; this is consistent with the absence of P elements in these parts of the chromosome.

In addition to the P elements in the w and mal regions, the \( \text{v}_6 \) X chromosome possesses P elements at 5E, 11A and 17C (Engels & Preston 1981); however, none of these was uncovered by the deficiencies used in this analysis. In this connection it is notable that the \( \text{v}_6 \) X chromosomes had the highest frequency of unlocalized lethal mutations; 45 of the 112 chromosomes did not have a lethal uncovered by any of the deficiencies. Perhaps some of the lethal mutations on these chromosomes were caused by the 5E, 11A or 17C P elements.

### Table 1. Complementation analysis of dysgenesis-induced lethal X chromosomes.

<table>
<thead>
<tr>
<th>Deficiency</th>
<th>Breakpoints</th>
<th>( \text{v}_6 ) dysgenic hybrids</th>
<th>( \text{v}_6 ) X</th>
<th>( \text{v}_2 ) dysgenic hybrids</th>
<th>( \text{v}_2 ) X</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Df(1)TEM(l) (y)</td>
<td>1A1-2; 1B6-9</td>
<td>28 (16.9)</td>
<td>5 (15.2)</td>
<td>22 (19.6)</td>
<td>25 (36.8)</td>
<td>80 (21.1)</td>
</tr>
<tr>
<td>Df(1)c(2) (ct)</td>
<td>6F1-2; 7C1-2</td>
<td>1 (0.6)</td>
<td>1 (3.0)</td>
<td>9 (8.0)</td>
<td>3 (4.4)</td>
<td>14 (3.7)</td>
</tr>
<tr>
<td>Df(1)m(259-3) (m)</td>
<td>10C1-2; 10E1-2</td>
<td>1 (0.6)</td>
<td>0 (0.0)</td>
<td>2 (1.8)</td>
<td>0 (0.0)</td>
<td>3 (0.8)</td>
</tr>
<tr>
<td>Df(1)+75e (r)</td>
<td>14B13; 15A9</td>
<td>48 (29.0)</td>
<td>20 (60.6)</td>
<td>28 (25.0)</td>
<td>25 (36.8)</td>
<td>121 (32.0)</td>
</tr>
<tr>
<td>Df(1)mal(3) (mal)</td>
<td>19A1-2; 20A</td>
<td>84 (50.9)</td>
<td>1 (3.0)</td>
<td>20 (17.8)</td>
<td>5 (7.4)</td>
<td>110 (29.1)</td>
</tr>
</tbody>
</table>

Total chromosomes with at least one lethal mutation localized

<table>
<thead>
<tr>
<th>Breakpoints</th>
<th>( \text{v}_6 ) X</th>
<th>( \text{v}_2 ) X</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>139 (84.2)</td>
<td>24 (72.7)</td>
<td>67 (59.8)</td>
<td>48 (70.6)</td>
</tr>
<tr>
<td>24 (14.5)</td>
<td>2 (6.1)</td>
<td>24 (21.4)</td>
<td>11 (16.2)</td>
</tr>
<tr>
<td>5 (3.0)</td>
<td>6 (18.2)</td>
<td>16 (14.3)</td>
<td>21 (30.9)</td>
</tr>
</tbody>
</table>

Total chromosomes analyzed

<table>
<thead>
<tr>
<th>Clusters</th>
<th>( \text{v}_6 ) X</th>
<th>( \text{v}_2 ) X</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>3 of 2</td>
<td>1 of 2</td>
<td>4 of 2</td>
<td>3 of 2</td>
</tr>
<tr>
<td>1 of 3</td>
<td>1 of 5</td>
<td>2 of 3</td>
<td>1 of 8</td>
</tr>
</tbody>
</table>

Entries are numbers of chromosomes, with percentages in parentheses.

Simmons et al. (1984) had detected the r and mal hotspots in an earlier study; however, because they did not use the y deficiency, they failed to notice the hotspot it uncovers. Each of the three mutable regions encompasses a P element site, as judged by in situ hybridization (Simmons et al. 1984), which is probably the major reason for their pronounced mutability. Another P element, located at 11A on the \( \text{v}_6 \) X chromosome, lies in a region that was not uncovered by any of the deficiencies used in this analysis, so its contribution to the production of lethals could not be assessed. It is noteworthy that the w, ct, and m regions on the \( \text{v}_6 \) X chromosome do not contain any P elements, and neither do they give rise to appreciable numbers of lethal mutations. The few lethals that were detected may have been due to P element insertions in essential loci, or to other events unconnected with dysgenesis. Significantly, only 29 (21.0%) of the 139 \( \text{v}_6 \) X chromosomes with locatable lethals had more than one lethal mutation. This is the lowest such frequency among the four sets of X chromosomes that we analyzed. Thus, despite the significant amount of time that elapsed between the recovery of these lethal chromosomes and their analysis by complementation testing, few secondary lethal mutations had accumulated.

Only 33 lethal M X chromosomes derived from \( \text{v}_6 \) dysgenic hybrids were analyzed. Nearly half of these were recovered in three different clusters, so the data probably do not represent independent mutational events. This must be taken into consideration when the distribution of the lethal mutations is evaluated. Although 20 (60.6%) of these M X chromosomes had lethals uncovered by the r deficiency, seven came from a cluster of eight and two came from another cluster of five. Counting each of these clusters as two mutational events and the other cluster as one, there were at least 23 independent mutational events in this sample of chromosomes. By comparison, there were at least 13 independent lethal mutations in the r region, suggesting a rather high frequency of mutation therein. Likewise, five independent lethals were uncovered by the y deficiency, pointing to another potential hotspot. Since the M X chromosomes were initially devoid of P elements, these two hotspots could represent sites for preferential P element insertion. These hotspots on the M X chromosome coincide with two of the three hotspots on the \( \text{v}_6 \) X chromosome, suggesting that on the latter chromosome, some of the y and r region mutations might have been caused by P element insertions rather than by local P element action.

Complementation analysis of dysgenesis-induced lethal X chromosomes.
The M X chromosomes that were derived from the \( \pi_2 \) dysgenic hybrids had a concentration of lethal mutations in the regions uncovered by the y and r regions; there were 25 in each case, compared to a total of 68 chromosomes analyzed. This is reminiscent of the distribution of lethals on the M X chromosomes derived from the \( \nu_6 \) hybrids. Evidently, these two regions are unusually receptive to P element insertions. The receptivity of the y region may reflect a general tendency for P elements to insert near the ends of chromosomes (W.R. Engels, pers. comm.), but the receptivity of the r region would have to be explained on other grounds. Perhaps, along with other frequently mutated loci, some part of the r region has an ideal sequence for P element insertion. Only a direct molecular analysis can settle this issue.

The M X chromosomes derived from the \( \pi_2 \) dysgenic hybrids had a high frequency of multiple lethal mutations; 32 out of 48 chromosomes with localized lethals had at least one extra lethal mutation. This suggests that secondary lethals occurred on these chromosomes, most likely while they were kept in stock.

From this complementation analysis, it appears that dysgenesis-induced lethal mutations are frequently found in chromosomal regions containing P elements. As has been speculated previously (Simmons et al. 1984), these lethals probably arise from local P element action. There is, however, an additional group of lethals that is not associated with local P element activity. The lethals on the M X chromosomes belong to this group, as do the lethals in the y and r regions on the \( \pi_2 \) X chromosome. Curiously, the lethals on the M X chromosomes are concentrated in the y and r regions. The concentration in the y region is all the more striking because the effective size of that region is quite small. Although the y deficiency spans bands 1A1-2; 1B6-9, the bands spanned by the deficiency in Df(1)Basc must be excluded; the reason is that on account of our methods, any lethal exposed by the latter deficiency could not have been tested against the y deficiency. This means that the lethals that were mapped to the y region in fact map to only a portion of that region, revealing a pronounced mutational hotspot. Together with the r region, this hotspot may reflect a tendency of P elements to transpose to particular sites. Eeken et al. (1984) performed very detailed genetic analyses of P-element-induced lethals on M X chromosomes and found quite a scattered distribution. There was no tendency for lethals to map in the y and r regions, as found here. This discrepancy might indicate that X chromosomes differ in the positions of their favored P element insertion sites, or that P elements from different strains have different target specificities.

In addition to the complementation analysis, 255 of the lethal X chromosomes were examined cytologically. Eighty-five (33.3\%) contained structural abnormalities, consisting primarily of deficiencies and simple two-point inversions. Excluding the deficiencies, there were 116 breaks in this sample of chromosomes, 32 (26.9\%) of which appeared to be associated with a lethal effect. Another 26 (22.6\%) may have been lethal, but the remaining 58 (50.4\%) definitely were not.

Breakage on the \( \nu_6 \) X chromosomes was concentrated in bands 14C7-E1 (in the r region) and in bands 18F-20A (in the mal region); 62, or nearly half of the 129 chromosomes that were analyzed, had some sort of structural abnormality. The distribution of breakpoints, which is shown in Figure 1, corresponded closely to the distribution of P elements on the \( \nu_6 \) X chromosome. Of the 62 chromosomes with rearrangements, 27 had a break in bands 14C7-E1; although none of these breaks was associated with a cytologically detectable deficiency, 26 appeared to have a lethal effect (the viability effect of the remaining break could not be determined). It should be noted, however, that many \( \nu_6 \) X chromosomes without breakage at this point had lethals in the r region, so breakage is not the only cause of this kind of lethality. The other breakage hotspot on the \( \nu_6 \) X chromosome was in bands 18F-20A. All 62 of the rearranged chromosomes had breakage in these bands, with the preponderance (40) of the chromosomes having breaks in 19C2-D1. Many of the breaks in 19C2-D1 were associated with lethal deficiencies, but the 14 that were not had no lethal effect. Some of the lethals in the mal region were clearly not associated with any breakage there. Simmons et al. (1984) studied over 30 such lethals and found that they were nearly all allelic to HF326, a lethal mutation obtained from George Lefevre. Since the publication of their paper, HF326 has been reassigned to bands 19B3-C1, rather than to 19C4 as Simmons et al. (1984) reported (George Lefevre, pers. comm.). Significantly, there is a P element in 19C on the \( \nu_6 \) X chromosome; presumably, the lethals allelic to HF326 arise from the activity of this element, as do the breaks in 19C2-D1.

Only one of the 23 lethal M X chromosomes derived from the \( \nu_6 \) hybrids which were examined cytologically was structurally abnormal. This chromosome had an inversion (breakpoints 11A3-9; 12E-F) which was not associated with any lethal effect. Thirteen of the chromosomes with a lethal in the r region were examined and none had a break in 14C7-E1, the breakage hotspot in the r region on the \( \nu_6 \) X chromosome.

Sixty-one of the lethal M X chromosomes derived from \( \pi_2 \) were examined cytologically and 19 were found to be abnormal. Nine had simple inversions, six had deficiencies and four had more complicated rearrangements. Excluding the deficiencies, there were 36 breakpoints altogether, 3 being lethal, 14 nonlethal and 19 with unknown effects on viability. The distribution of the breakpoints is shown in Figure 1. Except for some clustering near the base, the breakpoints are distributed rather uniformly over the chromosome map. There is no pronounced concentration in the vicinity of resident P elements, as was the
case for the $\nu_6$ X chromosome. This contrasts with the results of Berg et al. (1980) and Engels & Preston (1981), who found that the breakpoints of dysgenesis-induced rearrangements on the $\pi_2$ X chromosome preferentially involved P element sites. The discrepancy between our findings and theirs might have at least two explanations. First, we selected lethal X chromosomes whereas Berg et al. (1980) selected chromosomes which suppressed recombination and Engels & Preston (1981) selected chromosomes with a rearrangement-associated visible mutation; the latter were all nonlethal. In both instances, chromosomes with primary rearrangements were selected, whereas the chromosomes that we analyzed were selected because they had primary lethal mutations, which may or may not have been associated with rearrangements. Second, the chromosomes we analyzed were kept in stocks for 20-30 generations without controlling the cytotype. In the other studies cited, the rearranged chromosomes were analyzed immediately, or, if this was impossible, after they had been maintained in stocks with the P cytotype, which severely limits P element transposition and P-element-mediated chromosome breakage. Therefore, the scatter of breakpoints on the $\pi_2$ map in Figure 1 is probably due to an accumulation of secondary transpositions and rearrangements, which, evidently, did not occur in the sample of $\nu_6$ X chromosomes that we studied. This explanation is supported by the much higher frequency of chromosomes known to have multiple lethal mutations in the sample derived from $\pi_2$ (59.7% vs 19.6% from $\nu_6$).

The M X chromosomes derived from the $\pi_2$ dysgenic hybrids showed very few rearrangements. Two of the 42 chromosomes that were analyzed had small deficiencies and one had a simple inversion. One of the breakpoints of the inversion was nonlethal, but the viability effect of the other could not be determined. Both of the deficiencies were in the mal region and had lethal effects.

It is evident that the lethal X chromosomes derived from $\nu_6$ and $\pi_2$ had numerous rearrangements. In the case of the $\nu_6$ X chromosomes, the breakpoints coincided closely with P element sites. In the case of the $\pi_2$ X chromosomes, a coincident distribution of breaks and P elements was not observed. For the lethal M X chromosomes which had been exposed to $\nu_6$ or $\pi_2$ autosomes in dysgenic hybrids, few rearrangements were detected. Evidently P-element-mediated rearrangements are rare on these chromosomes, even after they have been maintained for many generations in the absence of the P cytotype.

Acknowledgements: This work was supported by the National Institute of Environmental Health Sciences (RO1 ES01960). R. Doll, E. Drier, G. Kocur and N. Raymond provided technical help.

Chromosome studies of *Drosophila melanogaster* collected five times with a month apart from mid-August to mid-December in 1982 from a large vineyard near Jeonju, South Korea, discovered seventeen inversions among a total of 1056 males tested. For analyzing the pairs of homologous chromosomes seven F1 larvae per male strain were selected randomly. All of the inversions found were paracentric and on two major autosomes only, and none on X- and fourth chromosomes. Among them, six were common cosmopolitan inversions, and the other eleven were new, rare and endemic inversions. The frequencies of each common cosmopolitan inversion are as shown in Table 2. Therefore the frequency of wild males carrying one or more cosmopolitan inversions and the mean number of cosmopolitan inversions per individual is 0.540 and 0.743, respectively. Although linkage disequilibrium appeared significantly within the third chromosome when all of the third chromosomal combinations were pooled, the data indicate a random distribution of inversions on the whole (Table 3). And, in the present population of *D. melanogaster* as shown in Table 4, there was no differential mortality for inversion homozygotes and heterozygotes by natural selection. Particularly, one of the interesting results in this study was the discovery of a cis-coupled overlapping inversion which was identified to be the cosmopolitan In(3R)C and In(3R)P in approximate breakage points. Therefore, if cosmopolitan inversions were originally produced by site-specific transposable elements, and also, if the cis-coupled overlapping inversion was in fact composed of the two cosmopolitans, the possibility that the origin of cosmopolitan inversions may be from each geographical population just as endemics cannot be excluded.

### References:

### Table 1. Polymorphic inversions from a Jeonju natural population of *Drosophila melanogaster*.

<table>
<thead>
<tr>
<th>Inversion</th>
<th>Approximate Breakpoints</th>
<th>Month collected (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Second, Left</td>
<td></td>
<td></td>
</tr>
<tr>
<td>In(2L) A</td>
<td>22B - 26A</td>
<td>A(0.5), S(0.9), O(1.4), N(0.5), D(0.5)</td>
</tr>
<tr>
<td>In(2L) B</td>
<td>22E - 34A</td>
<td>A11 samples (see Table 2)</td>
</tr>
<tr>
<td>In(2L) C</td>
<td>26A - 33E</td>
<td>D(0.5)</td>
</tr>
<tr>
<td>In(2L) D</td>
<td>30E - 33B</td>
<td>A(0.5)</td>
</tr>
<tr>
<td>Second, Right</td>
<td></td>
<td></td>
</tr>
<tr>
<td>In(2R) E</td>
<td>42E - 50B</td>
<td>D(0.5)</td>
</tr>
<tr>
<td>In(2R) F</td>
<td>42C - 56E</td>
<td>D(0.5)</td>
</tr>
<tr>
<td>In(2R) G</td>
<td>42C - 58B</td>
<td>S(0.4)</td>
</tr>
<tr>
<td>In(2R) H</td>
<td>53A - 56F</td>
<td>A11 samples (see Table 2)</td>
</tr>
<tr>
<td>Third, Left</td>
<td></td>
<td></td>
</tr>
<tr>
<td>In(3L) I</td>
<td>63C - 72E</td>
<td>A11 samples (see Table 2)</td>
</tr>
<tr>
<td>In(3L) J</td>
<td>640 - 67A</td>
<td>S(0.9)</td>
</tr>
<tr>
<td>In(3L) K</td>
<td>66C - 71B</td>
<td>N(0.5)</td>
</tr>
<tr>
<td>In(3L) L</td>
<td>67A - 71B</td>
<td>N(0.5), D(1.0)</td>
</tr>
<tr>
<td>Third, Right</td>
<td></td>
<td></td>
</tr>
<tr>
<td>In(3R) M</td>
<td>89C - 96A</td>
<td>A11 samples (see Table 2)</td>
</tr>
<tr>
<td>In(3R) N</td>
<td>91B - 96F</td>
<td>N(0.5)</td>
</tr>
<tr>
<td>In(3R) O</td>
<td>920 - 100F</td>
<td>A11 samples (see Table 2)</td>
</tr>
<tr>
<td>In(3R) P</td>
<td>930 - 98F</td>
<td>A11 samples (see Table 2)</td>
</tr>
<tr>
<td>In(3R) Q</td>
<td>96C - 980</td>
<td>A(1.0)</td>
</tr>
<tr>
<td>Overlapping Inversion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>In(3R) PC</td>
<td>89C - 96A</td>
<td>S(0.4), O(0.9): repulsion phase</td>
</tr>
<tr>
<td>In(3R) PMo</td>
<td>89C - 96A</td>
<td>N(0.4): coupling phase</td>
</tr>
<tr>
<td>In(3R) PMo</td>
<td>93D - 98F</td>
<td>N(0.5): repulsion phase</td>
</tr>
<tr>
<td>Included Inversion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>In(3R) CMo</td>
<td>920 - 100F</td>
<td>S(0.4): repulsion phase</td>
</tr>
<tr>
<td></td>
<td>93D - 98F</td>
<td></td>
</tr>
</tbody>
</table>

### Table 2. Frequencies of sperms carrying each of six cosmopolitan inversions from five samples of *D. melanogaster*.

<table>
<thead>
<tr>
<th>Sample</th>
<th>N (2L)t</th>
<th>(2R)NS</th>
<th>(3L)P</th>
<th>(3R)C</th>
<th>(3R)Mo</th>
<th>(3R)P</th>
<th>Fl</th>
<th>Mi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aug</td>
<td>400</td>
<td>0.135</td>
<td>0.103</td>
<td>0.013</td>
<td>0.010</td>
<td>0.013</td>
<td>0.025</td>
<td>0.500</td>
</tr>
<tr>
<td>Sep</td>
<td>462</td>
<td>0.071</td>
<td>0.102</td>
<td>0.052</td>
<td>0.033</td>
<td>0.013</td>
<td>0.096</td>
<td>0.524</td>
</tr>
<tr>
<td>Oct</td>
<td>416</td>
<td>0.041</td>
<td>0.142</td>
<td>0.031</td>
<td>0.051</td>
<td>0.010</td>
<td>0.099</td>
<td>0.567</td>
</tr>
<tr>
<td>Nov</td>
<td>428</td>
<td>0.094</td>
<td>0.140</td>
<td>0.056</td>
<td>0.051</td>
<td>0.019</td>
<td>0.110</td>
<td>0.617</td>
</tr>
<tr>
<td>Dec</td>
<td>406</td>
<td>0.084</td>
<td>0.079</td>
<td>0.039</td>
<td>0.030</td>
<td>0.017</td>
<td>0.086</td>
<td>0.493</td>
</tr>
<tr>
<td>Pool</td>
<td>2,112</td>
<td>0.084</td>
<td>0.113</td>
<td>0.039</td>
<td>0.035</td>
<td>0.014</td>
<td>0.085</td>
<td>0.540</td>
</tr>
</tbody>
</table>

N = no. of wild sperms treated; Fl = frequency of males carrying one or more cosmopolitan inversions; Mi = mean no. of cosmopolitan inversions per male.
Table 3. Correlation coefficients from chromosomal inversion associations between chromosome arms and between 2nd and 3rd chromosome.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>2L - 2R</td>
<td>-0.109 ± 0.065</td>
<td>-0.049 ± 0.055</td>
<td>-0.088 ± 0.073 ± 0.011</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3L - 3R</td>
<td>-0.027 ± 0.041</td>
<td>-0.078 ± 0.415**</td>
<td>0.219** ± 0.098 ± 0.095***</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2L : 3L</td>
<td>-0.044 ± 0.011</td>
<td>0.033 ± 0.201**</td>
<td>0.076 ± 0.051 ± 0.043</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2L : 3R</td>
<td>0.137 ± 0.077</td>
<td>0.043 ± 0.142*</td>
<td>0.013 ± 0.082 ± 0.025</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2R : 3L</td>
<td>-0.038 ± 0.050</td>
<td>0.085 ± 0.106</td>
<td>0.035 ± 0.048 ± 0.025</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2R : 3R</td>
<td>0.031 ± 0.065</td>
<td>0.163* ± 0.039</td>
<td>0.176** ± 0.095 ± 0.031**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 : 3</td>
<td>0.022 ± 0.045</td>
<td>0.081 ± 0.122</td>
<td>0.075 ± 0.069 ± 0.031</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* P<0.05; ** P<0.01; *** P<0.001.

Table 4. Homozygosity test to determine deficiency of homozygotes by differential mortality.

<table>
<thead>
<tr>
<th>Sample</th>
<th>No. Obs.</th>
<th>No. Exp.</th>
<th>t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aug.</td>
<td>4</td>
<td>5.99</td>
<td>18.956 ± 1.366 0.086</td>
<td></td>
</tr>
<tr>
<td>Sep.</td>
<td>4</td>
<td>6.76</td>
<td>22.971 ± 0.814 0.792</td>
<td></td>
</tr>
<tr>
<td>Oct.</td>
<td>3</td>
<td>7.30</td>
<td>22.350 ± 1.668 0.046*</td>
<td></td>
</tr>
<tr>
<td>Nov.</td>
<td>5</td>
<td>9.77</td>
<td>27.529 ± 0.394 0.653</td>
<td></td>
</tr>
<tr>
<td>Dec.</td>
<td>5</td>
<td>4.75</td>
<td>17.291 ± 0.284 0.388</td>
<td></td>
</tr>
<tr>
<td>TOTAL</td>
<td>21</td>
<td>34.77</td>
<td>106.624 ± 0.396 0.345</td>
<td></td>
</tr>
</tbody>
</table>

* P, probability.


Members of the family Drosophilidae are characterized by their tendency to forage on yeasts, other fungi, and bacteria associated with fermenting and decaying vegetable matter. The few known exceptions have been reviewed by Ashburner (1981), and Lachaise & Tsacas (1983). In the genus Drosophila itself, reports of facultative utilization of other substrates, as opposed to permanent shifts (such as the simulivora subgroup which has evolved predatory aquatic larvae, or D.carcinophila and D.endobranchia which live and reproduce in the excretory canals of land crabs), have been restricted to D.melanogaster, D.buskii, and D.latifaciesformis. These species have been reported breeding successfully in the wild on dead Lepidoptera caterpillars. Whether this ability is a species characteristic in each case or has only evolved in populations in certain habitats in particular geographical areas where there is strong selection for alternative substrates has not been addressed.

Apparently no account exists, anecdotal or otherwise, of Drosophila hydei breeding on alternative substrates. Here we report examples of this species reproducing on a dead cicada (Tibecen chloromera), a dead spider (Nuctenea cornuta), the carcasses of other adult hydei, other hydei pupae, and even ground beef (Bos taurus). The species identification of the cicada was not absolutely certain because of its deteriorated condition when the identification was made. The first example of facultative entomophagy came to our attention in an experiment in which adult hydei were to serve as prey for spiders. These adults were placed in one pint specimen jars containing single female spiders. Moisture was supplied by a small piece of damp sponge (2 cm x 2 cm x 1 cm) but no food source was provided for the spider. Twenty-four hours later the cicada was dead. It was not, however, killed by the spider nor did the spider feed on it. Six days after the death of the cicada, what appeared to be Drosophila puparia were noticed on the side of the jar. Eleven days after the cicada died and 12 days after it had been placed in the bottle, adult D.hydei began to emerge. This is decidedly faster than they emerge on the cornmeal-molasses-agar laboratory medium (17 days) or on Carolina instant medium (15 days). A total of 30 adults emerged over a four-day period. The possibility that the cicada had been previously parasitized by a Drosophila-like fly was ruled out by a careful comparison of the emerging adults with laboratory hydei stock.

Once our attention had been focused on this instance of entomophagy we noticed larvae and puparia of D.hydei in several other jars. In some cases the only food available to the larvae were the carcasses of dead adults. In other cases the spider had molting adding its exoskeleton as a potential food source. In one instance the spider itself died and was thus available for consumption by the hydei larvae. There is no evidence that the larva attempted to utilize the sponge as a food source.

When feeding on adult hydei carcasses the larvae were able, either by themselves or with the help of bacteria, to completely solubilize the adult exoskeletons except for the wings and the heads. In instances in which the spiders had molting, their exoskeletons were worked over by the larvae and partially solubilized. The larva which contained the dead spider had only a few living Drosophila remaining at the time the spider died. Consequently, only a few eggs were laid. Nonetheless three larvae developed, were able to solubilize the spider's abdomen, pupariate, and emerge as adults. These larvae appeared fully robust and vigorous. We cannot exclude the possibility of bacterial hydrolysis of the tissues and exoskeletons in these examples. However, the fact that they do not solubilize in the absence of larval activity leads us to conclude that the larvae themselves are secreting digestive enzymes onto the carcasses. It has been observed that larvae in the cardini group cannibalize their own pupae in laboratory cultures by producing entry holes into the puparia of other individuals (Heed & Krishnamurthy 1959). In that instance, too, it seems plausible that those larvae were secreting enzymes extra-somatically in order to gain entry. The larvae of hydei will also cannibalize puparia, sometimes pupariating within the original puparium. Larvae...
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provided with puparia as the only food source appear superficially to gain entry by a combination of chemical and mechanical attack.

Given hydei's quite surprising success in breeding on insects and spiders, we attempted to culture them on raw ground beef as a substrate. A small amount of raw ground beef was placed in a jar with some breeding adults. Females appeared reluctant to oviposit on this substrate, but nonetheless some eggs were laid and larvae hatched. After two days the adults were removed so that dead adults could not serve as substrate. For the first 10 days the larvae spent much time wandering about the jar apparently looking for a more appropriate food source. They seemed to be both attracted to, and repelled by, the ground beef. They did not grow well and were not robust and vigorous. In the length of time it took for adults to emerge from the cicada (11 days) these larvae had only developed to the second and early third instar stages.

However, after the 10th day the substrate seemed to become much more palatable and some of the larvae began to flourish. By the 26th day adults began to emerge, taking more than twice as long to develop as the flies developing on the cicada. On the 20th day a second batch of breeding adults was added to the jar and removed two days later. These females found the ground beef, which by now had a thin film covering it, a very attractive oviposition site and littered it with eggs. The thin film presumably was the result of bacterial action. The larvae immediately took to this aged ground beef, but the length of time required before adults began to emerge was still much longer than for the cicada or the lab media. Nonetheless, the developmental time was shortened from 26 days to 20 days.

The behavior of hydei on ground beef is consistent with the widely held belief that Drosophila actually feed on the yeast and/or bacteria that are growing on the fermenting and decaying substrate. However, in the case of the cicada the larvae apparently were feeding on the insect tissues directly. Six of the adults that emerged from the cicada were weighed and averaged 2 mg. If we assume that the puparium and larval sheaths weigh another 0.5 mg, an emerging adult accounts for 2.5 mg of biomass. Thus 30 adults would account for 75 mg of Drosophila biomass. The nine third instar larval that failed to pupariate properly probably account for another 10 or 15 mg for a grand total of 90 mg. The cicada exoskeleton, after it had been quite thoroughly cleaned out by the hydei larvae weighed 420 mg. The mean weight of 20 living cicadas was 1330 mg. Thus, a reasonable estimate is that the cicada contained from 900 to 1000 mg of consumable biomass. Based on the standard assumption that there is only a 10% efficiency in converting the biomass of one trophic level to that of a higher one, 900 mg of cicada could only have supported the production of 90 mg of Drosophila if the larvae consumed most of the cicada tissues directly, with very little of it having been first converted into the biomass of microorganisms. If the cicada had been first converted into microorganisms which were then consumed by the larvae, 9000 mg (9 grams) of consumable cicada biomass would have been required to produce 900 mg of microorganisms to be converted into 90 mg of Drosophila, and a 9 gram cicada (or really 13.3 grams including the exoskeleton) is out of the question. Even these rough calculations strongly suggest that the hydei consumed the cicada tissues directly.

Accepting this evidence that most of the tissues were consumed directly, questions arise concerning the role of hydei digestive enzymes. Do these enzymes accomplish the initial breakdown of Arthropod tissues, or is the initial solubilization of tissues accomplished by cellular autolysis and/or microbial digestion? If solubilization and utilization depend on hydei enzymes are these enzymes secreted onto the substrates to be digested? We conclude that not only are these larvae producing their own digestive enzymes, but that they are indeed secreting them extra-somatically onto the substrates being consumed. The evidence for these conclusions is based on (1) our calculation that there was not much excess cicada biomass to spare on microorganisms, (2) our observation that adult hydei carcasses and spider exoskeletons were not solubilized in the absence of larval activity, and (3) the fact that the larvae turned the viscera of the cicada, the spider, and the adult flies into juicy messes, much as they do the culture medium. In addition, extra-somatic secretion of digestive enzymes is consistent with the very large size of the salivary glands relative to the rest of the digestive system, a characteristic of the entire order, Diptera. Unfortunately, little is known about the role of salivary glands in the production of digestive enzymes. Extra-somatic secretion of digestive enzymes, whether by the salivary glands or not, is also consistent with the observation that Drosophila larvae, as well as those of other Dipterans, frequently cluster together in writhing masses as though there is a collective effort to solubilize the substrate. A third question is, if the larvae do produce enzymes for the initial solubilization of arthropod and mammalian substrates, are these the same enzymes used to digest microorganisms and plant material, or a different set specifically produced for this purpose?

The ability of the hydei to breed on unusual substrates in the laboratory does not insure their ability to compete for these resources in the wild. Nonetheless, it is plausible that this ability may enable hydei to opportunistically exploit unusual niches and thereby be part of the reason that hydei is a cosmopolitan species. These results also suggest that the ability of Drosophila in general to use a variety of resources has been underestimated, and their dependence on yeast and microorganisms exaggerated.

Salam, M.A.* Moscow State University, Moscow, USSR. The inhibiting effect of actinomycin-D on the yield of N-nitroso-N-ethylurea-induced second chromosome recessive lethal mutation in D.melanogaster.

Table.

<table>
<thead>
<tr>
<th>Mating pairs</th>
<th>Total no. of chromosomes tested</th>
<th>% of lethals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>Control</td>
<td>1009</td>
</tr>
<tr>
<td>Control</td>
<td>NEU</td>
<td>1245</td>
</tr>
<tr>
<td>Control</td>
<td>ACM-D</td>
<td>1171</td>
</tr>
<tr>
<td>ACM-D</td>
<td>Control</td>
<td>1141</td>
</tr>
<tr>
<td>ACM-D</td>
<td>NEU</td>
<td>1087</td>
</tr>
<tr>
<td>Control</td>
<td>ACM-D + NEU</td>
<td>1156</td>
</tr>
</tbody>
</table>

Domodedovskaya-32 (D-32) males and the Curly Lobe/Plum (CyL/Pm) females were used for the experiment. For the treatment with ACM-D flies were starved for 24 hr, and then a properly drenched filter paper in ACM-D solution (1.5 mg ACM-D + 2 mg sugar + 100 ml distilled water) was kept in the vials of flies. Treatment with NEU (30 mg NEU in 1 ml distilled water) was made by exposing flies to NEU-vapour in the air tight dessicator. Duration of treatment was 24 hr at 25°C. For the detection of the second chromosome recessive lethal mutation the classical CyL/Pm method was used. Results pooled for experiments are tabulated in the Table below.

The present study indicates that ACM-D holds the ability to reduce the NEU-induced second chromosome recessive lethal mutation.

Acknowledgements: The author is grateful to M.M. Aslaniyan, Assoc. Prof., Dept. of Genetics and Breeding, Moscow State University, Moscow, USSR, for guidance in experiments.

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Sanchez-Refusta, F. and E. Garcia-Vazquez.
University of Oviedo, Spain. Correlation between inversion polymorphism and extra bristles variation in isofemale lines of D.melanogaster.

In this work we perform the correlation in D.melanogaster between inversion polymorphism and the polygenic character: "extra bristles" both dorsocentrals and scutellars.

Wild flies were captured in banana-yeasted traps and belong to ten natural populations geographically apart from each other by at least 10 Km. Their offspring were analysed in first generation in the lab. This analysis reveals the variation for extra bristles that generally underlies hidden in natural populations, where individuals usually present a normal phenotype (Figure 1). Isofemale lines are classed by counting 60 males and 60 females into three groups (classes) according to the rate of individuals carrying extra chaetae: Class I gathers those lines with less than 5% of individuals with extra bristles (Low variability); Class II from 5% to 20%; and Class III more than 20% (High variability). Inversions are determined through direct observation of the chromosomes of one single larva from each population.

There are several studies which relate inversion polymorphism with morphometrical variation for different characters and species: White & Andrew (1960, 1962) and White et al. (1963) for body size in Moraba scurra; Prevosti (1965, 1967) and Aiguaè & Serra (1980) for wing length in D.subobscura; Butlin et al. (1982) for wing length in Coelopa frigida males.

Figure 1. Normal phenotype of bristles in natural populations (A), and some phenotypes of extra bristles (B).
Table 1. Correlation between the two levels of variability. L.E.= % of lines with extra bristles (classes I and II). I.I.= % of individuals with inversions. N.L.= number of lines. \( r = 0.893 \times 8 \text{ df.} \\

<table>
<thead>
<tr>
<th>Sample</th>
<th>L.E.</th>
<th>I.I.</th>
<th>N.L.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Villaviciosa</td>
<td>39.02</td>
<td>46.34</td>
<td>41</td>
</tr>
<tr>
<td>Somio</td>
<td>42.86</td>
<td>70.33</td>
<td>91</td>
</tr>
<tr>
<td>Soto del Barco</td>
<td>58.33</td>
<td>66.67</td>
<td>48</td>
</tr>
<tr>
<td>Proaza</td>
<td>64.44</td>
<td>75.55</td>
<td>45</td>
</tr>
<tr>
<td>Sandiche</td>
<td>65.63</td>
<td>82.81</td>
<td>64</td>
</tr>
<tr>
<td>Trubia c.</td>
<td>66.18</td>
<td>83.82</td>
<td>68</td>
</tr>
<tr>
<td>Aviles c.</td>
<td>69.05</td>
<td>92.86</td>
<td>42</td>
</tr>
<tr>
<td>Aviles nc.</td>
<td>72.73</td>
<td>87.27</td>
<td>55</td>
</tr>
<tr>
<td>Trubia nc.</td>
<td>73.08</td>
<td>88.46</td>
<td>26</td>
</tr>
<tr>
<td>Celorio</td>
<td>77.78</td>
<td>83.33</td>
<td>36</td>
</tr>
<tr>
<td>Average</td>
<td>62.91</td>
<td>77.74</td>
<td></td>
</tr>
</tbody>
</table>

Table 2. Distribution (in percent of chromosomes in each class) of each inversion in the three classes of lines. N.C. = number of chromosomes. \( d = \text{N.S.} \\

<table>
<thead>
<tr>
<th>Classes</th>
<th>2Lt</th>
<th>2RNS</th>
<th>3LP</th>
<th>3RP</th>
<th>3RC</th>
<th>NC</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>17.91</td>
<td>11.19</td>
<td>14.43</td>
<td>19.65</td>
<td>9.20</td>
<td>402</td>
</tr>
<tr>
<td>II</td>
<td>20.08</td>
<td>10.24</td>
<td>18.11</td>
<td>15.75</td>
<td>16.14</td>
<td>508</td>
</tr>
<tr>
<td>III</td>
<td>22.95</td>
<td>17.21</td>
<td>15.57</td>
<td>16.39</td>
<td>20.49</td>
<td>122</td>
</tr>
</tbody>
</table>

\( X^2 \text{df} = 1.67 \times 4.77 \times 2.29 \times 2.47 \times 13.92 \times \text{**} \)

Table 1 shows the correlation between the two levels of variability measured as percent of lines with extra bristles (Classes I and II) as well as percent of individuals carrying at least one inversion. This correlation is proved to be positive and significant \((p<0.01)\). Population variation for both levels undergoes increases and decreases of the same sign.

When we class the isofemale lines by their range of extra bristles frequency, we find that only inversion 3RC is unequally distributed among classes I, II and III: the higher rate of flies with extra chaetae; the higher frequency for this inversion is found (Table 2). Conversely this effect is not found for any of the other cosmopolitan inversions.

This 3RC effect can be related to the general chromosome 3 influence reported by other researchers in experiments dealing with selection for increase of the number of extra bristles (Scowcroft 1966; Whittle 1969). Rubio & Albornoz (1982) find in chromosome arm 3R a segment carrier of polygenic loci that enhances the expression of extra bristles in a selected line. This segment lies from 70.0 to 100.7 cm on 3R arm and overlaps 3RC inversion which lies from 84.7 to 110.0 cm. This fact suggests that in the natural populations analysed, In(3R)C carries as a polygenic complex, some factors that favour the expression of extra macrochaetae.

References:

Frequency dependent sexual activity.

Preliminary experiments have shown that the so-called "rare male mating advantage" was particularly evident when wild Canton S flies are in competition with flies from the well known mutant strain rucuca, whose curled wings could perhaps be a visual stimulus for the sexual activity of the wild males (Kaningini et al. 1986, this issue). The estimation of the "rare male advantage" was done using Wattiaux's equation (1964): a logarithmic transformation changes the sigmoid curves of mating successes into straight lines, which differ in slope or in elevation, and can be compared by covariance analysis (Snedecor 1956). Wattiaux suggested comparing the following regression lines: (1) homogamic and heterogamic matings, (2) matings of the males of both strains in competition, (3) matings of the females of both strains in competition. This formula has already been applied to sexual competition experiments where both types were in equal number (Elens et al. 1974). In the present experiment, the same method is applied to the sexual activities of the two types in competition, when their relative frequencies differ.

Besides the wild Canton S and rucuca strains, the blind flies of the ora strain have been used, in competition with the rucuca ones; as they are blind, the possibility of any visual stimulus can be excluded. The method of observation has been previously described: matings are observed in a small mating chamber, and individuals may mate more than once (Elens 1958; Elens & Wattiaux 1964; Ehrman 1966; Petit 1968). A total of 30 pairs of virgin flies, 4-5 days old, were used for each test, but the relative frequencies of both genotypes differed: 5/25, 10/20, 15/15, 20/10, 25/5 (the frequencies being the same for both sexes of the
Figure 1. Cumulated percentage of the "male sexual activity" of wild Canton S and rucuca flies: the wildtype males copulate more than once, when "rare". Relative frequencies: A = 5 +/-25 ru; B = 10 +/-20 ru; C = 15 +/-15 ru; D = 20 +/-10 ru; E = 25 +/-5 ru.

Figure 2. Relationships between the competing types wild Canton S and rucuca, when their relative frequencies differ: columns, from left to right = 5 +/-25 ru; 10 +/-20 ru; 15 +/-15 ru; 20 +/-10 ru; 25 +/-5 ru. Abscissa: time in minutes. Ordinate: $\ln dx/N - X$. $\ln$ is the naperian logarithm, N is the total number of flies of one sex and one type, dx the number of these flies mating in the considered interval of time dt (15 min), and X the number of these flies having already mated before.
Figure 3. Relationships between the competing types ora and rucuca, when their relative frequencies differ: columns, from left to right = 5 ora/25 ru; 10 ora/20 ru; 15 ora/15 ru; 20 ora/10 ru; 25 ora/5 ru. Abscissa: time in minutes. Ordinate: ln(dx/N - X). ln is the napierian logarithm, N is the total number of flies of one sex and one type, dx the number of these flies mating in the considered interval of time dt (15 min), and X the number of these flies having already mated before.

As in previous experiments, the greatest differences concern the "male sexual activities": the wild-type males are always more active. When they are "rare", they evidently copulate more than once (Fig. 1). The regression lines characterizing the competition with the mutant rucuca of the wild type flies and of the blind flies ora are given in Fig. 2 and 3. The most interesting observation is that the "frequency dependence" of both the male and the female activities is a much more general phenomenon than was expected. A "rare male advantage" is generally observed, not only for the wild or the ora flies but even for the rucuca ones. The difference is highly significant (P<0.001) between the male activity of the wildtype flies at the frequency 5 +/25 ru and at the other frequencies (P<0.001). The difference is significant (P at least <0.05 and sometimes <0.01 or even <0.001) between the activity of the ora males at the same type). 24 hours before the test, groups of 30 flies of the same sex, assembled in numbers corresponding to the above proportions, were stored in separate vials. The total number of flies per vial was always the same; such a habit avoids any bias which could result from differences in the number of stored flies. For the test, the females of both types were introduced together in the mating chamber. Afterwards, the male of both types were similarly introduced, in the same frequency as the females. All the observations were done simultaneously for the five relative frequencies, at 25°C, 1000 lux, and 40-60% relative humidity, with 10 repetitions for each test.
the frequency 5 ora/25 ru and the other ones. Even the rucuca males can be significantly more active when rare (Fig. 2, 0 and Fig. 3 0), although they are never "advantaged" in the competition. The wild females are significantly more active, when rare, than the rucuca ones (Fig. 2, F); they are less active when the rucuca flies are rare (Fig. 2, J). For the intermediate frequencies, the differences between the two types are never significant (Fig. 2). In the competition between ora and rucuca, the rucuca females are more active than the ora ones only when they are "rare" (Fig. 3, J); at the other frequencies, the activity of the ora females is always higher (Fig. 3). A "rare female advantage" is observed for the rucuca flies as well as for the wild ones (Fig. 2, F and J), and for the ora as well as for the rucuca ones when they are in competition (Fig. 3, F and J). If one compares the relative frequencies of "homogamic" and "heterogamic" matings, one sees that the frequency of "homogamic" mating is significantly higher only when the rucuca flies are "rare" (Fig. 2, D & E; Fig. 3, E). The "heterogamic" matings are more frequent (P<0.05) only when the relative frequency between wild and rucuca flies is: 20 rucuca for 10 wild (Fig. 2, B). As a general conclusion, one could say that in our experiment the sexual activity appears as "frequency dependent" (for females as well as for males) in almost every condition, whatever the causes of such a phenomenon.


Sharma, A.K. and K.S. Gill. Punjab Agricultural University, Ludhiana, India. Heterosis, homeostasis and genotype x environment interaction for acid phosphatase in D.malerkotliana.

Acid phosphatase (ACPH), a dimeric enzyme in D.malerkotliana, is controlled by a single locus Acph-1. We prepared homozygous stocks of two alleles Acph-11.05 and Acph-10.90, hereafter referred to as fast (F) and slow (S) depending upon their mobility towards anode. Biochemical properties of various enzyme preparations were studied at 25±1°C. There was no significant difference in the two sexes for enzyme activity per unit enzyme concentration, specific activity, Vmax and Km values. At pH 5.0 specific activity of the enzyme in crude extracts of S/S, F/F and F/S flies is 5.85, 10.78 and 16.39, respectively. Higher specific activity of the enzyme in crude extract of heterozygotes was also observed over a range of nine substrate concentrations (0.1 mM to 10.0 mM) and a range of 20 pH values (4.7 to 7.1). Thus with respect to specific activity, heterosis is being exhibited by heterozygotes. Vmax value of enzyme from S/S, F/F and F/S genotypes was found to be 1.51, 1.91 and 3.51 and Km value 0.50, 0.39 and 0.31, respectively. Thus enzyme from heterozygote has a better quality. For slow, fast and hybrid allozymes, partially purified from heterozygotes, specific activity was 4.50, 8.20 and 9.75, respectively. Thus was shown the higher efficiency and the better quality of the heterodimeric allozyme, which explains the heterosis observed in heterozygotes.

For homeostatic studies, specific activities in crude extracts from three genotypes were determined at pH values ranging from 4.7 to 7.1. The optimum pH of crude enzyme preparations from S/S, F/F and F/S genotypes was 5.9, 6.2 and 6.2, respectively. Partially purified slow, fast and hybrid allozymes showed peak activity at pH 5.9, 6.2 and 6.4, respectively. The enzyme from F/F extract showed a better homeostatic ability than that from S/S extract at pH value lower than the optimum pH, after which the relation is reversed. The extract from heterozygotes, however, exhibited higher stability than that from either homozygote on each side of the optimum pH. After comparing the homeostatic ability of the enzyme in a 1:1 mixture of crude extracts from the two homozygotes with those described above, it has been concluded that higher homeostatic ability of the heterozygotes is entirely due to the presence of heterodimeric allozyme. Subsequently, it was shown that homeostatic ability of heterodimeric allozyme is higher than that of either homodimeric allozyme. Higher stability of ACPH in heterozygotes thus seems to arise from superior stability of heterodimeric allozyme.

For determining genotype x environment interaction, enzyme activities at different pH values and at different substrate concentrations were measured. It was observed that in most of the cases, for a similar change in pH or concentration of substrate, the enzyme in crude extract of S/S, F/F and F/S flies and partially purified allozymes from heterozygotes showed significantly different response.
Weak points (breaks, constrictions, attenuations, etc.) can be considered as best diagnostic characters for definition of intercalary heterochromatin in squashed polytene chromosomes. They are known since rediscovery of polytene chromosomes in early thirties (see Zhimulev et al. 1982, for ref.). However, it is not known whether they appear as a consequence of squashing the cells or exist in nonsquashed chromosomes. To clarify the situation, we analysed serial sections of nonsquashed salivary gland cells of late third instar larvae of giant (gt/gt) strain.

Salivary glands were dissected in Ephrussi-Beadle solution, then fixed in 2.5% glutaraldehyde, postfixed in 1% osmium tetroxide, dehydrated in ethanol and acetone, and embedded into Epon. Serial sections (ca 1 micron) of whole salivary glands made by Ultratome IV (LKB, Sweden) were put on slides, stained with methylene blue and analysed under light microscope.

On the section of nucleus chromosome twists can mimic weak points; therefore only those chromosome fragments were analysed which were clearly seen before and after weak point. Totally 12 nuclei were studied; weak points in each were mapped according to Bridges map (Table 1, Figure 1). If the exact location of weak point appeared to be difficult, such case was marked as "nonlocated" in the Table.

Weak points were found in 15 chromosome regions; the 35B was found to be broken in three and five regions (11A, 12E, 36D, 42B and 75C) in two different nuclei (Table 1). In all 15 regions, weak points in squashed preparations were found with high frequency (Zhimulev et al. 1982).

In one section thread of ectopic pairing was seen (Fig. 1f).

So, data obtained demonstrate that weak points are rather numerous in nonsquashed chromosomes.


Table 1. Localization of weak points in sections of nonsquashed salivary gland nuclei.

<table>
<thead>
<tr>
<th>No. of nuclei</th>
<th>No. of weak points in nucleus Location</th>
<th>No. of sections studied</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1 non located</td>
<td>12</td>
</tr>
<tr>
<td>2</td>
<td>8 3C, 7C, 11A, 12E, 35B, 36D, 42B, 75C</td>
<td>13</td>
</tr>
<tr>
<td>3</td>
<td>2 35B, 75C</td>
<td>7</td>
</tr>
<tr>
<td>4</td>
<td>3 11A, 12E, 17A</td>
<td>5</td>
</tr>
<tr>
<td>5</td>
<td>3 22A, 42B, 67D</td>
<td>4</td>
</tr>
<tr>
<td>6</td>
<td>2 36D, 64C</td>
<td>5</td>
</tr>
<tr>
<td>7</td>
<td>2 35B, 70C</td>
<td>7</td>
</tr>
<tr>
<td>8</td>
<td>3 7B, 98C, non located</td>
<td>7</td>
</tr>
<tr>
<td>9</td>
<td>1 non located</td>
<td>2</td>
</tr>
<tr>
<td>10</td>
<td>0 ---</td>
<td>5</td>
</tr>
<tr>
<td>11</td>
<td>0 ---</td>
<td>2</td>
</tr>
<tr>
<td>12</td>
<td>0 ---</td>
<td>1</td>
</tr>
</tbody>
</table>

Figure 1. Polytene chromosome weak points in sections of salivary glands are shown with arrows: 12E(a), 35B(b), 98C(c), 11A(d), 42B(e) and 7B(f). Arrow with open head in (f) shows ectopic thread.
and Nallur (South India). Mysore, located along 12°5' N latitude between 76° and 77° E longitude is a planar region, about 450 metres above the sea level with moderate rainfall. Nallur is located at the border of Western Ghats, 120 km from Mysore at an altitude of 900 m above sea level, with heavy rainfall and humid climate. Collections were made at 5 sites about 5 km apart in Mysore and in Nallur during the months of August and September 1985. Fermenting banana seeded with yeast was used as the bait. Collection records are given in Table 1.

A total of 22 species were recorded. Of these, 13 species belong to the subgenus *Sophophora*, 7 species to the subgenus *Drosophila*, one to the subgenus *Dorsilopha* and one to the subgenus *Scaptodrosophila*. *D.n.nasuta* of the *immigrans* species group is abundant in the population of both Mysore and Nallur indicating its ecological versatility. *D.malerkotliana* of the *melanogaster* species group is dominant in all populations of Mysore, but its frequency is remarkably low in the Nallur population. On the other hand, *D.bipictinata* and *D.nagarholensis* dominate Nallur population apart from *D.n.nasuta*.

The faunal constellations of Mysore and Nallur show qualitative and quantitative differences owing to macroenvironmental variations, but such differences found within Mysore populations can only be attributed to variations in microenvironmental factors. Most of the species collected belong either to the *melanogaster* or to the *immigrans* species groups. This is in agreement with the earlier findings of Reddy (1973), Gowda (1979), Prakash (1979) and Muniyappa (1982) and supports the view of Bock & Wheeler (1972), that both these species groups have originated and diversified extensively in South East Asia.

**Acknowledgements:** Authors are grateful to Prof. N.B. Krishnamurthy, Head of the Dept. of Post-Graduate Studies and Research in Zoology, University of Mysore, Manasagangotri, Mysore, for his help and encouragement, and to Prof. T. Okada for his advice in identifying the flies. One of the authors (BVS) wishes to thank UGC, New Delhi, for granting a research fellowship.


**Table 1. Distribution of different species of Drosophila in Mysore and Nallur.**

<table>
<thead>
<tr>
<th>Species</th>
<th>Collection sites</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MYSORE</td>
</tr>
<tr>
<td></td>
<td>I</td>
</tr>
<tr>
<td>Subgenus: <em>Sophophora</em></td>
<td></td>
</tr>
<tr>
<td><em>D.malerkotliana</em></td>
<td>516</td>
</tr>
<tr>
<td><em>D.bipictinata</em></td>
<td>115</td>
</tr>
<tr>
<td><em>D.parabipictinata</em></td>
<td>2</td>
</tr>
<tr>
<td><em>D.pseudoanamassae</em></td>
<td>5</td>
</tr>
<tr>
<td><em>D.takahashii</em></td>
<td>1</td>
</tr>
<tr>
<td><em>D.eugracilis</em></td>
<td>3</td>
</tr>
<tr>
<td><em>D.rajasekari</em></td>
<td>7</td>
</tr>
<tr>
<td><em>D.jambulina</em></td>
<td>1</td>
</tr>
<tr>
<td><em>D.punjabiensis</em></td>
<td>--</td>
</tr>
<tr>
<td><em>D.mysorensis</em></td>
<td>3</td>
</tr>
<tr>
<td><em>D.agambensis</em></td>
<td>--</td>
</tr>
<tr>
<td><em>D.nagarholensis</em></td>
<td>--</td>
</tr>
<tr>
<td><em>D.anomelani</em></td>
<td>2</td>
</tr>
</tbody>
</table>

| Subgenus: *Drosophila* |
| *D.n.nasuta* | 543   | 673   | 3     | 116    | 1336  | 169   |
| *D.s.neonausta* | 2     | --    | --    | --     | 2     | --    |
| *D.formosana* | --    | 15    | --    | 2      | 17    | 15    |
| *D.hypogoesta* | 3     | 18    | --    | --     | 21    | 11    |
| *D.nigra* | 10    | 17    | 18    | 5      | 50    | --    |
| *D.brindavani* | --    | 53    | 10    | --     | 63    | --    |
| *D.repleta* | 8     | 44    | --    | --     | 52    | --    |

| Subgenus: *Dorsilopha* |
| *D.busckii* | 1     | 15    | --    | 16     | --    |

| Subgenus: *Scaptodrosophila* |
| *D.coracina* | 6     | 12    | 5     | --     | 22    | --    |

Total no. of flies 1227 3023 141 399 4708 375

Total no. of species 17 19 7 8 21 9

Shyamala, B.V. and H.A. Ranganath. University of Mysore, India. Metaphase karyotype of *Drosophila nagarholensis*. **melanogaster** species group of *Drosophila*. Its karyotype is described here for the first time. Permanent air dry preparations of the neural ganglia were made as described by Ramachandra & Ranganath (1985). The male and female karyotypes are represented in Figures 1 and 2. The metaphase karyotype reveal the following: The diploid chromosome number (2n) is 8. It is made up of two pairs of metacentric chromosomes, a pair of dot chromosomes and a pair of acrocentric X chromosomes. In male, one of the X chromosomes is replaced by a submetacentric Y chromosome. The metaphase karyotype of
D. nagarholensis resembles the basic metaphase pattern of the montium species subgroup of the melanogaster species group of Drosophila as reported by Baimai (1980).

Acknowledgements: Authors are thankful to Prof. N.B. Krishnamurthy, Head of the Dept. of Zoology, for his help and encouragement. We are thankful to Mr. M.G. Vasudeva Rao, for preparing photographs. BVS is thankful to UGC, New Delhi, for granting a research fellowship.


Last year we reported that X chromosomes which had been unstable, as judged by high recessive lethal mutation rates, had lost their propensity to mutate to the lethal condition. These chromosomes were derived from an unstable X chromosome discovered by Lim (1979, 1981), which, in addition to high mutability, accumulated structural rearrangements and was able to confer these instabilities on previously stable X chromosomes (Lim et al. 1983). This last phenomenon, called homologue destabilization, occurred independently of recombination between the stable and unstable X chromosomes and therefore suggested the action of a transposable element. The transposable element postulated to explain these instabilities has been called the L factor, where the letter "L" signifies its lethal-inducing properties. Evidently, this element has either been lost or inactivated in the stocks whose previously unstable X chromosomes have become stable.

Our recent efforts to study the instabilities attributed to the L factor have focused on lethal X chromosomes derived from Lim's unstable X, and on lethal X chromosomes which had been destabilized by it. Because these chromosomes were already lethal, we could not study their recessive lethal mutation rates; instead, we investigated their ability to destabilize nonlethal, stable X chromosomes in heterozygous females. As before, the primary criterion of instability was the propensity to mutate to the lethal condition. We therefore determined the recessive X-linked lethal mutation rates of the chromosomes which had been paired with the lethal and putatively unstable X's. A high mutation rate for one of these chromosomes would indicate that the lethal X had destabilized this chromosome, presumably through the action of the L factor.

To gather the data, we conducted several experiments following the same general plan. Males from a stock with a marked, stable X chromosome (symbolized "X") were mated at 25° to single FM6, y^{71} dm B/\kappa^* females, where \kappa^* denotes a marked, lethal X chromosome derived from Lim's unstable X or from an X chromosome destabilized by it. In the next generation, the \kappa^*/"X" females were mated to their FM6 brothers to produce progeny (also at 25°), from which we selected only the males with the nonrecombinant "X" chromosome. The markers on the \kappa^* and "X" chromosomes were chosen so that we could be sure that the males recovered from the \kappa^*/"X" females had a nonrecombinant "X" chromosome. These males were then mated individually to FM7/sc^7 \kappa females (see Simmons et al. 1980, for the full genotype of this balancer stock) to begin the X-linked lethal test. In the next generation, FM7/"X" females who had mated with their FM7 brothers were placed individually in culture tubes to produce progeny. If males with the "X" chromosome did not emerge, we tested their FM7/"X" sisters in mass cultures to determine whether or not the "X" chromosome had acquired a recessive lethal mutation. The methodological details for these X-linked lethal tests are given fully in Simmons et al. (1980). In addition to the "X" chromosomes that had been paired with \kappa^* for one generation, we tested "X" chromosomes extracted directly from the stable stock. These latter tests documented the intrinsic stability of the "X" chromosomes used in the destabilization experiments.

Five different \kappa^* chromosomes were tested, each in combination with one of three "X" chromosomes. The full genotypes of the potential destabilizing chromosomes, as well as those of the target "X" chromosomes, are given in Table 1. This table also gives the experimental results. Complementation...
Table 1. Lethal mutation rates of stable and putatively destabilized X chromosomes.

<table>
<thead>
<tr>
<th>Target &quot;X&quot; Chromosome</th>
<th>Destabilizing £* Chromosome</th>
<th>No. Males tested</th>
<th>No. Chromosomes tested</th>
<th>No. Independent events</th>
<th>Number of lethals</th>
<th>Mutation rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>cho cv m</td>
<td></td>
<td>373</td>
<td>2,739</td>
<td>2</td>
<td>2</td>
<td>0.07</td>
</tr>
<tr>
<td>cv v f</td>
<td></td>
<td>397</td>
<td>3,795</td>
<td>5</td>
<td>5</td>
<td>0.16</td>
</tr>
<tr>
<td>H7 = y^{59b} z w^1 ct^6 f</td>
<td></td>
<td>426^b</td>
<td>3,628</td>
<td>0</td>
<td>0</td>
<td>---</td>
</tr>
<tr>
<td>cho cv m</td>
<td></td>
<td>615</td>
<td>5,119</td>
<td>9</td>
<td>9</td>
<td>0.18</td>
</tr>
<tr>
<td>H7</td>
<td></td>
<td>104^b</td>
<td>975</td>
<td>14</td>
<td>1.43</td>
<td></td>
</tr>
<tr>
<td>cv v f</td>
<td></td>
<td>551</td>
<td>5,530</td>
<td>25</td>
<td>32</td>
<td>0.58</td>
</tr>
<tr>
<td>cv v f</td>
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<td>244</td>
<td>2,065</td>
<td>2</td>
<td>2</td>
<td>0.09</td>
</tr>
<tr>
<td>cv v f</td>
<td></td>
<td>178</td>
<td>1,591</td>
<td>3</td>
<td>3</td>
<td>0.19</td>
</tr>
</tbody>
</table>

a = Data from Simmons et al. 1985, DIS 61:157-159.  b = For additional data, see text.

Lim et al. (1983) had established that chromosomes I.14, II.10 and V.16.38 each had recessive lethal mutations uncovered by Df(1)ct^{78} (breakpoints 6F1-2 and 7C1-2); the lethal mutations on these chromosomes were also complemented by the y^{+} ct^{+} Y chromosome, suggesting that these were the only lethal mutations present. These results were confirmed immediately prior to the beginning of the destabilization experiments. Lim et al. (1983) showed that the lethal-containing region on these three chromosomes is a hotspot for L factor activity. The other two £* chromosomes, III.18 and V.16.20, also had lethal mutations in this region, but by the time of these experiments, they had lost them. Their lethal mutations were outside the X chromosome segments carried by the y^{+} ct^{+} Y chromosome. It should also be noted that chromosomes V.16.20 and V.16.38 were derived from the same original lethal X chromosome; evidently, during the four years that elapsed between the identification of the original lethal chromosome and the time of these experiments, one lethal mutation reverted and another occurred, possibly as a result of L factor-induced instability.

As can be seen from the table, the "X" chromosomes are intrinsically quite stable. After having been paired with an £* chromosome, only H7 showed any indication of an elevated mutation rate. Therefore, further tests were performed with this chromosome, using, as in the first set of experiments, the same £* chromosome, II.10, in an attempt to destabilize it. In these additional tests, 1,463 H7 chromosomes were screened and only two proved to have acquired recessive lethal mutations. We also tested the destabilizing ability of the FM6 chromosome from the II.10 stock, just in case it had acquired L factor activity. H7 males were recovered from FM6/H7 females and used in a set of X-linked lethal tests. No lethals were detected in a sample of 1,166 H7 chromosomes screened. Therefore, we conclude that none of the £* chromosomes has the ability to destabilize the specific target "X" chromosomes that were tested.

These results indicate that the five unstable or destabilized X chromosomes that we studied could not destabilize chromosomes paired with them for one generation. One explanation is that the target "X" chromosomes were immune to the destabilizing action of the L factor; however, in light of last year's report on the stabilization of several unstable X chromosomes, a more likely explanation is that the putatively unstable chromosomes that were tested also had become stable. These manifestly negative results may be turned to good use when the mutational hotspot for L factor activity has been characterized at the molecular level. It will then be possible to examine this locus in the stabilized X chromosomes and to infer the reasons for their stabilization.

Acknowledgements: This work was supported by the National Institute of Environmental Health Sciences (R01 ES01960). M. Boedigheimer, C. McLarnon, R. Morrison and J. Zunt provided assistance.


*Drosophila ananassae* shows spontaneous crossing over in males (Kikkawa 1938; Moriwaki 1940; Mukherjee 1961; Ray-Chaudhuri & Kale 1966; Kale 1969; Hinton 1970). It has been suggested by various workers (Mukherjee 1961; Kale 1969; Hinton 1970; Moriwaki et al. 1970) that this extraordinary phenomenon is meiotic in origin. A large number of mutants are known in this species and linkage maps have been constructed (Moriwaki & Tobari 1975). *D. ananassae* presents a high degree of chromosomal polymorphism (for references, see Singh 1985). The three inversions, namely subterminal (alpha or ln(2L)A) in 2L, terminal (delta or ln(3L)A) in 3L and basal (eta or ln(3R)A) in 3R, are coextensive with the species. The location of these inversions is shown in Figure 1. When the strains are maintained in the laboratory, these inversions have been found to persist for several years (Singh 1982, 1983). The suppressive effect of inversion heterozygosity on crossing over in *D. ananassae* has been indicated earlier (Moriwaki 1940; Mukherjee 1961; Hinton 1970). Singh (1973, 1974) reported the rate of crossing over between delta and eta inversions in the third chromosome in different strains of *D. ananassae*.

In this note we report the results of our preliminary study on crossing over in females and males of *D. ananassae* under different karyotype combinations by using two wild type stocks and a triple recessive mutant stock.

**cu b se mutant stock:** The three markers, cu (curled wings), b (black body colour) and se (sepia eye colour) are located on the second chromosome. Kale used this trio of chromosome 2 markers in his study of crossing over. The cytological examination of this stock revealed that 2L is homozygous for AL inversion (AL/AL) whereas both the arms of third chromosome are homozygous for standard gene sequence (3L-ST/ST; 3R-ST/ST).

**Wild stocks:** Two wild stocks were used: (1) VN-ST; it is homozygous for standard gene order in 2L, 3L and 3R, and (2) VN-AL; it is homozygous for alpha gene arrangement in 2L (AL/AL) but homozygous for standard gene sequence in 3L and 3R (3L-ST/ST; 3R-ST/ST).

The results show that crossing over is completely absent in the first region (cu - b) in females and males when they are heterozygous for inversion (ST/AL). In the second region (b - se), the frequency of crossing over is above 40 percent in females and less than 1 percent in males. When F1 flies are homozygous for alpha inversion (AL/AL), crossovers are found in both the regions in both the sexes. In the first region the rate of crossing over is nearly 30 percent and second region shows crossing over above 40 percent in females. In males crossing over is less than 1 percent in both the regions.

Further experiments to study intra- and interchromosomal effects of inversions on crossing over in *D. ananassae* are in progress.

**Acknowledgements:** The financial support from the Dept. of Science & Technology, New Delhi, is thankfully acknowledged. We also thank Miss Sujata Chatterjee for her help provided during this study.

**References:**


Thompson (1985) suggested that among the factors which might influence the rate at which flies emigrate to death vials in population cages were locomotor activity and genotype. This report is an examination of not only these two attributes, but also the effect of age on activity and death rate. The possible relationship of these factors to death vial emigration is also examined.

A rough estimate of locomotor activity was established by determining the distance, in centimeters, naive flies of known age, genotype and sex moved in an activity chamber during a five minute measurement period. The activity chamber was constructed of a 100 cm long segment of 1.0 cm i.d. glass tubing, capped at both ends with plastic foam plugs, and mounted on a 0.625 x 7.5 x 150 cm lucite base. A metric tape was affixed to the lucite base, alongside the chamber, to facilitate measurement of the distance the flies moved. The chamber was illuminated along its entire length by two fluorescent bulbs, so that any phototropic effect would be minimized. For each measurement run, about twenty flies were inserted into the chamber by connecting an adaptor segment, containing the flies, to the activity chamber. The adaptor segment was a glass tube, one end of which was the same diameter as the activity chamber and which allowed quick connection to the chamber, the other end of the adaptor was expanded to the diameter of a standard shell vial (2.5 cm). This allowed flies to move from a shell vial into the adaptor, and then into the chamber, with little manipulation.

The effect of age and genotype on the overall death rate (% dead + moribund) was measured as in Thompson (1985). Flies of known age and genotype were inserted into population cages at a cage density of 2000 flies/cage, with equal numbers of females and males. The number of dead and moribund flies in the "death vial" was enumerated each day for a period of seven days, with a new death vial inserted at each count.

Figure 1 shows the relationship between adult age and activity in Oregon-R and yellow flies; each point is based on no fewer than 200 flies. As can be seen, males have a higher activity, in both Oregon-R and yellow, until about eleven days at which time, and thereafter, there is no significant difference in activity between the sexes. Initially yellow flies have a higher activity rate than do Oregon-R flies, but with age the activity rate dropped at a younger chronological age and to a lower level with yellow than it did with Oregon-R.

Table 1, which is a comparison of locomotor activity and death rate in Oregon-R and yellow, shows that the influence of activity on death rate in population cages appears to be inversely related, i.e., flies with higher locomotor activity generally have a lower death rate and flies with lower locomotor activity have a higher death rate. The following specific relationships attest to this observation. (1) Young, three-day, yellow flies have a lower death rate than Oregon-R at the same age, and the activity of young yellow flies is higher. (2) Male flies usually have a lower death rate than females and their activity levels are generally higher. The exception to this is in young yellow flies, where the apparent death rate in males is somewhat higher than in females. (3) With increasing age, Oregon-R exhibits a lower death rate than yellow, and the locomotor activity is also higher.

Similar observations on average locomotor ability and death rate were seen in preliminary experiments with the mutants ebony and white, at seven days of age. In ebony, a mutant strain with poor optomotor behavior, the locomotor activity was the lowest observed (7.8) with a very high death rate (55.3%). The locomotor activity of white (11.1) was near that of yellow, but the white strain had the lowest death rate observed (2.8%). Comparison between strains is difficult as flies of the same chronological age may be of quite different biological ages. It may be that what is being examined, with both locomotor activity and death rate, is a reflection of different rates of aging.

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Tobari, Y.N. and M. Matsuda. Tokyo Metropolitan University, Tokyo, Japan. Recombination in SM1/In(2L)t heterozygotes of D. melanogaster.

extracted from Katsunuma population by using the balancers. Of the 615, 90 chromosomes carried In(2L)t (23/123 in 1969, 36/269 in 1970, and 31/233 in 1979). Most In(2L)t's, 88 of 90, were linked with AdhS; only 2 In(2L)t-AdhF's were found, 1 in 1969 and 1 in 1979.

In view of the rare appearance of ln(2L)t-AdhF and the proximity of the breakpoints of ln(2L)t and ln(2L)Cy, we suspected that double crossing-over within 2L in SM1-AdhF/ln(2L)t-AdhS heterozygous\

Table 1. Recombination frequencies in SM1, Cy Bl/ln(2L)t heterozygous females of D. melanogaster.

<table>
<thead>
<tr>
<th>Chromosome</th>
<th>Nonrecombinant</th>
<th>Recombinant</th>
<th>Cy Bl</th>
<th>Cy</th>
<th>B1</th>
<th>Progeny freq. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>OK 1</td>
<td>324</td>
<td>322</td>
<td>7</td>
<td>6</td>
<td>649</td>
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<td>678</td>
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<td>11</td>
<td>14</td>
<td>515</td>
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<td>13</td>
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<td>OK 29</td>
<td>463</td>
<td>528</td>
<td>14</td>
<td>11</td>
<td>1016</td>
<td>2.46</td>
</tr>
<tr>
<td>OK 30</td>
<td>355</td>
<td>375</td>
<td>11</td>
<td>5</td>
<td>746</td>
<td>2.14</td>
</tr>
<tr>
<td>OK 31</td>
<td>440</td>
<td>440</td>
<td>13</td>
<td>20</td>
<td>913</td>
<td>3.61</td>
</tr>
<tr>
<td>OK 32</td>
<td>774</td>
<td>770</td>
<td>17</td>
<td>28</td>
<td>1599</td>
<td>2.83</td>
</tr>
<tr>
<td>OK 33</td>
<td>383</td>
<td>427</td>
<td>47</td>
<td>37</td>
<td>984</td>
<td>9.40</td>
</tr>
<tr>
<td>OK 34</td>
<td>126</td>
<td>126</td>
<td>3</td>
<td>3</td>
<td>258</td>
<td>2.33</td>
</tr>
<tr>
<td>OK 35</td>
<td>194</td>
<td>199</td>
<td>2</td>
<td>6</td>
<td>401</td>
<td>2.00</td>
</tr>
<tr>
<td>OK 36</td>
<td>229</td>
<td>284</td>
<td>23</td>
<td>31</td>
<td>567</td>
<td>9.52</td>
</tr>
<tr>
<td>OK 37</td>
<td>341</td>
<td>374</td>
<td>9</td>
<td>8</td>
<td>732</td>
<td>2.32</td>
</tr>
<tr>
<td>OK 38</td>
<td>206</td>
<td>265</td>
<td>6</td>
<td>4</td>
<td>481</td>
<td>2.08</td>
</tr>
<tr>
<td>OK 39</td>
<td>260</td>
<td>262</td>
<td>8</td>
<td>11</td>
<td>941</td>
<td>3.61</td>
</tr>
<tr>
<td>OK 40</td>
<td>338</td>
<td>337</td>
<td>8</td>
<td>11</td>
<td>942</td>
<td>2.74</td>
</tr>
<tr>
<td>OK 41</td>
<td>167</td>
<td>210</td>
<td>3</td>
<td>8</td>
<td>388</td>
<td>2.84</td>
</tr>
<tr>
<td>OK 42</td>
<td>280</td>
<td>338</td>
<td>11</td>
<td>18</td>
<td>647</td>
<td>4.48</td>
</tr>
<tr>
<td>OK 43</td>
<td>301</td>
<td>282</td>
<td>5</td>
<td>6</td>
<td>594</td>
<td>1.85</td>
</tr>
<tr>
<td>OK 44</td>
<td>131</td>
<td>126</td>
<td>7</td>
<td>4</td>
<td>268</td>
<td>4.10</td>
</tr>
<tr>
<td>OK 45</td>
<td>110</td>
<td>117</td>
<td>5</td>
<td>7</td>
<td>240</td>
<td>5.42</td>
</tr>
<tr>
<td>OK 46</td>
<td>163</td>
<td>175</td>
<td>4</td>
<td>2</td>
<td>344</td>
<td>1.74</td>
</tr>
</tbody>
</table>

Table 2. Recombination frequencies in SM1, Cy L/ln(2L)t heterozygous females of D. melanogaster.

<table>
<thead>
<tr>
<th>Chromosome</th>
<th>Nonrecombinant</th>
<th>Recombinant</th>
<th>Cy L</th>
<th>Progeny freq. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>KT 2</td>
<td>663</td>
<td>670</td>
<td>5</td>
<td>1 1339</td>
</tr>
<tr>
<td>KT 3</td>
<td>175</td>
<td>249</td>
<td>2</td>
<td>1  426</td>
</tr>
<tr>
<td>KT 4</td>
<td>893</td>
<td>955</td>
<td>3</td>
<td>0 1851</td>
</tr>
<tr>
<td>KT 5</td>
<td>319</td>
<td>388</td>
<td>1</td>
<td>2  710</td>
</tr>
<tr>
<td>KT 6</td>
<td>181</td>
<td>189</td>
<td>0</td>
<td>0  370</td>
</tr>
<tr>
<td>KT 7</td>
<td>213</td>
<td>219</td>
<td>2</td>
<td>1  433</td>
</tr>
<tr>
<td>KT 9</td>
<td>145</td>
<td>132</td>
<td>0</td>
<td>2  279</td>
</tr>
<tr>
<td>OK 4</td>
<td>852</td>
<td>885</td>
<td>17</td>
<td>11 1765</td>
</tr>
</tbody>
</table>
Table 3. Linkage of recombinant chromosomes.

<table>
<thead>
<tr>
<th>Population</th>
<th>Cy-Adh^F</th>
<th>Cy-Adh^S</th>
<th>Adh^F-B1</th>
<th>Adh^S-B1</th>
<th>No. of recomb. tested</th>
</tr>
</thead>
<tbody>
<tr>
<td>OK</td>
<td>73</td>
<td>29</td>
<td>20</td>
<td>45</td>
<td>167</td>
</tr>
<tr>
<td>OG</td>
<td>1</td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>KT</td>
<td>0</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>8</td>
</tr>
<tr>
<td>Total</td>
<td>74</td>
<td>37</td>
<td>21</td>
<td>46</td>
<td>178</td>
</tr>
</tbody>
</table>

Table 4. Gene arrangement and linked alleles of recombinant chromosomes.

<table>
<thead>
<tr>
<th>Recombinant chromosome</th>
<th>No. of chromosomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>In(2L)t, Cy Adh^F</td>
<td>37</td>
</tr>
<tr>
<td>In(2L)t, Cy Adh^S</td>
<td>21</td>
</tr>
<tr>
<td>In(2L)t, Adh^F B1</td>
<td>0</td>
</tr>
<tr>
<td>In(2L)t, Adh^S B1</td>
<td>0</td>
</tr>
<tr>
<td>SM1, Cy Adh^F</td>
<td>0</td>
</tr>
<tr>
<td>SM1, Cy Adh^S</td>
<td>0</td>
</tr>
<tr>
<td>SM1, Adh^F B1</td>
<td>8</td>
</tr>
<tr>
<td>SM1, Adh^S B1</td>
<td>26</td>
</tr>
</tbody>
</table>

Figure 1. Schematic configuration of chromosome pairing in SM1/ln(2L)t heterozygotes.

B1/ln(2L)t F1 females or SM1, Cy L/ln(2L)t F1 females were individually mated to Or-R males. The F2 progeny were scored for recombination of markers. Of the F2 progeny recombinant alleles, Cy, B1, or L, were individually mated to Or-R females. After several days these F2 males were analyzed for Adh alleles. Several F3 larvae from each mating were individually examined for the inversions and Adh alleles simultaneously.

Sites of markers are approximately placed on chromosome loops which are to be formed when SM1 and ln(2L)t pair (Fig. 1). Frequencies of viable recombinants in SM1/ln(2L)t females are listed in Tables 1 and 2. Substantial recombinants were recovered in every female, in various proportions. This demonstrates that the SM1 did not balance chromosome 2 carrying ln(2L)t. It is clear that there is a considerable chance that the Cy and/or AdhF allele from the SM1 may have been introduced into ln(2L)t. Inspection of Tables 3 and 4 indicates that SM1/ln(2L)t x SM1/ln(2L)t cf crosses produced F3 progeny composed of visibly indistinguishable blend of a variety of recombinants, including such as SM1, +Cy and ln(2L)t, Cy, and nonrecombinants.

There, therefore, the existence of rare ln(2L)t-Adh^F in natural populations of *D. melanogaster* seems very unlikely.


Trejan, K.S. and K.S. Gill. Punjab Agricultural University, Ludhiana, India. Effect of inbreeding on wing morphology in *Drosophila malerkotliana*. III effects of inbreeding on wings were studied while preparing isogenic lines for two acid phosphatase alleles (Acph^1,05 and Acph^0,95) in *Drosophila malerkotliana*. Homozygous stocks for the two alleles were established from wild-caught flies. From the running cultures of these two stocks, 20 single pair matings (one member homozygous for one allele and the other for the other allele) were set-up. From the progeny of a single mating pair, further 20 single pair matings (F1 x F1) were set up. From the progeny of one of the 20 F1 x F1 matings, again 20 single pair matings (F2 x F2) were set up. After pupation the mating pairs were removed and subjected to electrophoresis, one of the vials, in which both the alleles were represented, was chosen for selecting the parents of next generation. This plan of mating
Table 1. Effect of inbreeding on wing morphology in Drosophila melarkotliana.

<table>
<thead>
<tr>
<th>No. of flies studied</th>
<th>Types of wing/abnormalities</th>
<th>% Total abnormal flies</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>one wing held out</td>
<td>both wings</td>
</tr>
<tr>
<td>I₄</td>
<td>1673</td>
<td>55</td>
</tr>
<tr>
<td>I₅</td>
<td>1444</td>
<td>132</td>
</tr>
<tr>
<td>I₆</td>
<td>1155</td>
<td>177</td>
</tr>
<tr>
<td>I₇</td>
<td>884</td>
<td>242</td>
</tr>
<tr>
<td>I₈</td>
<td>584</td>
<td>--</td>
</tr>
<tr>
<td>I₉</td>
<td>No progeny</td>
<td></td>
</tr>
</tbody>
</table>

In each generation and for any specific generation in the three experiments, pooled data is being presented in Table 1.

In I₄ generation approximately 3% flies had one or the other wing permanently held out horizontally. The frequency of such flies increased in subsequent generations. Moreover, in I₅ generation approximately 16% of the abnormal flies had both the wings permanently held out and about 2% of the abnormal flies had shrivelled wing (resembling the vestigial wing mutant of Drosophila melanogaster). In I₇ generation abnormal flies with both the wings held out did not appear. However, the frequency of flies with shrivelled wings increased to approximately 21% of the abnormal flies. All the flies of I₉ generation were abnormal; more than 99% of the flies had shrivelled wings and only 3 flies had both the wings held out permanently. These flies were very weak, could not mate and died within a week.

A perusal of data in the table also indicates that the number of pooled flies was 1673 in I₄ and 584 in I₈ generation, and no progeny was obtained in I₉ generation. It then appears that inbreeding not only induces wing abnormalities but also drastically reduces the fertility.

Turelli, M., C. Burkhard, V. Fong, J. Moore, S. Van Horn and T. Prout. University of California, Davis, USNA. Does dusting distort Drosophila dispersal?

Since Crumpacker & Williams's (1973) original application of UV-fluorescent dusts to Drosophila pseudoobscura, these dusts have become a standard tool in the study of Drosophila field behavior. One of the most astounding results obtained is that Drosophila can disperse 10 km within 24 hr (Jones et al. 1981; Coyne et al. 1982). A central assumption in studies using dusts is that dusting does not significantly affect the behavior under investigation. Crumpacker (1974) performed control experiments which indicated that dusting does not decrease viability under laboratory conditions, but there are no comparable studies concerning the effects of dusts on dispersal. To test the hypothesis that dusts do not influence dispersal, we simultaneously released dusted and undusted Drosophila simulans homozygous for the recessive plum.

We reared approximately 100,000 flies in half-pint bottles. On August 31, 1982, we divided them at random into two groups of approximately equal size. The first group was heavily coated with red micrized UV-fluorescent dust (Radiant Color, Richmond, CA) by shaking the flies pooled from several culture bottles in a half-pint bottle containing several milligrams of dust. After two transfers to clean bottles to remove excess dust, the dusted flies were placed in a plexiglass cage, approximately 42 cm on a side. The second (control) group of approximately 50,000 was transferred from the culture bottles into bottles without dust then placed in a second plexiglass cage. No anesthesia was used and the flies ranged in age from 0-5 days.

At dusk, approximately three hours after we began collecting them from the culture bottles, we released the flies at an intersection of two rarely used roads crossing experimental fields on the Davis campus. The north-south road is asphalt and lined on both sides with mature olive trees. The east-west road is dirt with no vegetation along its borders. At the time of release, the red dust was only faintly visible on the marked flies. The next afternoon (day 1) we set out 41 five-liter buckets baited with orange/banana mash and Fleischmann's yeast. The buckets were placed along the roads at 30 m intervals, with ten in each direction and one at the intersection. During the hour and a half before nightfall, we collected the flies three times from each of the buckets. The baits were left out overnight. Because several flies had been captured in the outermost traps, we extended the north-south trapline during the afternoon of September 2 (day 2) by ten baits to the south and five to the north. During the hour and a half before nightfall, we again collected the flies three times from each bucket.
Table 1. \( \bar{d} \), SD, N, and P for each direction and day.

<table>
<thead>
<tr>
<th></th>
<th>North</th>
<th>South</th>
<th>East</th>
<th>West</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dusted</td>
<td>( \bar{d}(N) )</td>
<td>124.1(233)</td>
<td>96.0(740)</td>
<td>132.6(74)</td>
<td>66.5(32)</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>79.8</td>
<td>68.6</td>
<td>83.4</td>
<td>49.8</td>
</tr>
<tr>
<td>undusted</td>
<td>( \bar{d}(N) )</td>
<td>123.1(185)</td>
<td>106.8(564)</td>
<td>140.2(61)</td>
<td>96.1(44)</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>81.6</td>
<td>73.5</td>
<td>96.2</td>
<td>79.4</td>
</tr>
<tr>
<td>P</td>
<td>&gt;0.1</td>
<td>0.006</td>
<td>&gt;0.1</td>
<td>&gt;0.05</td>
<td></td>
</tr>
<tr>
<td>Day 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dusted</td>
<td>( \bar{d}(N) )</td>
<td>147.4(141)</td>
<td>82.6(365)</td>
<td>163.6(31)</td>
<td>105.0(8)</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>95.5</td>
<td>82.3</td>
<td>60.4</td>
<td>69.9</td>
</tr>
<tr>
<td>undusted</td>
<td>( \bar{d}(N) )</td>
<td>149.9(304)</td>
<td>114.9(415)</td>
<td>189.4(32)</td>
<td>131.7(46)</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>89.9</td>
<td>94.7</td>
<td>67.7</td>
<td>72.0</td>
</tr>
<tr>
<td>P</td>
<td>&gt;0.1</td>
<td>&lt;0.001</td>
<td>&gt;0.1</td>
<td>&gt;0.1</td>
<td></td>
</tr>
</tbody>
</table>

Our results are summarized in Table 1. For each of the four directions on each day, we give the mean (\( \bar{d} \)) and standard deviation (SD) of the capture distance (in meters) from the release point (flies captured at the central bucket were ignored in these calculations), the number of released flies captured (N), and the significance level (P) of an approximate t-test (allowing for unequal variances, see Sokal & Rohlf 1981) for the hypothesis of equal mean dispersal distances for dusted and undusted flies. We have not statistically analyzed the data pooled across directions because of the heterogeneity of the environment.

Our data indicate that dusting tends to decrease dispersal distances slightly. From the two significantly different means, the estimated decrease is 10-20\%. Thus, our major qualitative result is that estimates of dispersal distances based on dusting are fairly reliable but may be slightly conservative.

Other differences appear in our data. On day 1, we captured 1117 dusted and 893 undusted flies (including 38 dusted and 19 undusted from the central bucket). On day 2, we captured 554 dusted flies and 800 undusted flies (including 9 dusted and 3 undusted from the central bucket). Hence the dusted flies were significantly more likely to have been captured on day 1 and significantly less so on day 2 (\( \chi^2 = 68.2, P < 0.001 \)). Based on Nigro and Shorrocks's (1982) results concerning the persistence of dust on flies in the field, it is very unlikely that this difference is attributable to misclassifying dusted flies as undusted on day 2. The difference may have resulted, at least in part, from more of the more mobile undusted flies dispersing beyond the trapline on day 1, combined with lower viability of the dusted flies under field conditions. Alternatively, the dusted flies may have been more likely than the undusted flies to be captured on day 1, because they may have been more "stressed" (perhaps dessicated) and hence responded to the baits differently as observed by Hoffman & Turelli (1985). Chi-square tests also show that the proportions of dusted and undusted flies captured over the four directions were homogeneous on day 1 (\( \chi^2 = 5.6, P > 0.1 \)) but significantly heterogeneous on day 2 (\( \chi^2 = 43.9, P < 0.001 \)). Thus, even though dusts only slightly affect dispersal distances, they may alter field behavior. The significance of these effects for other types of experiments remains to be determined.

Acknowledgements: Supported by NIH Grant 22221 and Larry Harshman.


I'll never forget the day I died!
A human friend to make I tried;
I waved and buzzed and did a dance
In hope that I might earn a glance;
And when his hand curled 'round my vial,
I thought perchance he'd seen my smile,
But then my world began to shake --
Inversion followed in its wake --
I coughed and struggled and fought for air
As ether fostered dark despair.
A bright light shone as I came 'round,
A poker pinned me to the ground.
As I slid off the porcelain plate
I realized what had sealed my fate.
The oil it clung and pulled me under
As I rue my fatal blunder;
My dance was not my human's gripe --
'Twas pure and simple my phenotype!
Ushakumari, A. and H.A. Ranganath. University of Mysore, India. Egg to adult viability and rate of development of five strains of Drosophila in four different media. Viability and egg-to-adult developmental time are two important components of fitness in Drosophila and they can be related to phenomena such as larval facilitation, as well as concepts of microniches and microenvironments (Castro & Mensua 1985). Using these two parameters, experiments have been undertaken to study some aspects of nutritional requirements of Drosophila under laboratory conditions. The present note deals with four different types of wheat cream agar media. They are: (1) media without yeast and sugar ($S^*/Y^*$); (2) media with yeast and without sugar ($S^*/Y^*$); (3) media with sugar and without yeast ($S^+/Y^*$); and (4) media with yeast and sugar ($S^+/Y^*$).

The Drosophila strains used in this experiment are as follows: D.sulfurigaster sulfurigaster, P-11 Port Moresby, Papua, New Guinea; D.s.albostrigata, S-11 Sandakan, Sabah, Malaysia; D.s.bilimbata GUM-8 Guam; D.s.neonasuta 209.2 Mysore, India; D.pulaua S-18 Sandakan, Sabah, Malaysia.

These five strains belong to the orbital sheen complex of the nasuta subgroup of the immigrans species group of Drosophila. These members are morphologically indistinguishable from one another.

Eggs of approximately four hours of age of these five strains were collected following the modified procedure of Delcour (Ranganath & Krishnamurthy 1974). Ten vials were made for each strain in each medium. Each vial contains 50 eggs of any one strain in any one medium. After the onset of emergence the number of flies eclosed on each day was recorded and from this the mean developmental rate in days were calculated in four different media under study. The total number of flies emerged in 10 vials (10 vials used for the analysis of rate of development) in each medium were recorded and from this the egg to adult viability was estimated for the above said strains in four different media.

The results pertaining to the egg to adult rate of development are compiled in Table 2. The notable findings of this study include:

1. In the four different types of media under study the rate of development of D.sulfurigaster ranges between 12.76 to 13.43 days. On the other hand, the spectrum of variation for D.s.bilimbata, is between 13.46 to 21.00 days.

2. In the present experimental set up, the fastest rate of adult eclosion was noticed for the eggs of D.s.albostrigata in the media with sugar and with yeast, and the slowest rate of development was recorded in the media without sugar and without yeast for the eggs of D.sulfurigaster.

Thus, these experiments reveal (a) the importance of sugar and yeast on the manifestation of two parameters, namely the rate of development and viability, and (b) the differential response of closely related strains of Drosophila to different types of media.

### Table 1. Egg to adult viability of the 5 strains of Drosophila in four different media (500 eggs were sown in each group) with summarised chi-square test.

<table>
<thead>
<tr>
<th>Strain</th>
<th>Media: $S^<em>/Y^</em>$</th>
<th>$S^<em>/Y^</em>$</th>
<th>$S^+/Y^*$</th>
<th>$S^+/Y^*$</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>D.sulfurigaster</td>
<td>P-11</td>
<td>62</td>
<td>76</td>
<td>107</td>
<td>174</td>
</tr>
<tr>
<td>D.albostrigata</td>
<td>S-11</td>
<td>2</td>
<td>10</td>
<td>84</td>
<td>185</td>
</tr>
<tr>
<td>D.bilimbata</td>
<td>GUM-8</td>
<td>15</td>
<td>28</td>
<td>52</td>
<td>83</td>
</tr>
<tr>
<td>D.neonasuta</td>
<td>(209.2)</td>
<td>167</td>
<td>284</td>
<td>290</td>
<td>303</td>
</tr>
<tr>
<td>D.pulaua</td>
<td>(S-18)</td>
<td>0</td>
<td>7</td>
<td>12</td>
<td>131</td>
</tr>
</tbody>
</table>

### Table 2. Mean development time in days (mean ± SE) for the five strains of Drosophila in four different media.

<table>
<thead>
<tr>
<th>Strain</th>
<th>Media: $S^<em>/Y^</em>$</th>
<th>$S^<em>/Y^</em>$</th>
<th>$S^+/Y^*$</th>
<th>$S^+/Y^*$</th>
</tr>
</thead>
<tbody>
<tr>
<td>D.sulfurigaster</td>
<td>P-11</td>
<td>13.00±0.10</td>
<td>12.76±0.14</td>
<td>13.36±0.15</td>
</tr>
<tr>
<td>D.albostrigata</td>
<td>S-11</td>
<td>17.00±0.00</td>
<td>17.00±0.00</td>
<td>12.48±0.14</td>
</tr>
<tr>
<td>D.bilimbata</td>
<td>GUM-8</td>
<td>21.00±0.00</td>
<td>16.54±0.19</td>
<td>14.50±0.14</td>
</tr>
<tr>
<td>D.neonasuta</td>
<td>(209.2)</td>
<td>16.59±0.13</td>
<td>13.52±0.11</td>
<td>15.42±0.11</td>
</tr>
<tr>
<td>D.pulaua</td>
<td>(S-18)</td>
<td>15.43±0.20</td>
<td>13.08±0.19</td>
<td>12.98±0.17</td>
</tr>
</tbody>
</table>
**Acknowledgements:** Authors are grateful to Prof. N.B. Krishnamurthy, Head of the Dept. of Zoology, for help and encouragement; to the Indian National Science Academy, New Delhi, for awarding research grants to HAR; and to Prof. O. Kitagawa for sending flies.


Fecundity is defined as the number of eggs laid by an individual. Experiments were conducted to assess the egg laying capacity of the ten strains of five species of the orbital sheen complex of the *nasuta* subgroup of the *immigrans* species group of Drosophila. Males and females from each strain were collected within four hours after eclosion, and after aging them for five days, pair matings were made. These pairs were transferred to fresh vials without etherisation once in every 24 hr. Then the number of eggs laid in each vial was recorded. Likewise egg counts for 60 pairs of flies were made for each strain for over a period of ten days. Chi-square test computed for these ten strains has revealed statistically significant differences in their fecundity.

The results summarized in Table 1 reveal the following: (1) the number of eggs per individual per day varies between 3 to 12; (2) except *D.pulaua*, the other four members show significant interstrain differences with regard to their egg laying capacity; (3) of the ten strains, the polymorphic strain of *D.s.neonasuta* has shown the highest fecundity, while HNL-III strain of *D.s.bilimbata* has the least.

The sequence as to the egg laying potentialities of the strains under study is *D.s.neonasuta* (polymorphic) > *D.s.albostrigata* (W-3) > *D.s.albostrigata* (S-li) > *D.s.bilimbata* (GUM-8) > *D.s.pulaua* (V-6) > *D.s.pulaua* (S-18) > *D.s.sulfurigaster* (3019.8) > *D.s.sulfurigaster* (P-li) > *D.s.bilimbata* (HNL-III).

Even though all the ten strains are morphologically identical, phylogenetically and taxonomically closely related, they show significant differences in their egg laying capacity. The variability recorded in Table 1 reflects the genetic variability present in the ten strains under study for the character "fecundity".

**Acknowledgements:** Authors are grateful to Prof. N.B. Krishnamurthy, Head of the Dept. of Zoology, for help and encouragement; to the Indian National Science Academy, New Delhi, for awarding research grants to HAR; and to Prof. O. Kitagawa for sending flies.

### Table 1. Fecundity of ten strains of the Orbital sheen complex of the *nasuta* subgroup of Drosophila.

<table>
<thead>
<tr>
<th>Strains</th>
<th>No. of pairs</th>
<th>Total no. of eggs</th>
<th>Eggs/individual</th>
<th>Eggs/individual/day</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>D.s.sulfurigaster</em> (3019.8)</td>
<td>60</td>
<td>4337</td>
<td>72.26</td>
<td>7.23</td>
</tr>
<tr>
<td><em>D.s.sulfurigaster</em> (P-il)</td>
<td>60</td>
<td>2550</td>
<td>42.50</td>
<td>4.25</td>
</tr>
<tr>
<td><em>D.s.albostrigata</em> (W-3)</td>
<td>60</td>
<td>5480</td>
<td>91.33</td>
<td>9.13</td>
</tr>
<tr>
<td><em>D.s.albostrigata</em> (S-li)</td>
<td>60</td>
<td>2550</td>
<td>42.50</td>
<td>4.25</td>
</tr>
<tr>
<td><em>D.s.bilimbata</em> (GUM-8)</td>
<td>60</td>
<td>2802</td>
<td>46.70</td>
<td>4.67</td>
</tr>
<tr>
<td><em>D.s.bilimbata</em> (HNL-III)</td>
<td>60</td>
<td>2350</td>
<td>43.00</td>
<td>4.30</td>
</tr>
<tr>
<td><em>D.s.neonasuta</em> (polymorphic)</td>
<td>60</td>
<td>7401</td>
<td>123.35</td>
<td>12.34</td>
</tr>
<tr>
<td><em>D.s.neonasuta</em> (monomorphic)</td>
<td>60</td>
<td>5267</td>
<td>87.78</td>
<td>8.78</td>
</tr>
<tr>
<td><em>D.pulaua</em> (V-6)</td>
<td>60</td>
<td>4693</td>
<td>78.22</td>
<td>7.82</td>
</tr>
<tr>
<td><em>D.pulaua</em> (S-18)</td>
<td>60</td>
<td>4716</td>
<td>78.60</td>
<td>7.86</td>
</tr>
</tbody>
</table>

∑ X² = 5754.95; df = 9; P < 0.0001.

**Van Delden, W. and A. Kamping.** University of Groningen, Haren, Netherlands. The relationship between ethanol tolerance and ethanol utilization in *Drosophila melanogaster*.

Many reports have demonstrated the relationship between the polymorphism at the alcohol dehydrogenase (Adh) locus in *D.melanogaster* and tolerance to alcohols (reviewed in Van Delden 1982). When the Adh genotypes, which differ in vitro ADH activity, are compared for survival on toxic concentrations of ethanol or other alcohols, it is found that survival and ADH activity are positively correlated. It has further been shown that, in the absence of other food components, ethanol may serve as a food component when it is present at low concentrations (references in Van Delden 1982).

In the present study we tested strains of the Groningen population which had been selected for increased tolerance to ethanol, for their ability to utilize low concentrations of ethanol as the only food source. For this purpose we used one strain which was homozygous for the Adh<sup>F</sup> allele (FFE) and one strain homozygous for the Adh<sup>S</sup> allele (SSE). Both strains had been kept continuously on regular food
Mortality—.

Figure 1. Adult mortalities after 100 h at various concentrations of ethanol supplemented to agar. SSE and FFE: strains selected for ethanol tolerance; SSN and FFN: control strains.

supplemented with ethanol for 112 generations (details in Kerver & Van Delden 1985). During that period the ethanol concentration was raised from an initial 12% by volume to 18% in three steps. These selected strains (FFE and SSE) were compared with their controls (FFN and SSN, respectively) which had been kept on regular food. The aim of our study was to establish whether the considerably increased tolerance to toxic concentrations of ethanol in the E lines is accompanied by an improved ability to utilize ethanol at low concentrations as the only food component.

Flies from each of the four strains were transferred to regular food and their offspring were used for the experiments. For the survival tests adults, 6-8 days old, were put into vials with 9 ml agar supplemented with 0, 1, 2, 4, 6, 8, 10 or 12 percent ethanol by volume. The number of flies (either males or females) per vial was 20; the number of replicates per sex and per strain was 5. The number of dead flies was recorded at regular intervals. The results are shown in Figure 1, where mortality after 100 h is given. Several conclusions can be drawn from the results.

(1) The genotypes differ considerably in survival percentages, even when food is absent (0% ethanol); FF homozygotes perform better under starvation conditions than SS homozygotes. (2) Mortalities decrease with increasing ethanol concentrations until almost 8%, due to ethanol utilization. Mortalities at 12% ethanol are higher than at lower concentrations because at that concentration the toxic effects overcome the beneficial effects. (3) The increase in life span at the lower ethanol concentrations is higher in FF homozygotes than in SS homozygotes. This result confirms the findings of Daly & Clarke (1981), who measured survival at 2% ethanol. (4) The E strains survive better at the toxic ethanol concentrations than the N strains, which is in agreement with the findings of Kerver & Van Delden (1985) that tolerance is increased in the selected strains. (5) The E strains utilize ethanol at low concentrations better than their control strains. This is in agreement with the results of Dorado & Barbancho (1984) that strains selected for ethanol tolerance had a better ability to use ethanol (11%) as food than controls. Also Van Herrewege & David (1980) found this relationship, but not for all their strains. They therefore proposed different genetic mechanisms for tolerance and for utilization. Their selection procedure, however, was different from ours as selection for ethanol tolerance was only exercised during a limited number of days of the adult life phase.

The results show that selection for ethanol tolerance at high ethanol concentrations also improves the ability of the selected strains to utilize low concentrations of ethanol when present as the only food source. This relationship between ethanol tolerance and utilization has also been found, though in a different way, by Van Herrewege & David (1984) who selected for increased ethanol utilization and found an increase in ethanol tolerance at high ethanol concentrations. This result was, however, only found in one of the two strains tested.

The mechanism behind the observed relationship between tolerance and utilization could at first sight be sought in the difference in ADH activity of the strains in view of the different behavior of the SSN and FFN strains. Adults of the E strains used in our experiments, however, possess no higher ADH activities than their respective unselected controls (Kerver & Van Delden 1985); thus total ADH activity is apparently not the key factor involved.

**Villarroel, H. and P. Zamorano.** Academia Superior de Ciencias Pedagogicas, Valparaiso, Chile. Distribution altitudinal of the Drosophila genus which inhabit the National Park "La Campana", Valparaiso, Chile.

Genetic investigations carried out concerning the Drosophila genus are extensive and varied; however, very little is known about its ecology (Ford 1974), particularly in our zone. The studies carried out on these diptera at the National Park "La Campana" have shown the existence of nine Drosophila species (Villarroel & Zamorano 1984).

The purpose of the above mentioned work is to determine the composition and distribution of species of the Drosophila genus in one altitudinal transect at cerro "La Campana", a place forming part of the National Park under the same name, its maximum altitude being 1880 m above sea level (Serey, Ortiz, Meza and Solervicens 1976) and located at Olmue, at approximately 70 Km from Valparaiso.

The zone has a Mediterranean climate, where most of the more significant biotic communities of Central Chile are represented, such as: Nothofagus forest, hygrophilous forest, sclerophyll forest, matorral, bamboo thicket, succulent scrub, high altitude communities and palm forests (Oberdorffer 1960; Rundel & Weisser 1975).

Sample collection was made from October 1982 to March 1983. Three sampling stations were chosen at 560, 800 and 1100 m above sea level. Capture was performed by means of the usual trapping method (fermented banana bait).

A total of 1983 flies was collected, corresponding to nine species of the 33 described for Chile by Brncic (1957a; 1962a) (see Table 1).

The distribution of each species at different altitudes indicates very particular behaviours, so we can distinguish three well defined groups.

**I:** *D.amplipennis* and *D.araucana* reach a maximum abundance at 1100 m above sea level. They are species belonging to the Southern zone of the country; therefore, the search of their native habitat will cause them to distribute more abundantly in natural places, away from man and where weather conditions are more or less resembling those of the south of Chile.

**II:** *D.immigrans* and *D.melanogaster* + *D.simulans* are more abundant at 560 m above sea level; they decrease while altitude increases. They are clearly a cosmopolitan species and therefore closely associated with man, hence they are preferably located at an altitude which is closer to human environment.

**III:** *D.repleta, D.pavani* and *D.subobscura* are more frequent at 800 m above sea level. At this altitude environmental conditions are a bit more xeric than in the remaining sampling stations, and they are more exposed to weather conditions such as wind and solar radiation; therefore, these species here would presumably find conditions resembling their natural environment. Moreover, a certain competition concerning food resource was apparently glimpsed between *D.subobscura* and other species.

Another interesting feature of the collection is a marked decrease in the number of individuals at 800 m above sea level. Afterwards it slightly increased at 1100 m above sea level. The intermediate altitude is characterized by a not very dense vegetation and by a floor of a scarce vegetable cover, while at maximum altitude a transition zone is placed where there exists a floristic convergence (Villasenor 1980), thus increasing the diversity of the area which would consequently cause an increase in the abundance of Drosophila at this altitude.

Under these considerations the 1100 m above sea level would constitute a favorable place for the establishment of the species.

Finally, we can indicate that environmental conditions play an interesting role in altitudinal distributions of the Drosophila species, not withstanding the importance of genetic factors in this particular situation.

Arnason, E. University of Iceland, Reykjavik, Iceland. Yet another population cage. Many different population cages have been used by drosophilists. The following is an extension of a design by Lewontin (1965); it is cheap, convenient in use and durable. The materials needed are: (1) a perishable shipper-foam unit only (available from Polyfoam Packers Corporation, 6415 N. Chicago Ave., Chicago, IL 60445 @ $4.35). Inside dimensions are $8\frac{1}{4}$" wide x $11\frac{3}{4}$" long x $11\frac{3}{4}$" deep with $1$" thick walls made of high density polyfoam. (2) two 1000 ml polypropylene beakers, Tri-Pour design (@ $0.60) available from any biological supply house. (3) a piece of clear plexiglass $8\frac{1}{8}$" x $11\frac{3}{4}$" x $1/8$" to fit on top. (4) strips of plexiglass $1\frac{3}{8}$" x $3/16$" that can be cut and broken into desired lengths; plexiglass is available locally in most places. (5) a piece of 36 or 40 gauge nylon mesh $6" \times 5"$ cut with a hot soldering iron to seal the edges. (6) A piece of broadcloth $15" \times 20"$. (7) a tube of silicon rubber. (8) nine $1/16"$ metal bolts $1\frac{3}{4}$" long with 18 nuts and 18 washers to fit. (9) a role of duct tape or masking tape. The total cost of materials is about $6-7$; labor is cheap.

Make the cage as follows: Cut the height of the box down to $8"$ using a sabre saw mounted with a knife edge blade. Cut a circular hole, $4\frac{3}{4}"$ diameter, in the one end of the box, and a square hole, $3\frac{1}{2} \times 2"$, in the other end. Cut the bottoms off the Tri-Pour beakers leaving a $1\frac{1}{2}"$ rim with the triangular top of one and a $3/4"$ rim on the other. Saw a long sleeve from the broadcloth, tight enough to fit snugly on the beaker with the larger rim; tape the sleeve to the rim. Drill or punch holes large enough for the bolts in the corners of the beakers. Fit the beaker top with the sleeve through the circular hole, seal on the inside with silicon rubber and fit the other beakertop opposite on the outside. Push a sharp needle through the walls to make holes and push bolts through from the inside out. Use washers on both sides and fit and secure tightly two nuts on each bolt on the outside. Put some silicon rubber on top of the bolts and washers on the inside to prevent rust. Make two $6"$ long and two $2"$ long plexiglass strips and drill two holes in the longer strips and one in the shorter. Lay the nylon mesh in silicon rubber and fit over the square hole. Lay the plexiglass strips over and punch holes and bolt through the walls as before. Lay the clear plexiglass sheet in silicon rubber on top of the box and tape tightly. If desired, cut the lid of the polyfoam shipper to fit inside the box with holes cut out to hold food cups.

The cage is completely fly tight as constructed but care should be taken when introducing and removing food cups. I use disposable plastic cups made for yogurt as food cups. The cups measure 2" bottom diameter, 3" top diameter and 3" high and can hold 180 ml; the amount of medium is about 90-100 ml. A cage holds 12 such cups that can be rotated according to a schedule suitable for the species being studied.

The cage gets dirty after a while. To have a clear view into the cage, the inside of the plexiglass can be scraped clean with a razor blade. Alternatively, the food cups and the adults can be transferred to a clean cage. The adults are collected with a pooter using gentle suction provided by a diaphragm air pump (a suitable air pump is catalog number '1047-D'10 from Thomas Scientific, Swedesboro, NJ). The top of the dirty cage can then be removed and the cage cleaned with a detergent and plenty of hot water. The top can be fitted in fresh silicon rubber and the cage reused.

Cages made after this design have been in continuous use in the laboratory for close to 5 years. They are still in good condition.

Acknowledgements: I thank Magnús Magnússon for the photography. During the designing of the cage I was supported by a Fulbright-Hays grant, a Fogarty International Research Fellowship NIH #F05 TWO3027-01, and a grant from the Icelandic Science Foundation (Visindasjóður Islands).


Figure 1. Population cages.
It is well known that male song plays an important role in Drosophila courtship behavior (e.g., Bennet-Clark & Ewing 1967). This song which is produced during wing vibration consists of two elements: sine song and pulse song (Schilcher 1976). Several variables of this song are species specific (Ewing & Bennet-Clark 1968) and it is likely that such interspecific variation plays a role in sexual isolation. Furthermore, intraspecific variation in courtship song might play a role in mate choice. To study the role which these variables play in the courtship song, a simulator is necessary.

However, in trying to build the simulator developed by Johnson & Cowling (1980), we found that many of their time controlling elements were not locked in phase. This caused difficulties in tuning all the variables. For instance, the sine used to create one pulse would start at a random phase rather than phase zero, thus causing many uncontrolled harmonics. More seriously, however, even the smallest misalignment would change the original sequence: pulse; sine; silence to, for instance, pulse; sine; pulse; silence. Furthermore, Kyriacou & Hall (1980) showed that the interpulse interval is not constant, but modulated by a slow wave of approximately one minute. This modulation also proved to enhance mating speed (Kyriacou & Hall 1982); we therefore also wanted to incorporate such a modulation in our simulator. These two modifications (locking the time controlling elements in phase and the introduction of the modulation) necessitated the design of a new system. This new courtship song simulator will be discussed here.

The logic diagram indicates how the repeating song sequence, as shown by the output signal, is produced. (See Table 1 for the variable definitions.) Every t1 sec. a trigger pulse generator triggers the pulse song duration timer (IC7) to give a block of t2 sec. which enables the generation of ipi trigger pulses with an interval of t4 sec. during t2. These very short pulses are responsible for synchronising the pulse song generator (IC4) and for starting a pulse bit counter (IC6). The pulse bit counter's output signal gates the pulses from IC4 to the output mixer in such a way that, with the switch on N, only N pulses are passed.
to the mixer each t4. The trailing edge of t2 starts the sine song duration timer to give one block of t3 sec. During this time the sine song generator is gated to the output mixer and gives a pure tone with a wavelength of t6 sec. However, after the start of t3 the gate opens only when the sine signal crosses groundlevel for the first time. Similarly, after the end of t3 this generator will continue to finish its last wave.

The two timers symbolised by IC7 in the circuit diagram belong to the same integrated circuit 1457813. Similarly, MC14066B serves three functions, CA3290 two, and HEF4049B two functions. The controls are ordinary one turn potentiometers and specify all the times. The n switch, however, is a ten position switch and determines the exact number of cycles per pulse. The power supply, audio amplifier and speaker, being commercially available, are not given. The power consumption is 100 mA, the output (peak to peak) 1V, and the output impedance 50 KiloOhm.


Drosophila workers of tropical countries (like India, Bangladesh, etc.) repeatedly attempted to develop a cytological procedure that shows a better preparation of polytene chromosome of Drosophila for in situ transcription and in situ hybridization techniques. But, unfortunately, none of the procedure would give the optimum result after hybridization and transcription. This present procedure should exhibit well spread and evenly flattened polytene preparations, that are more essential for in situ transcription as well as in situ hybridization. Such a preparation shows a unique morphology of all the chromatins and gives the maximum transcription and hybridization signals.

The procedure is as follows:

[1] Salivary glands of well-nourished third instar larvae were dissected out by hand in buffered Ringer’s solution (NaCl - 7.5 gms, KCl - 0.14 gms, NaHCO3 - 0.2 gms, CaCl2 - 0.12 gms, Na2HPO4 - 0.01 gm in 1 litre double distilled water at pH 7.2) (Berendes 1973).

[2] Two pairs of glands were shifted with a droplet of 50% acetic acid (the fixative) on a small siliconised coverslip (18 mm²). Small coverslips provide such facilities that chromosomes will fix in a restricted zone. Ringer’s solution should never be transferred along with the glands, since acetic acid dilution causes poor fixation of the glands.

[3] The glands were fixed for 2-3 minutes. A clean slide was slowly lowered on to the coverslip. The slide was turned over in upside down direction. The coverslip was tapped gently with a pointed object, such as the tip of the needle. With the help of this tapping, coverslip possibly moves slightly sideways. This step is intended to force the chromosomes out of the cells and to settle in a well spread manner. The preparation was examined by a phase contrast microscope.

[4] The slides containing well spread chromosomes were placed on a filter paper with its coverslip down and wrapped with this paper. The coverslip was pressed very hard directly with the thumb. At this point, precaution should be taken that coverslip must not change its previous position. The thumb pressure will only flatten the chromosomal arms without improving its spreading, and thus make a good morphology.

[5] The prepared slides were placed on a hot plate (that was previously warmed to 40°C) for 10 seconds. The heat shock makes the chromosome to adhere to the surface of the slides and thus help the retention of maximum number of nuclei upon the slides.

[6] Immediately, after the heat shock, the slides were then placed into a horizontal coupline jar containing 50% acetic acid vapour and those were kept for one hour in coupline jar. In this step, maintaining appropriate time is very much vital; if the incubation time is reduced, it leads to poor morphology of the chromosome. Otherwise, if the incubation time is more than one hour, acetic acid vapour will reduce the amount of DNA, and will exhibit poor transcription and hybridization.

[7] The slides were immersed in a mixture of acetic acid and 50% ethanol (1:1) for 20 minutes, then the coverglass was flicked off by sliding a sharp razor blade under one corner of the slide within the mixture. Just after the removal of coverslips, the preparations were plunged into 70% ethanol at room temperature for 5 minutes.

[8] The slides were put through one wash in 90% ethanol and one more wash in 100% ethanol, 5 minutes each.

[9] The slides were allowed to air dry. In dry condition, slides could be stored for a longer period. The best preparation for in situ transcription and in situ hybridization is flat and grey chromosomes with no refractivity. The banding pattern should be easily distinguishable.

In this procedure, use of formaldehyde, acetoacetyl alcohol as fixatives were avoided, because these interfere with the denaturation of the double stranded DNA. And fixative, like acetic acid:methanol (3:1) mixture may help to lose of large amount of DNA and reduced transcription and hybridization. Again, excessive acid treatment (such as aceto-orcein, aceto-carmine) may depurinate the DNA and exhibits the poor level of hybridization and transcription.

Acknowledgement: The authors are grateful to Council of Scientific and Industrial Research for financial assistance.

with reduced flight ability were found stuck to the lower levels of the cylinder having fallen further before reaching the sides of the column.

While using this test system we encountered the problem that the temperature-sensitive nature of the viscosity of the oil critically affected the efficiency of the cylinder to trap flies. To counter this problem, we have replaced the paraffin oil with a commercial compound for ecological sticky traps: "Oecotak" [Oecotak A5 from OECSOS Ltd, 130, High Street, Kimpton, Herts., ENGLAND]. Its viscosity is unaffected in changes in temperature between 15-30°C. The "Oecotak" is applied with a hand roller to acetate sheets which are used to line the inside of a glass cylinder (G) (Figure 1). Flies are introduced directly from culture vials through a plastic funnel (F) and the flightless flies collected in a beaker (B). Measures of the flight ability of different strains can be made, either as a percentage of introduced flies recovered in the beaker, or from the positions of stuck "flighted" flies. In the latter case, the acetate sheets are removed from the cylinder, flattened, and placed on a grid to record the positions of stuck flies. This can also be done on a digitiser pad connected to a microcomputer. The sample mean and variance of the distance travelled from the point of introduction can then be computed.

Both Benzer's "greased" cylinder, and our "Oecotak" cylinder cause destruction of tested flies. This prevents repeated flight-testing of a sample. Such repetition could improve the separation of flighted from flightless flies, and facilitate the investigation of such phenomena as temperature sensitivity and age degeneration of flight ability. A non-destructive test could also be used to select flighted revertants of flightless mutations.

We have therefore devised a non-destructive flight-tester. In this system, the cylinder is formed from blotting paper (P) held in place by two PVC collars (C), onto which polypropylene funnels (F*) are taped (Figure 2). As before, flightless flies fall through the column and are collected. Flighted flies land and grip the paper. The first collecting vial (CV) is removed, and replaced by a new vial (whose base has been removed and replaced by a fine nylon mesh). The flighted flies are then simply blown out of the cylinder into the collecting vial.

The effectiveness of the paper column in separating flighted and flightless Drosophila is demonstrated in the following experiment: Wild-type Oregon flies were mixed in a vial in varying proportions with flies of a marked flightless strain, st pp M320 (M320 is a mutation of actin 88F causing a reduction in FM-specific actin). The flies were tested for flight ability in the paper column, and the flies in both the "flightless" and "flighted" vials were then re-tested (separately). The flies finally collected in the "flighted" vials were scored as to eye colour phenotype. The results are displayed in Table 1.

Using this non-destructive column, we plan to select for flighted revertants of flightless mutants. Such revertants may serve to isolate further genes involved in the flight system.

A quick and easy method is described to stain the chromosomes of whole Drosophila embryos (prior to blastoderm formation) using the fluorescent dye, 4, 6-diamidino-2-phenylindole (DAPI): eggs of *Drosophila hydei* in early developmental phases were collected and transferred into small baskets (baskets according to Widmer & Gehring 1974). During the whole procedure the eggs remained in them. Between each preparation step till fixation, they had been washed, pipetting Drosophila Ringer several times into the basket. After a careful wash in running water to remove medium, the eggs had been laid in, dechorionization was carried out placing the baskets into a 3% sodium hypochloride solution (Hill 1945; Rickoll 1976) for 2-3 minutes. Proper dechorionization was checked by means of a stereo microscope. To permeabilize the vitelline membrane, the baskets were immersed into octane (Limbourg & Zalokar 1983) for 2 minutes. Fixation in a mixture of methanol and glacial acetic acid (3:1) followed during the ensuing 30 minutes. Thereafter eggs were carefully washed in Ringer’s solution and placed into tris-buffered solution (tris-(hydroxy-methyl)-aminomethan; 0.18 M; pH 7.5) of DAPI (10^-6 g/ml; Stohr et al. 1980). The eggs were then washed in tris-buffer and -- after opening the baskets -- cautiously transferred into a droplet of tris-buffer on an object-slide. They were protected using a coverslip and observed by means of a fluorescence microscope (Zeiss No. II; filter combination: BP 365/10, FT 420, LP 418). In that way it is possible to visualize the chromosomes in situ. The technique is of interest for embryological as well as cytological studies (Fig. 1 and insert).


Figure 1. Whole Drosophila embryo with the chromosomes stained using DAPI. (x480, inset: x 1200).

Kalisch, W.-E. and T. Whitmore. Ruhr-Universität Bochum, FR Germany. The SSP chromosome preparation technique as applied for *D.melanogaster*. The intention of this technical note is to give a reply to the requests we have received asking for a more detailed description of the surface-spread polytene (SSP) chromosome preparation technique (Kalisch & Hägele 1981; Kalisch et al. 1984) and especially its adaption for the use with *D.melanogaster*. Quite recently, the technique was used successfully for high resolution mapping of in situ hybridized biotinylated DNA to salivary gland chromosomes of *D.melanogaster* (Kress et al. 1985). Since the technique used by Kress et al. (1985) differs in several methodological details from the optimized one we are now using, we feel it thus necessary to give the following up-to-date description of our technique with specific reference to the salivary gland chromosomes of *D.melanogaster*. The spreading effect and the chromosome cytology is given in Fig. 1 as an example and compares the reference map of Lefevre (1976) (from squash preparations) and a SSP chromosome preparation.

Preparation of salivary glands. After a brief washing of the larvae in a pre-treatment solution, the salivary glands are excised with forceps3 in ca. 16 µl of pre-treatment solution. It is not recommended to use Ringer-solution for this preparation step (for detailed reason, see Kalisch & Whitmore 1983). The glands are immediately transferred to a 10 µl vial, which is filled with 8 µl of pre-treatment solution (60 vials on one disposable test plate: No. 163118; NUNC GmbH, D-6200 Wiesbaden, FR Germany). After removal of any remaining parts of the fat bodies, the glands are mechanically lacerated with forceps. This procedure is controlled under the binocular, where the bursting of the cells is visible. The lacerating of the glands is, if necessary, continued until the glands disappear into a turbid ‘chromosome suspension’. The pre-treatment procedure is terminated after 5 min (from excising the glands) by the spreading procedure.
Figure 1. Salivary gland chromosome divisions 71-80 (3L) of D. melanogaster: Comparison between the electron micrograph of a single SSP chromosome preparation and the light micrograph of well-extended chromosome squash preparations (reference map of Lefèvre 1976). Under the assumption that the squash preparation represents a chromosome diameter of 5-8 µm, then both chromosomes show the same magnification. It should be noted that the quality of the light micrograph is reduced by copying it from a reprint. Furthermore, it has to be considered, that Lefèvre's reference map is a composition of several well-extended chromosome preparations. By this, the longitudinal (axial) degree of spreading of the SSP chromosome appears reduced from what is normally observed in comparison with routine squash preparations. Bar represents 10µm.
Spreading procedure. (Fig. 2): Half a spherically shaped drop (Ø = ca. 1.5 cm) is formed by 1.5 ml of a spreading medium on a double siliconized microscope slide. A siliconized MICROCAP is used to transfer the pre-treated chromosomes (the 'chromosome suspension') from the 10 µl vial to the surface of the spreading medium. For each spreading procedure, a ca. 4 µl drop of the 'chromosome suspension' is formed at the lower end of the MICROCAP and after waiting for about 30 sec the drop is brought into contact with the zenith of the spreading medium surface. At the moment when both drops touch each other the half spherically shaped drop of spreading medium is clearly flattened. In those cases where the spreading medium does not show this effect, the MICROCAP has either been pushed through the surface of the spreading medium or too small amount of 'chromosome suspension' has been used. In either case the spreading procedure has to be started again using a new drop of spreading medium.

SSP chromosomes: The spread chromosomes remain on the surface of the spreading medium. They can be visualized using a phase optic (Achromat 10/0.22 Ph, No. 460401.9904.000, ZEISS). To enlarge the area of visibility with the phase optic in connection with the curved surface of the spreading medium, we mechanically flatten the surface of the spreading medium. Spread chromosomes slipping to the rim of the surface of the spreading medium are brought back by blowing air over the surface of the spreading medium from different directions.

Picking up the chromosomes (Fig. 3): SSP chromosomes are picked up on Carbon-coated Formvar grids. In order to obtain the entire band-interband pattern of a chromosome or even an entire genome, we use grids with one 500 x 1000 µm mesh (Type G200, No. 01197; BALZERS GmbH, D-6200 Wiesbaden-Nordenstadt, FR Germany). For the process of picking up a chromosome, a pair of forceps (holding the grid) are secured in a seesaw type construction. By this arrangement, it is possible to visualize both the grid and the SSP chromosome simultaneously allowing the proper adjustment of the mesh over the chromosome.

After picking up a SSP chromosome by touching the spreading medium with the grid, the EM grid is carefully washed by floating the grid, filmed side down, for 10 min on the surface of aqua bidist., dipping several times in 50% and transferring to 96% isopropanol for 10 min. Air-drying of the grids (filmed side up) is done on a filter paper (since the grids have been bent slightly before being covered with the Formvar-film, a contact between the film and the filter paper can be avoided). There is no contrasting procedure used with this technique. The grids can be stored without a changing of the band-interband pattern, but time may weaken the strength of the Formvar film.

For LM analyses, SSP chromosomes can be picked up on subbed slides or cover slips (for this procedure, we hold the subbed slide at an angle of 90° to that of the slide where the spreading medium is on). If the SSP chromosomes are blown from the rim to the middle of the surface of the spreading medium after the pick up, it is possible to get even a second and third LM preparation from the same drop of spreading medium. The washing procedure is the same as for EM grids.

Cytological analysis: We start to analyze the degree of spreading and the quality of the SSP chromosomes on the EM grids by LM analysis (16/0.40 Ph, Zeiss). For the EM analysis of the SSP chromosomes we use a Siemens ELMISCOP 101 at 40 and 60 kV, respectively. Usually we take
micrographs with a x1,600 or a x2,400 magnification and use a final magnification of x3,200 and x4,800, respectively, unless overviews with a lower magnification have to be given as in Fig. 1.

1. Raising of larvae: Third-instar larvae of a D. melanogaster wild-type strain (Berlin) were used in this case. Culture conditions were the same as commonly used by others for yielding proper chromosome squash preparations. Second- and third-instar larvae were raised at 18°C and fed with a daily supplement of baker’s yeast. In order to ensure an optimal food supply third-instar larvae were transferred once to fresh medium.

2. Pre-treatment solution: For this 3.18 M citric acid 1-hydrate and 8.82 M propionic acid solution, 10 g of a citric acid 1-hydrate (No. 244; MERCK, D-6100 Darmstadt, FR Germany) is dissolved in aqua bidist. by stirring and gentle heating and brought to 15 ml. Separately, 19.8 ml of propionic acid (No. 800605; MERCK) is mixed with 10.2 ml aqua bidist. Both acid solutions are carefully mixed together and can be stored in a tight vial for several days. Depending upon the conditions of the stock being studied, other ratios of the solutions than the 1:2 above (for example, 1 part propionic to 1 part citric acid) may be necessary to provide the required degree of chromosome spreading.

3. Forceps: For preparation of the salivary glands and for picking up the spread chromosomes from the surface of the spreading medium, we use the following forceps: EREM SWISS 5SA (BALZERS). For salivary glands preparations, the forceps are sharpened (we use a special instrument for sharpening: No. B8010 03 031; BALZERS). For handling the EM grids during the washing procedures we use an extra forceps (No. B8010 03 097; BALZERS). Forceps are demagnetized (No. B8010 03 032; BALZERS) after sharpening and cleaned daily either in alcohol or ultrasonically.

4. Spreading medium: The 4 M urea and 0.1 M HCl solution is prepared as following: 24 g urea (No. 8487; MERCK) and 10 ml of 1M HCL are brought to 100 ml aqua bidist. A pH of 2.0 is achieved by additional 1M HCL.

5. Siliconizing: For siliconizing we use exclusively a commercially available silicone solution (No. 35130; SERVA, D-6900 Heidelberg, FR Germany). After siliconizing the microscope slides we dry them horizontally for 60 min at 100°C. Otherwise, there will be a gradient of silicone from one side to the other, which results in a movement of the spreading medium to one side when the drop is formed or during the spreading process. The slides are siliconized twice. We use each slide for only one spreading preparation.

6. MICROCAP: The 10 µl MICROCAP (Drummond Scientific Co., USA) is connected (using the MICROCAP holder) with a parafin-oil filled plastic tube (see Fig. 2) which itself is connected with a parafin-oil filled 100 µl HAMILTON syringe (Typ 1710LL). The plunger of the syringe is connected directly with a micrometer (NSK, Japan Micrometer). An airspace of max. 3 cm should be left between the MICROCAP connection and the start of the parafin-oil. The siliconized MICROCAP is discarded after every few spreading procedures.

7. Spread chromosomes: Well-spread chromosomes are more or less free of those types of thick protein conglomerates which strongly reflect light as usually seen in squash preparations. Extremely well-spread chromosomes have a very low contrast by the optical system used making them difficult at first to locate. Contamination through too much accompanying cellular material can influence the quality and degree of spreading.

8. Flattening of the spreading medium: We use a disposable hypodermic syringe with a 20G1/2 (0.90 x 38 mm) needle which is connected with a rubber tube to a plastic tube connector which serves as a mouthpiece. The syringe can be injected at any area of the spreading medium. With the needle near the center of the drop (to avoid too much circulation) the spreading medium is sucked off very slowly to avoid a deformation of the drop at the rim. In those cases where inlets do appear, they can be removed by moving the hypodermic needle (together with the adherent spreading medium) to the old border of the rim.

9. Air blowing: We use a mouth controlled rubber tube, which is capped with a disposable PIPETMAN C20TJ tip. The extent to which this technique works depends upon the amount of cellular material accompanying the SSP chromosomes on the surface of the spreading medium. Furthermore, it should be emphasized, that a flattened surface of the spreading medium supports the efficiency.

10. Formvar grids: An extremely durable film is needed for the 500 x 1000 µm mesh of the grids used (No. B8010 01 197; BALZERS). We use a concentrated Formvar solution which yields a film ca. 3-4 times thicker than that normally used. The filming of the grids is carried out with certain modifications as described by Lickfield (1979), whereby the humidity used plays an important role in the preparation of optimal films: (1) Several microscope slides are cleaned with a linen cloth which has been boiled beforehand in distilled water. This is repeated with a new cloth. (2) A 0.25-0.35% solution of Formvar (Formvar 1595 E, No. 21740, SERVA) is prepared in chloroform (or in 1.2-dichlorethane) and stored in a bottle with an opening large enough for a microscope slide. (3) By 38% humidity one of the clean microscope slides is dipped into the Formvar solution, pulled out slowly and left to dry for ca. 24 hrs at the same humidity. (We use a plexiglass box for this step which has an opening for a hand. Use a vinyl (rubber) glove, otherwise the film may appear fogged. To keep the humidity at ca. 38% any of the typical dry gels should work. The opening in the plexiglass box should also be kept relatively closed with any type of
flexible (vinyl) plastic. (4) The dried, film-coated slide is scored along the edges with a razor blade. (5) The filmed microscope slide can be now slowly slid into a petri-dish filled with distilled water at an angle of ca. 5-10°. Thereby the film on the microscope slide floats onto the surface of the water. (6) The defilmed microscope slide is pulled out of the petri-dish. (7) Electron microscope grids are placed with the dull side down on the film. (8) A strip of either filter paper or parafilm is lowered onto the grids and as soon as it sticks to the film, the strip is pulled with the grids out of the petri-dish. To prevent a contact between the film and the filter paper the grids are bent slightly beforehand. (9) The filter paper (parafilm) strip is placed with the filmed EM grids on top in a petri-dish lined with filter paper to dry. (10) The petri-dish is closed with a glass lid. (11) In order to further stabilize the film we often carbon coat the grids.

11. Seesaw: A forceps frame consisting basically of two parts (1 and 2 in Fig. 3) which serve the function of a pivot point for the forceps. The forceps can be swivelled with the one end holding the grid and the other one being used for hand-directioning. Part 1 of the seesaw (a 25 x 20 x 15 mm aluminum block with a 4 x 10 x 15 mm lip on which the part 2 is attached with a screw) can be moved on the microscope table. Part 2 is an 'instrument holder' (part of the micromanipulator ‘de Fonbrune’, type PM1; BACHOFER GmbH, D-7410 Reutlingen, FR Germany). As a very primitive alternative, the forceps can even be secured by a clothespin.

12. Washing procedure: The Formvar film is not very strong when it is wet. For the wash in 50% isopropanol the grids should only be moved with the film in the vertical direction. We use exclusively plastic vials (35 x 10 mm, No. 15031; NUNC) for these processes, which are disposed of after half a day to avoid protein contamination.

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Laverty, T.R. University of California, Berkeley, USNA. A device for dispensing instant Drosophila food.
The instant food is poured onto the top of the box. The food is spread over the top with a brush filling all the holes. The false bottom is then removed and the food falls into the vials. Water with the appropriate chemical can then be easily added to the food with a repeating syringe.

Using this device all the vials can be prepared identically and quickly. Therefore, any differences observed between crosses will not be due to inconsistencies in the media. Also this device is excellent to use when preparing instant food for normal uses.

There are several obvious advantages to using biotinylated probes for in situ hybridization in place of radioactive probes. Unfortunately, many laboratories have encountered serious difficulties in their attempts to make this technique work routinely. During the last few months, we have established a procedure which allows us to achieve consistently good results. Our protocol is derived from published accounts as well as personal communications of details that often escape journal reports. We are especially indebted to Dr. John Thomas of Stanford University for many valuable suggestions which were essential to the development of this procedure.

1. Preparing polytene chromosomes for in situ hybridization. Chromosome squashes were prepared essentially as described by Atherton & Gall (1972). Drosophila larvae were harvested from well-yeasted, uncrowded culture bottles. The best chromosome squashes were obtained from third instar larvae that had crawled up the side of the bottle and had stopped moving. Larvae were dissected in 45% acetic acid. The salivary glands were removed and most or all of the attached fat bodies were teased away. Dissection of some Drosophila species was particularly difficult. In such cases, larvae were dissected in Meyer's solution and the salivary glands were transferred immediately to 45% acetic acid.

After dissection, one pair of salivary glands was transferred to a 10-15 microliter drop of 45% acetic acid placed on a very clean (dust free) coverslip. (The coverslips were siliconized using the instructions contained in a bottle of Prosil-28 (SCM Specialty Chemicals), dried, and stored in the original container. Each day, the siliconized coverslips were placed in 95% ethanol until needed. Immediately before use, a single coverslip was dried with a paper lab wipe and blown clean with a stream of pressurized gas.) The drop was placed directly at the center of the coverslip so that it would be easy to locate the chromosomes later. The temptation to put more than one pair of glands under the coverslip should be avoided. In an ideal squash, the cellular debris flowed away from the chromosomes; too much tissue precluded this. An equally clean "subbed" slide (Gall & Pardue 1971) was lowered onto the coverslip so that three edges of the coverslip were closely parallel to and equidistant from the three edges of one end of the microscope slide. The coverslip was picked up with the subbed slide, with the coverslip "hanging" down. The coverslip was displaced laterally in all four directions in turn (to facilitate spreading of the chromosomes) by tapping the edges of the slide against a pencil (W.J. Dickinson, pers. comm.). The slide was turned over and examined with a phase contrast microscope to determine whether the nuclear envelope had been broken. If the chromosomes were sufficiently free of the nuclear envelope, the slide was placed on blotting paper, coverslip down, and the excess acetic acid was carefully removed by pressing lightly on the slide. As the acetic acid was blotted, the chromosomes spread. If too much pressure was exerted at this stage, the chromosomes were stretched and/or broken. The spreading was monitored frequently with the microscope. As the chromosomes were flattened onto the subbed slide, more pressure could be applied without stretching the chromosomes. The need for diligent care and patience at this stage cannot be over-emphasized. Sufficently flattened chromosomes had little or no refraction along their edges. The slide was placed coverslip up on a 45 C warming plate to increase flatness. After at least one minute, the warm slide was placed, coverslip face up, on a flat table top. With one sheet of blotting paper over the slide and the coverslip, the coverslip was pressed very firmly with the thumb. If the chromosomes were not squashed very flat, resolution was greatly impaired.

When the slide had cooled to room temperature (to prevent fracturing the glass), it was dipped into liquid nitrogen for a few seconds (until boiling subsided). The frozen coverslip was then removed immediately by inserting a razor blade under a corner of the coverslip and flipping it off. The slide was immediately plunged into freshly made absolute ethanol:acetic acid (3:1) which had been chilled to -20 C. After 2-3 minutes, the slide was transferred to 95% ethanol and left there for 30 minutes to 2 hours. It was then rinsed in 100% ethanol for about 2 minutes and air dried. Slides were examined without coverslips by phase microscopy to determine if they were suitable for hybridization. Suitable slides had undistorted chromosomes, very little refraction along the edges of the chromosomes, and clearly visible and distinct bands. If most of the chromosomes on a slide were not suitable, the slide was discarded.

Piley, M.D., J.L. Farmer and D.E. Jeffery. Brigham Young University, Provo, Utah USNA. In situ hybridization of biotinylated DNA probes to polytene salivary chromosomes of Drosophila species.
The slide was incubated for 30 minutes in 2XSSC (SSC is standard saline citrate: 0.15 M NaCl, 0.015 M sodium citrate) at 70 C. The slide was then dehydrated in 70% and 95% ethanol for 5 minutes each and air dried. Chromosomal morphology seemed to be improved by the 2XSSC wash, air drying, and dehydration.

The chromosomes were denatured by soaking the slide in 0.07 N NaOH for 3-7 minutes. The time required to denature the chromosomes varied from species to species (D. melanogaster chromosomes denatured within 3-4 minutes; D. silvestris and D. virilis chromosomes seemed to require at least 7 minutes). The slide was transferred immediately to 70% ethanol, dehydrated through 95% ethanol, and air dried, as above. It was essential for good hybridization that the chromosomes were denatured sufficiently, even at the cost of some decrease in resolution of chromomeres. Chromosomes were denatured immediately before hybridization. Denatured chromosomes which had been stored before use did not hybridize well.

II. Nick translation. The probes which we used were recombinant plasmids which contained D. melanogaster DNA. The probes were purified using an alkaline lysis technique, RNase A and proteinase K digestions, phenol extraction, and ethanol precipitations in the presence of ammonium sulfate and then sodium acetate. Where possible, the precipitated DNA was collected with a glass hook rather than by centrifugation. They were not purified by density gradient centrifugation or by gel exclusion chromatography. The OD260/OD280 ratio was generally in the range of 1.8. The preparations were free of bacterial DNA and all but a trace of digested RNA fragments as judged by ethidium bromide staining of agarose electrophoretic gels. DNA concentration was estimated assuming that a 50 mcg/ml solution has an OD260 of 1.

Recombinant D. melanogaster DNA probes were labeled with biotinylated dUTP using a nick translation kit. (Although the details given below refer to the Bethesda Research Laboratories nick translation kit with biotin-11-dUTP, we have obtained equivalent results using the nick translation kit from Enzo Biochem with either biotin-11-dUTP or biotin-16-dUTP.) We followed the protocol on the BRL biotin11-dUTP data sheet or the protocol supplied with the Enzo Biochem kit. When using the BRL kit we stopped the reaction by adding stop buffer (omitting the SDS) and incubating at 65 C for 10 minutes. We no longer use radioisotopes to monitor the nick translation reaction, since we have never had one fail.

The labeled DNA was separated from unincorporated nucleotides and other molecules on a 0.9 cm diameter x 15 cm Sephacryl-200 (Pharmacia) column equilibrated with TE (0.01 M tris [trishydroxymethyl]aminomethane), 0.001 M EDTA, pH 7.5). Labeled DNA was mixed with 12.5 mcI of 0.5% blue dextran (BRL Focus 7(1):8). The blue dextran co-chromatographed with the DNA and did not interfere with hybridization. Fractions of 7 drops (approx. 39 mcI/drop) were collected. The DNA and blue dextran peak was contained in 2 or 3 tubes which were pooled. Polypropylene microcentrifuge tubes were used for the subsequent steps to minimize the loss of DNA. The DNA was precipitated with 0.1 volume of 1 M sodium acetate, 20 mM magnesium acetate, pH 7.7, and 2.5 volumes of 95% ethanol and placed at -20 C overnight or at -70 C for 10-15 minutes. The DNA was collected by centrifugation at 10,000 rpm for 10 minutes in a microcentrifuge at 4 C. The supernatant was carefully removed by aspiration and the blue pellet was air dried.

The pellet was dissolved in 100 mcI of distilled water and 17 mcI of 12 mg/ml sonicated salmon testes DNA and heated in a boiling water bath for 5 minutes. The tube was immediately plunged into an ice bath. The remaining ingredients of the hybridization solution were then added: 40 mcI 20XSSC, 4 mcI 50X Denhardt's solution (Denhardt 1966) and 40 mcI 10% dextran sulfate (MW 5,000). The final concentration of the labeled DNA was approximately 5 mcg/ml. This mixture can be frozen, thawed in an ice bath, and used for hybridization at least 5 times. Alternatively, the DNA solution can be divided into smaller portions so that it is thawed fewer times before use.

III. Hybridization. Although we have described the following procedure for a single slide, we have routinely hybridized and stained several slides at a time.

The probe concentration used in our standard procedure, described below, was higher than it needed to be for good hybridization. We have tried hybridizing two or four probes to the same slide, thus decreasing the amount of each probe accordingly. Two probes hybridized as well as a single probe. Four probes did not hybridize as well as a single probe. Therefore, we suspect that the probe concentration could be reduced to one-half or perhaps one-third of the concentration which we normally used.

About 12-14 mcI of the probe solution were placed over the chromosomes on a slide which had been prepared for hybridization as described in Section I above. A coverslip that had been washed briefly in 0.5 N HCl, rinsed in distilled water, and wiped and blown free of dust was set on the drop of probe solution. To prevent evaporation from the edge of the coverslip, it was sealed to the slide with rubber cement which had been thinned to an appropriate consistency. The rubber cement was allowed to flow from the brush as rosy strands around the edges of the coverslip. Aesthetic values were ignored.

In order to concentrate for possible poor seals, the slides were incubated in a humid chamber. A paper towel which had been cut to fit the bottom of a plastic sandwich box was placed in the box and saturated with 2XSSC. The slide was incubated in the sealed box at 65 C for about 12 hours. Some reports
have indicated that as little as 3 hours is sufficient for hybridization when using dextran sulfate and
Denhardt's solution. Other reports have suggested that hybridization for more than 18 hours damages
chromosomal DNA (Barbera et al. 1979).

The rubber cement and the coverslip were removed. The slide was washed four times in 2XSSC at
60 C for 10 minutes. It was then washed twice in 1XSSC at room temperature for 10 minutes. The slide
was transferred from the final 1XSSC wash to the first wash in the next section without allowing it to dry.

IV. Detection of the probe. The probe was detected using the BRL DNA Detection Kit. The slide
was washed for 60 minutes at room temperature in BRL Buffer #1 (0.1 M Tris-HCL, pH 7.5, 0.1 M NaCl,
2 mM MgCl2, 0.05% (v/v) Triton X-100) supplemented with 1% BSA. (The BSA is used to remove label
adhering to chromosomal proteins. Since BSA is an excellent bacterial nutrient, the solution should be
sterilized or else BSA should be added to Buffer #1 immediately before use. All steps involving BSA or
subsequent to its use should be done quickly, preferably the same day, to minimize bacterial growth.
Bacterial proliferation may render the chromosomes unacceptable for microscopy.) After the 60 minute
wash, the slide was allowed to drain but not to dry.

Several drops (about 50-200 mcl) of streptavidin solution (2 mcl of BRL streptavidin solution in 1
ml buffer #1) were dropped gently onto the chromosomes. Evaporation was minimized by placing a
coverslip over the chromosomes, placing the slide in a shallow container, and covering the container with
aluminum foil. The slides were incubated at room temperature for 2 hours (John Thomas, pers. comm.).
The coverslip was removed and the slide was washed three times for 10 minutes each at room temperature
in buffer #1. The slide was allowed to drain but not to dry.

Several drops (about 50-200 mcl) of poly-alkaline phosphatase solution (2 mcl of BRL poly-alkaline
phosphatase solution in 2 ml Buffer #1) were layered over the chromosomes. Coverslips and an aluminum
foil-covered container were used as before to minimize evaporation. The slides were incubated at room
temperature for two hours. The coverslip was removed and the slide was washed twice with BRL Buffer
#1 for 10 minutes at room temperature and twice with BRL Buffer #3 (0.1 M Tris-HCL (pH 9.5), 0.1 M
NaCl, 50 mM MgCl2) for 10 minutes at room temperature. The slide was allowed to drain but not to dry.

A few drops (about 50-200 mcl) of BRL dye solution (3.3 mcl NBT, 2.5 mcl BCIP, 0.75 ml Buffer #3)
were placed on the slide under a coverslip and incubated for 1-4 hours at room temperature in the dark.
Staining was occasionally monitored with the phase microscope. The stained band has sometimes become
visible in as few as 15 minutes but more time may be required. When the staining was adequate as
determined by observation with the microscope, the reaction was terminated by rinsing the slide
extensively in distilled water. Extensive rinsing was needed to remove salt crystals which interfered with
microscopy.

Slides were air-dried and stored dry without coverslips. For microscopic examination, re-hydration
with distilled water under a coverslip provided excellent resolution for viewing and photographing with
phase contrast.

The stained, hybrid bands did not fade over a period of 4-6 months when slides were stored in the
dark. We routinely photographed chromosomes on color slide film through a phase microscope without
filters. This procedure gave excellent resolution and provided a permanent record of the color contrast.
Good results were also obtained with black-and-white film.

False-positive stained areas may occur at the broken ends of broken chromosomes and in folds where
a chromosome is kinked or where two chromosomes cross each other. False positives in folds are usually
much narrower and sharper looking than stained areas over hybridization sites.

V. Conclusion. While the above procedure works well in our laboratory, we recognize that it is
almost certainly not optimal. There are several areas of uncertainty that should be mentioned: (1) In order
to achieve hybridization on a chromosome which still has easily resolved chromomeres, there needs to be
a balance between sufficient and excessive denaturation. Too little denaturation prevents DNA
hybridization, but excessive denaturation causes the chromomeres to fade. This elusive "balance" is not
the same for all species. (2) The details of the squashing technique, primarily timing and squashing
pressure, are not the same for all species. (3) The percent homology between probe DNA and chromosomal
DNA necessary to allow detectable hybridization is unclear. We have found that D.melanogaster coding
sequences hybridize well to D.virilis and D.silvestris chromosomes, but non-coding sequences hybridize very
poorly or not at all. However, we have not yet used a large number of probes. (4) We generally obtain
hybridization on every slide, but we seldom see hybridization to all of the appropriate chromosomes on a
single slide.

Notes added in proof: 1. We now add the carrier DNA to the labeled DNA immediately after we
stop the nick translation, before chromatography. 2. We have hybridized in the presence of dextran sulfate
of two sizes (5,000 and 500,000 daltons) and both worked equally well. The smaller size is easier to work
with because it is less viscous in solution. 3. We seem to get better hybridization if we lower the
temperatures given above by about 2 to 5°C.

References: Atherton, D. & J. Gall 1972, DIS 49:131-133; Barbera, E., M.J. Caliani, M. Pages & C. Alonso 1979,
Rabinow, L. and J. Birchler. Harvard University, Cambridge, Massachusetts USNA. Reciprocal translocations between balancers of the second and third chromosomes carrying a dominant temperature sensitive lethal and larval markers.

For a variety of reasons, we wished to construct a chromosome that would simultaneously balance both of the major autosomes as well as carry a dominant temperature sensitive (DTS) locus that would allow the investigator to eliminate individuals with this chromosome from the progeny of crosses involving it. It was also desired that this balancer would have scored without affecting the gross morphology. The simultaneous balancing of the major autosomes and subsequent elimination of the balancer in the second generation should be of general use in making homozygous recessive mutations or transformants.

The procedure used in the construction was as follows. The second chromosome balancer, CyO, carrying 1(2)513 DTS was crossed to a stock carrying the third chromosome balancer TM3, red e Ser. Males from the progeny of the above cross that had the Cy as well as the Ser markers were irradiated with 3000 rads and mated en masse to females of an Oregon-R stock. After three days the males were removed. Sons of this mating that had both the Ser and Cy markers were again mated to Oregon-R females. Of the 1012 pair matings, four gave progeny in which the two dominant markers invariably segregated together. These were the presumptive translocations between the two balancers.

One of these, designated #11, has been used to balance a variety of second and third chromosome lethals. Our experience with it to date suggests that it is an adequate balancer for mutants on either chromosome two or three. The 1(2) 513 DTS is reliably lethal at 29°C during larval and pupal stages, killing all progeny carrying the mutation. Adults are not affected, which permits crossing and laying at the restrictive temperature. Available upon request.

Redkar, V.D. Tata Institute of Fundamental Research, Bombay, India. A population cage designed to grow Drosophila melanogaster flies on a large scale. This cage can also be used to obtain embryos, larvae or pupae in large numbers. 20 to 30 gm of flies were obtained from this cage using about 500 ml of medium supplemented with glucose, sucrose and yeast tablet powder.

The cage consists of a rectangular box (27.5 cm x 23.0 cm x 28.0 cm), made of plexiglass glued with chloroform, with a plexiglass lid screwed on the flanged top, with a foam gasket (Fig. 1 to 4). The lid has windows fitted with fine nylon mesh for ventilation. Inside the cage there is an assembly of eight vertical plexiglass plates (pupation plates), held rigidly at its center by one end of a plexiglass screw with nuts N1 and N2 (Fig. 2 and 4). The other end of the screw passes through a smooth hole in the center of the lid and is held by a plexiglass nut N3 from the top. By turning this nut N3 the pupation plate assembly can be lowered or raised by about 2 cm guided by two plexiglass guide pins (Fig. 2 and 4). At the bottom of the cage a plexiglass tray (22.5 cm x 29.0 cm x 2.5 cm) slides smoothly inside the cage with a clearance of about 1 mm such that one such tray can be displaced and pushed out from one side of the cage by another similar tray into the cage from the other side.

Figure 1. Perspective view of the population cage.
Figure 2. Sectional front view of the cage. (1) Plexiglass screw 0.9 cm OD x 5 cm, that carries the pupation plate assembly. (2) Top lid of 0.5 cm plexiglass sheet. (3) Plexiglass screw, 0.5 cm OD x 2 cm, secures lid to the flanges. (4) Foam gasket 0.3 cm thick. (5) Plexiglass top plate of pupation plate assembly to which pupation plates are glued with chloroform and plexiglass strips. (6) Plexiglass guide pins, 0.5 cm x 5.0 cm. (7) A vertical plate in pupation plate assembly. (8) Outer wall of the cage of 0.3 cm plexiglass sheet. (9) Plexiglass tray.

Figure 3. Sectional side view of the cage.
Figure 4. Shows details indicated in Figs. 2 and 3.

Figure 5. Transfer boxes, in perspective view, shown attached to the cage.
Operation: The lid is fitted on the cage with the pupation plate assembly in raised position. 5 to 10 gm of ether-anaesthetised flies collected from bottle cultures are placed in a tray and the tray is introduced in the cage. The small gap between the tray and the cage is closed by cellophane-tape. Meanwhile another tray with normal agar-based Drosophila medium filled to about 5 to 7 mm below the top edge of the tray is prepared. After the medium cools and solidifies, 2 to 3 mm deep scratches, about 2 cm apart and parallel to the length and width of the tray are made on the surface. The surface of the medium is then made uniformly wet by 10 to 20 ml of an autoclaved solution containing 20% glucose and 10% sucrose. Finely ground yeast tablet powder is placed on the surface of the medium in small patches of about 0.5 cm in diameter with a clear space of about 2 cm in between. The yeast tablet powder patches should become wet; if not, some more sucrose-glucose solution is added. After this, half a gram of yeast granules (Baker's yeast) are sprinkled uniformly all over the surface. After the flies revive, the medium tray thus prepared is introduced in the cage by simultaneously pushing out the empty tray that was used to introduce the anaesthetised flies. To prevent flies from escaping while transferring the plates, two small flat boxes of plexiglass (see Fig. 5 and 6) are attached tightly to the two lower openings of the cage by cellophane-tape. Each of the two boxes is open from two sides and has inner dimensions same as a tray with a clearance of about 1 mm such that a tray slides smoothly through them. The left hand side box B1 has a small window in the centre at the top fitted with nylon mesh and a closed rectangular tube of plexiglass with a long slot of about 2 mm. In the righthand side box B2 the tray to be pushed inside the cage is inserted and then further pushed inside the cage by means of a wooden plate or another tray. While the tray inside the cage is being pushed out into the box B1, compressed air is forced in the rectangular plexiglass tube through the nozzle, connected by a tygon tube. This arrangement forces the flies sitting on the medium to fly back into the cage. After introducing the medium tray in the cage, the two boxes B1 and B2 are separated. The small gap between the tray and the cage is again sealed with cellophane tape. Any escaped flies in box B1 are ether-anaesthetised through the window and discarded. Eggs are collected on this medium for 24 hr with alternating 12 hr dark and light periods at 26°C. Using the transfer boxes B1 and B2 the egg-laden tray is removed and replaced by an empty tray. After introducing empty tray and sealing with cellophane tape, two tissue paper wads soaked in 1 to 2 ml of anaesthetic ether are placed on the two ventilation windows of the cage and covered with petridishes. After 15-20 minutes anaesthetised flies that drop on the tray are collected.

The medium on which eggs are laid is made moist by sprinkling some drops of the glucose-sucrose solution and this egg-laden tray is then introduced in cleaned, alcohol swabbed dry cage. This medium with
eggs is kept moist for two days by sprinkling 5-6 ml glucose-sucrose solution twice a day by sliding the medium tray out of the cage. On the third day when larvae appear to grow bigger, a little more glucose-sucrose solution is added to make the surface wet and fine powder of yeast tablets is sprinkled all over the surface in a thin uniform layer. At this stage the pupation plate assembly is lowered to touch the medium surface. This operation of adding sucrose-glucose solution and yeast tablet powder on the surface is continued for 5 to 7 days till all larvae pupate on the side walls of the cage and on the pupation plates. Every time glucose-sucrose solution and yeast powder have to be added, one has to raise the pupation plate assembly up and slide out the tray.

After the sixth or seventh day when no larvae are seen in the medium and before the flies start emerging from pupae, the bottom tray is removed, the lower part of the cage and pupation plate is washed and cleaned without disturbing the pupae, and a fresh medium tray is introduced every 24 hr. When all the flies have emerged and matured, the next batch of eggs is collected. Collection of flies is made in the manner described above by ether anaesthesia using the transfer boxes B1 and B2.

Comments: The cage is entirely made of plexiglass. All the morphogenic stages of the fly can be grown in one single unit of the cage. After initiation of the cage with 5 to 10 gm of flies from bottle cultures, the subsequent generations yield more flies. The humidity in the cage is maintained by the water-vapour evaporated from the medium. The flies lay eggs preferentially on the plane surface of the medium rather than on yeast powder. The hatched larvae grow well on the nutrient yeast powder with sugars, as seen by the crowding of larvae on yeast patches. Optimization of each of the operations and other factors such as size of the yeast tablet powder, moisture in the medium, humidity in the cage, feeding schedule of larvae, degree of crowding of flies, etc., can be achieved by experience.

Acknowledgements: The author is grateful to Prof. O. Siddiqi, Prof. U.W. Kenkare and Dr. K.S. Krishnan for useful suggestions and guidance.
Geimsa staining of emulsion coated in situ slides often results in a precipitate from the stain solution appearing as dark spots similar to high background labelling. Although it can be distinguished from real silver grains, the precipitate impedes scoring of slides and prevents good photography. Filtering the Geimsa solution before use can help to prevent this problem, but we have found an easy and more convenient method to clean up slides after they are stained and dried. This is to immerse slides affected by deposits in 100% methanol for 1-2 minutes, then to air dry the slides. This treatment clears background deposits with no noticeable reduction in chromosome staining. If some deposits remain, the slide can be retreated with methanol but prolonged treatment can destain the chromosomes. Ethanol is not suitable as the deposits are not removed quickly and the chromosomes are subject to destaining.

Tsuno, K. Josai Dental University, Sakado, Saitama, Japan. A new apparatus for horizontal electrophoresis.

In order to facilitate the analysis of mutation experiments involving isoenzymes, I improved the apparatus used in a horizontal thin-layer agar gel electrophoresis method previously described (Sasaki 1974, Tsuno 1981). For the purpose of shortening the time for both the transference of homogenates onto a gel and penetration of sample juice into gels, the apparatus was modified by using sampling combs (spatula for sampling the juice of flies).

The apparatus consists of three parts: homogenate plates, sampling combs, and an insertion gauge. (a) Sampling comb (made from a 0.8 mm thick acrylic sheet): as seen in Figure 1, the comb has 10 teeth placed in register with one row of holes on the homogenate plate. The tips of the teeth are roughened with sandpaper to increase the amount of sample solution that can be taken. (b) Homogenate plate (made from an 8 mm thick acrylic plate): as shown in Fig. 1, the plate has 50 holes (10 holes x 5 rows), spaced at intervals of 13 mm, each of which is 5 mm in diameter, 3 mm in depth, and has a flat bottom. (c) Insertion gauge (made from 5 mm thick polyvinyl chloride plates): it consists of two plates (400 x 200 mm and 350 x 150 mm) connected by a firm hinge, one plate being bent at the edge where a scaled guide plate is fixed so that the sampling comb is correctly positioned, as seen in Figure 2. In the resting position, the gel is protected by a stopper pad from being damaged by the upper plate. Both attachment and detachment of the sampling comb and positioning of the stopper pad are done by hand for each application.

The procedure for electrophoresis of *D. virilis* esterases is as follows: (1) A drop of water or buffer solution and one or more flies is put into each hole of the homogenate plate. Then, the samples are homogenized by a motor-driven glass rod 4.8 mm in diameter. (2) The sampling comb is then dipped into the homogenates in order to load each of the ten tips. (3) Next, the comb is transferred and fitted to the scaled guide plate of the insertion gauge, and the sample-laden comb teeth are then inserted two or three times into a 0.8 mm thick thin-layer agar gel prepared previously on a glass plate (180 x 165 mm). [Agar

Figure 1. Sampling comb and homogenate plate.

Figure 2. Insertion gauge prepared for delivery of sample into gel plate.
Figure 3. Examples of electrophoresis (α- and β- esterases of D.virilis).

Because of the possible necessity of further analysis, used homogenate plates having residual sample solution should be kept in a deep freezer to prevent drying until the electrophoretic results become clear. Sampling combs can be used repeatedly after a wash, but they should be dried vertically so as not to become bent.

Using this apparatus, I have carried out mutation experiments and examined about \(1.5 \times 10^6\) alleles of D.virilis α-esterase (Tsuno 1985). An example of electrophoregrams are shown in Figure 3. In Fig. 3, in order to accelerate the analysis of the experiments, a drop of sample solution containing homogenate of two flies was applied to one lane, so 2 (flies) \(\times\) 10 (teeth) \(\times\) 3 (times of insertion) \(\times\) 3 (sets) = 180 (samples) were examined on a single gel plate at a time. In this case, a mutant was found as indicated by the arrow on the gel.

So far, I have succeeded in electrophoresing isozymes of the following enzymes using this apparatus: α-esterase, β-esterase, acid phosphatase, alcohol dehydrogenase, α-glycerophosphate dehydrogenase in D.virilis, and some of the same ones in D.simulans and D.melanogaster. Moreover, a polyacrylamide gel instead of an agar one may be used in the test, and good results have been obtained with esterases of D.virilis. However, the agar gel method is better than the polyacrylamide gel one, because the preparation of agar gels is easier compared with that of polyacrylamide gels. Particularly, agar gels poured from agar boiled in an autoclave at 120°C and 1.2 kg/cm\(^2\) for 25 min gave good results.


Yamamoto, A.H. N.I.E.H.S., Research Triangle Park, North Carolina USNA. New protocol to prepare probes labelled by biotinylated dUTP for in situ hybridization of polytene chromosomes. Biotinylated dUTPs have been used to label probe DNA because of its long life and the safety relative to radioisotopes. However, I have experienced difficulty in getting probes sufficiently labelled with biotinylated dUTP. According to the usual nick translation method for \(^{3}H\)-dUTP or using nick translation kits supplied by several companies, incorporation rates of biotinylated-\(^{11}\)dUTP or biotinylated-\(^{16}\)dUTP measured by simultaneous incorporation of \(^{3}H\)-dUTP was less than 5% and those probes did not work. Now I recommend to separate the reaction of DNase I from that of polymerase I, as follows. For two slides:

1st reaction: 10 x nick translation buffer (Maniatis et al. 1982) 1.0 µl
DNA 0.5 µl
DNase I (fresh 1:80 dil. of 0.1 mg/ml stock solution) 1.0 µl
distilled H\(_2\)O up to 10.0 µl
Incubate at room temperature for 15 to 60 min (depending on quality of DNA) and heat at 65°C for 15 min.

2nd reaction: mixture of 1st reaction
10 x nick translation buffer 10.0 μl
2 mM spermidine 1.0 μl
dATP, dGTP, dCTP mix (0.3 mM each) 2.0 μl
Bio-16-dUTP (0.3 mM, from Enzo Biochem) 5.0 μl
3H-dATP (as a tracer) 0.5 μl
polymerase I 2.0 μl

Incubate at 14°C for 90 min. Add 2 μl of 0.5 M EDTA, and heat at 65°C for 15 min. Separate DNA from unincorporated dNTPs. Check incorporation rate (>10% is good enough).

The advantages of this protocol are not only being able to get probes of high quality but also being applicable to a range of DNA quality which may vary among batches, among methods for preparation or among persons who prepare them, by controlling the period of 1st reaction. Basically better quality DNA needs a shorter period for the 1st reaction.


SUBMITTED STOCK LISTS - all species

BERHAMPUR UNIVERSITY. Cellular Research Laboratory, Dept. of Zoology, Bhanja Bihar, Berhampur -7, Orissa, India.

Wild-type stock
1. D.melanogaster Oregon R
2. -do-
3. -do- local type
4. D.ananassae
5. D.hydei
6. D.virilis
7. D.nasuta

Mutant stocks

Chromosome 1
1. gt w
2. Ba sc (Muller - 5)
3. y w sn
4. y w f
5. Df(1)62 g
6. y w v g f l. e

Multiple Chromosomes
1. fs(1) K10 w
2. wco

HANYANG UNIVERSITY. School of Medicine, Dept. of Genetics, Seoul 133, Korea.

Wild stocks - D.melanogaster
Canton S
Oregon R
Seoul (Korea)

Chromosome 1
f
m
sc cv v f
v
w
w m f

Chromosome 2
a1 dp b pr ap b1t bw/SMS
b
β vg
bw
B1/In(2L + 2R)Cy, Cy bw45a sp2 or45a
bw
cn bw
dp cn bw
vg
vg bw

Chromosome 3
cu
e5
G1
ru h ta st cu sr e5 ca/TM3 ru Sb Ser
Sb/In(3LR) Ubx130, Ubx130 e5

Chromosome 4
ci sy n
gv1

Chromosome 2-3
T(2; 3) Pm; Sb/Cy, Ins; Ubx, Ins
LINKAGE DATA

Dept. of Biology, Boston College, Chestnut Hill, Massachusetts 02167 USNA.

Update of linkage information on the $s_{70}$ chorion gene of *D. melanogaster*.

The $s_{70}$ chorion protein gene was initially reported to be located within the A94 deletion (1E3-4;2B11-12) on the first chromosome. Using more detailed deletion mapping and the Staket strain which carries an electrophoretic variant of the protein, we have now shown the $s_{70}$ gene to be within the S39 deletion (1E1-2;2B5-6) and outside the Sta deletion (1E1-2;2B3-4). This indicates that the $s_{70}$ gene resides within the region 2B3-4 to 2B5-6.

Report of I.D. Alexandrov and M.V. Alexandrova. Research Inst. of Medical Radiology, Obninsk, USSR.

Genetics and cytogenetics of the black mutations induced by gamma-rays, 252Cf and fission neutrons.

The following list is a part of our general stocklist described in DIS 61 and contains information on the 117 out of 142 transmissible black mutations which were scored in various experiments designed for estimating the relative proportion of intra- versus inter-genic changes at the locus of interest after action of the low- (mainly gamma-rays of 60Co) or high- (252Cf, fission neutrons) LET radiation on the different post-meiotic germ cells of the wild type or c(3)G males (see the last column list) pre-treated with radiomodifiers used (the sixth column). All other conditions of the experiments have been described elsewhere (Alexandrov et al. 1985; Alexandrov, Research Note, this issue). The black mutations were named (first column) by the accepted alphanumeric code. The second, third, fourth and fifth columns are respectively giving information on the phenotype, pattern of complementation with both su(b)31 and su(b)18 as well as cytology for each mutation.

As seen, 61 transmissible and viable in homozygote black mutations as well as 24 non-viable ones, but with separable lethal phenotype, i.e., so-called "twin" black mutants (Alexandrov, ibid.), have a normal 34D region of 2L chromosome (the putative location of the gene in question) and can be accepted as true gene mutations, VV8, which found to be suppressed by both su(b)31 and su(b)18, whereas 31 out of 117 mutations preserved proved to be associated with either chromosome alterations among which deficiencies shown to be predominant chromosome changes (20 out of 31 rearrangements studied) after irradiation of any genotype by neutrons and of c(3)G genotype by gamma-rays. On the contrary, in the wild-type genome gamma-photons induce predominantly exchange-type rearrangements. Also, the "twin" black mutants found to arise more frequent (18 out of 24) after action of photons than after neutron irradiation. The data of genetic mapping of the chromosome rearrangement breakpoints listed are reported elsewhere (Alexandrova, this issue).

Acknowledgement: We are grateful to Dr. Al. Sherald, George Mason University, Virginia USNA, for supplying the su(b)31 and su(b)18 stocks.


<table>
<thead>
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<th>Designation of mutation</th>
<th>Phenotype</th>
<th>Suppressed by su(b)31</th>
<th>Suppressed by su(b)18</th>
<th>Cytology</th>
<th>Modifier used, radiation dose</th>
<th>Genotype, germ cells irradiated</th>
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<td>D-32,MS</td>
</tr>
<tr>
<td>79b3</td>
<td>Lethal</td>
<td>DF(2L)34C7-D2;35A4±</td>
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<tr>
<td>79b7</td>
<td>bθ</td>
<td>+</td>
<td>+</td>
<td>Normal</td>
<td>&quot;</td>
<td>D-32,MS</td>
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<tr>
<td>79b8</td>
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<td>79d2</td>
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<tr>
<td>79d4</td>
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<tr>
<td>79d6</td>
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<td>T(2:3)34A2-3;34DB-E1.2;79B;80C</td>
<td>0.85 MeV n + γ-rays, 10 + 10 Gy</td>
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<tr>
<td>79d8</td>
<td>bθ as b50d</td>
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<td>79d10</td>
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<td>79d11</td>
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<td>83a</td>
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<td>81c</td>
<td>b</td>
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<tr>
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<td>83b1</td>
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<td>Y-rays, 40 Gy</td>
<td>D-32,MS</td>
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<td>83b40</td>
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An extreme allele of vestigial in D. melanogaster: vgX.

The vgX allele was extracted from a screen for X-linked lethals after gamma ray irradiation, using a Cs-137 source, of a highly inbred line of Oregon-R. The wings of homozygotes invariably appear as very small, veinless clubs; the halteres are reduced to tiny knobs or, in 80% of flies, are absent entirely on one or both sides; the postscutellar bristles are erect and, usually directed anteriad. These features, common to some other vestigial alleles, are accompanied by additional defects in the legs, abdominal tergites, and notum. Half or all of the notum is missing in 10% of flies, a consequence of failure of the dorsal mesothoracic disk to evert; one or more legs may be severely bent or truncated terminally with loss of tarsi, or both; and abdominal tergites may be deleted on one side, or severely misaligned (8.5%). The metathoracic legs are most often affected, with the tibia being bent posteriorly 90° or more; walking is difficult, and the defective leg trails uselessly. Females are more than twice as likely to show severe leg bending (45%) as males (20%); occasionally mesothoracic legs are affected.

Both males and females show good viability, but females are completely sterile; ovaries appear to be structurally normal, but no eggs are laid. The allele can be maintained in heterozygous condition, and fertile homozygous males are regularly produced. Although somewhat smaller and virtually wingless, males have been capable of mating with all females with which they have been tested. Many heterozygous combinations of vgX show mild to severe wing notching or scalloping.
Three mutants, $su(dp^{ov})_{TK8-84}$, $su(dp^{ov})_{TK25-84}$ and $su(dp^{ov})_{TK26-84}$, were induced by EMS. The suppressors were scored on $dp^{ov}$ males (Fig. 1) with a normalized wing phenotype (Fig. 2 compared with Fig. 3). Additionally, it turned out that $su(dp^{ov})_{TK8-84}$, but not $su(dp^{ov})_{TK25-84}$ or $su(dp^{ov})_{TK26-84}$, suppressed the vortex phenotype of $dp^{ov}$. However, none of the suppressors was able to suppress the vortex phenotype in a trans-configurated $dp^{ov}/dp^{ov}$. On the other hand, a trans-configurated $dp^{ov}/dp^{ov}$ was totally suppressed at the wing phenotype. Of the three suppressors, $su(dp^{ov})_{TK26-84}$ has the lowest penetrance since only about 70% of the flies are totally suppressed, while the two other suppressors have approximately 100% penetrance. The three suppressors seem to be alleles, since none of them complement each other. Viability and fertility of both $su(dp);dp^{ov}$ and $su(dp);+$ are good.
The new open wings mutant of *Drosophila melanogaster*.

In a sample of wild population of *D. melanogaster* from Petrinja locality (ca. 50 km southeast of Zagreb), cultured for several years in our laboratory, a phenotype with opened wings spontaneously appeared in 1983, indicating a new mutation. To determine the site of this mutation, we performed a genetic experiment with flies maintained nine generations by brother-sister mating. The crosses of our homozygous mutants with wild flies have shown that mutation is autosomal and recessive.

The crosses with brown-strain (bw 2-104.3) and scarlet-strain (st 3-440 showed that our mutant should be mapped on the third chromosome. In order to locate precisely the site of mutation, we applied the regular mapping procedure, by determining the frequency of crossing-over between the sepia and ebony markers (se 3-26; e 3-70.7). The obtained frequencies of recombinations (Table 1) suggested that the new mutation occurred at the locus 91.09 of the third chromosome. This is in the close neighborhood of well known locus crumpled (cmp 3-93), which has been supported by the appearance of cmp (up to 3%) on wings of the new phenotype.

**Table 1.** The frequencies of recombinations between jumper (ju) mutant vs sepia and ebony.

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<th>+ +</th>
<th>se e ju</th>
<th>+</th>
<th>se e ju</th>
<th>se + +</th>
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<td>68</td>
<td>26</td>
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<td>e</td>
<td>ju</td>
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<tr>
<td></td>
<td>47.85%</td>
<td>47.85%</td>
<td>31.76%</td>
<td></td>
<td>13.32%</td>
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<td>7.07%</td>
<td></td>
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</table>

Total flies recorded 976

26  70.7  \sim 91.09

\[38.83 \text{ cM} \quad 20.39 \text{ cM}\]

**Figure 1.** The phenotype of jumper mutant.

The main characteristics of the new phenotype is the presence of opened wings, oriented at the angle of 65° with respect to the longitudinal axis, and 10-30° to the horizontal axis (Figure 1). As of the other properties, the constant wing span and a decreased flying ability are so far the only recorded features, with a characteristic movement of individuals by jumping from one place to another. As far as we know (e.g., Zacharov 1979), this mutation is still not recorded in genetic maps of *D. melanogaster*.

We propose the following term to denotate this new mutation: jumper (ju 3-91.09).

**Acknowledgements:** The advices of Prof. D. Marinkovic and Asst. Z. Smit are very much appreciated.

Localization of terminal deficiency breakpoints on the X chromosome.

Figure 1. Positions of breakpoints for y deficiencies relative to nearby genetic markers. Names of genes are listed across the top. Most of these aberrations are true terminal deficiencies, although at least three (RT151, RT155, RT191 and possibly RT184) are half translocations.
Approximately 1500 putative terminal deficiencies with breaks between 1B1 and 1F were isolated by crossing irradiated +I/+;mu-2/mu-2 females to y w/y2 sc Y males (see Mason et al. 1984 for details). All y sc deficiencies were examined for their effects on y and ac, approximately 500 sc deficiencies were examined for complementation with lethals in this region, and 129 random deficiencies were examined cytologically. The genetic complementation studies are summarized in the figure. The cytological positions of the breakpoints are not given because, with the number of lethal complementation groups available, and the absence of stretching in this region, the cytology does not have the resolving power of the genetic tests. Suffice it to say that the positions of the breakpoints as determined cytologically correlate well with the positions determined genetically. The cytological results also confirm the earlier conclusion that most of these deficiencies extend to the tip of the X chromosome. About 8% are X-autosome translocations; the remainder are terminal deficiencies. Four translocations are included in the figure: [RT151=T(1;2R)1B7-10;57E10-F1. RT155=T(1;2L)1B9-12;21E4-F1. RT184 consistently ecotopically pairs with the tip of 3L, but no breakpoint is visible on chromosome 3. RT191=T(1;2L)1B4-10;21D1-2].

The apparent hot spots on the figure may not be real. The largest group of breakpoints lies between ac and sc, but the number of irradiated chromosomes screened to identify this sample is 2-3X larger than the number screened to identify breaks to the right of sc. The differences in the numbers of breakpoints in regions to the left of sc reflect differences in the physical distance between loci (Biessmann 1985, Campuzano et al. 1985). Apparent hot spots to the right of sc may be due to an incomplete genetic map as well as to differences in physical distance.


Dept. of Biology, Tokyo Metropolitan University, Tokyo, Japan.

Description of new mutants of Drosophila ananassae.

X chromosome


m84 : miniature84 Moriwaki 84e8. Spontaneous as two males of miniature wings accompanied with b ri-b, from a cross ? L bs/b ri-b x ?, b ri-b. Wings only slightly longer than abdomen and somewhat round. Expression variable, sometimes indistinct. Allelic to m.

w1 : white-ivory Moriwaki 83 x12. Spontaneous as a single male in a D15 stock. Eye color very light buff. Allelic to w. w1 > w.

w80 : white80 Moriwaki 80 g25. Spontaneous as a single male, white-eyed with D5-wing, from an inbred line of D5. Inseparable from w.

w84-2 : white84-2 Moriwaki 84Q 27. Two males with white and D5 L phenotype spontaneously appeared in a D71 b L/E IM4 stock. Allelic to w.

y74 : yellow74 Moriwaki 74h17. Spontaneously a female of yellow phenotype among progeny of a cross o ct y f w x o+ AM 1. Allelic to y.

ba-79 : balloon-79 Tobari 79b7. Spontaneously several flies arose in a M(2)b' stock. Wings inflated, blistered and a little extended. Penetration incomplete.


D5 : Delta5 Moriwaki 80d7. Spontaneously a male and a female appeared in a cross, ? L b/D5 x o m(12)K159. More extreme than D5. Reduced degree of expression depending on combined counterpart. Allelic to D5.

M(2)83 : Minute(2)83 Moriwaki 83f1. Spontaneous as a single male in a cross, ? D5 x o Pu+/ve- TBU. Minute bristles; dominant; homozygous lethal.

M(2)b' : Minute(2)b' Moriwaki 83x-84a. Arose spontaneously, in the M(3)b67 stock. M(3)b67 allele had been lost coincidently.

pr : purple Moriwaki 77k12. Spontaneous as two females in a wild strain, PT-2, from Papua, New Guinea, in 1977. Eye color purplished ruby.

ri-b : radius incompletus-b Moriwaki 75d6. Spontaneously arose in a wild strain, A36, from Taiwan, in 1971. Vein L2 incomplete. Non allelic to ri. The name of ri, which was reported as radius interruptus in DIS 46 (1971) and in "Drosophila ananassae" (Handbook of Genetics, vol. 3, 1975), is revised, reading incompletus for interruptus.


3rd chromosome

briV : brightV Moriwaki 81k11. Spontaneous as a male in a wild strain, VAV151, collected at Vavau, Tonga, in 1981. Eye color bright red; ocelli colorless. Allelic to bri.

c : curved Tobari 82b23. Spontaneously arose in a wild strain, MYS178, collected at Mysore, India, in 1981. Wings curved downward throughout the length. Mixed with ones of curved and spread wings, which, by selection, were fixed as csp (curved spread).

cn : cinnabar Moriwaki 84k5. Spontaneous as male and two females in a D1 stock. Eye color bright red, becoming dull with age.

Cy1 : Curlyoid Moriwaki 84k27. Spontaneously appeared in a cn stock. Wings curled up. Dominant; homozygous lethal. Dominancy incomplete, overlapping wild type occasionally.

dke : dark-eye Moriwaki 82a25. Spontaneous two males and one female in a wild strain, NAN80, collected in Fiji, in 1981. Eye color dark red.

dp : dumpy Moriwaki 79k3. Spontaneous as three males and two females in a wild strain, B13, collected in Thailand, in 1979. Wings obliquely truncated and reduced to 2/3 normal length; expression variable, often wings not shortened, narrow and somewhat pointed.

M(3)e : Minute(3)e Moriwaki 82j15. Spontaneously a male and two females arose in a cross, + AABBG1 x σ ml ru. Minute bristles; dominant; homozygous lethal. Expression variable. Likely near M(3)d or an allele of the M(3)d, but undetermined.

ml : minute-like Tobari 78fl. Spontaneously arose in a M(3)d ru stock. The locus seems likely near M(3)d or an allele of the M(3)d, but undetermined.


pm : plum Moriwaki 81k18. Spontaneously two males and one female appeared in a wild strain, TBU130, collected in Tongatapu, in 1981. Eye color brownish wine, darkening to garnet with age. Combination of pm bri gives colorless eye and pm cn showing diluted orange color.

pxd : plexusd Moriwaki 77k12. Spontaneous as a female arose in a wild strain, 25, collected in Papua, New Guinea, in 1977. Extra venation slightly near the tip of marginal cell; only dot-like at times. Allelic to px.


px4 : plexus4 Moriwaki 83h8. In a M(3)d dke stock, spontaneously appeared as many flies. Venation plexus, extra vein mostly in submarginal cell and marginal cell. Allelic to px.


4th chromosome

bbHYD : bobbedHYD Moriwaki 84e21. Spontaneously several females with bobbed bristles appeared in a spa82 stock. spa82 bbHYD stock was fixed. Allelic to bb.

spa : sparkling Moriwaki 79k5. Spontaneous as a female and ten males in a wild strain, D6, collected in India, in 1979. Eye surface rough and mottled in varying degrees. A linkage relation in spa-bb (4th chromosome) was confirmed. A similar mutant mo (mottled), found before the War(36k24), though discarded, also located in 4th chromosome (Moriwaki 1938).


Report of C. Najera. Dept. of Genetics, Faculty of Biology, University of Valencia, Spain.

List of the different eye colour mutants of Drosophila melanogaster obtained in two different captures carried out in a pine-wood from La Canada, Valencia (Spain).

Localized mutants

se80i (sepia-80)
cg80i (cardinal-80) - two alleles at same capture
cg80d (cardinal-80) - two alleles at same capture
sr80i (safranin-80) - four alleles at same capture
sr80d (safranin-80) - four alleles at same capture
sed80d (sepiaoid-80)
v80d (vermilion-80)
p80d (purple-80)

Non-localized (cont.)

70,81. eye colour bright brown
71. eye colour bright brown darkening with age
72. eye colour bright red
73. eye colour bright garnet darkening with age
74,91. eye colour bright red
75. eye colour vermilion
76,84. eye colour dark wine
77. eye colour reddish brown
78. eye colour coppery red
79. eye colour reddish brown darkening with age

(two alleles)

65,80. eye colour chestnut
66. eye colour light chocolate
67,83. eye colour dark red
68,82. eye colour like wild darkening with age
69. eye colour dull red darkening with age
83. eye colour dark chocolate
86. eye colour ruby
87. eye colour purple
88. eye colour orange red darkening with age
89. eye colour orange red
90. eye colour shining red

Report of Yong K. Paik. Dept. of Genetics, Hanyang University, School of Medicine, Seoul 133, Korea.

New inversions of Korean D.melanogaster recovered from natural populations. All inversions are maintained as balanced stocks.

<table>
<thead>
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<th>Inversion</th>
<th>Breakpoints</th>
<th>Inversion</th>
<th>Breakpoints</th>
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<td>22A;26B</td>
<td>In(3R)KA</td>
<td>82E/F;99E/F</td>
</tr>
<tr>
<td>In(2L)KB</td>
<td>22D(2R);57A</td>
<td>In(3R)KB</td>
<td>83D/E;86D</td>
</tr>
<tr>
<td>In(2L)KC</td>
<td>24C/D;36B/C</td>
<td>In(3R)KC</td>
<td>83C/D;93A</td>
</tr>
<tr>
<td>In(2L)KD</td>
<td>26B/29B/C</td>
<td>In(3R)KD</td>
<td>84E;94D/E</td>
</tr>
<tr>
<td>In(2L)KE</td>
<td>26C/29B/C</td>
<td>In(3R)KE</td>
<td>85C;88F</td>
</tr>
<tr>
<td>In(2L)KF</td>
<td>26C/D;32B/C</td>
<td>In(3R)KF</td>
<td>86B/87B/C</td>
</tr>
<tr>
<td>In(2L)KG</td>
<td>28A/B;32D</td>
<td>In(3R)KG</td>
<td>86D/E;97C</td>
</tr>
<tr>
<td>In(2R)KA</td>
<td>42A;57F</td>
<td>In(3R)KH</td>
<td>88C;96D</td>
</tr>
<tr>
<td>In(2R)KB</td>
<td>42A;58C/D</td>
<td>In(3R)KI</td>
<td>88D/94A</td>
</tr>
<tr>
<td>In(3L)KA</td>
<td>66C;71B/C</td>
<td>In(3R)KJ</td>
<td>90D;93B</td>
</tr>
<tr>
<td>In(3L)KB</td>
<td>68C;91D</td>
<td>In(3R)KK</td>
<td>92E;97C</td>
</tr>
</tbody>
</table>
Description, localization and recombination values of several mutants of *Drosophila buzzatii*.

### Table 1. Recombination values between some of the mutants of *Drosophila buzzatii*.

<table>
<thead>
<tr>
<th>Chromosome markers*</th>
<th>Cross-over size</th>
<th>Sample recombination value</th>
</tr>
</thead>
<tbody>
<tr>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fum - N</td>
<td>68</td>
<td>0.2352 ± 0.0059</td>
</tr>
<tr>
<td>Fum - w</td>
<td>94</td>
<td>0.3868 ± 0.0121</td>
</tr>
<tr>
<td>Fum - y</td>
<td>22</td>
<td>0.1176 ± 0.0028</td>
</tr>
<tr>
<td>Fum - v</td>
<td>68</td>
<td>0.3333 ± 0.0110</td>
</tr>
<tr>
<td>N - w</td>
<td>68</td>
<td>0.0280 ± 0.0009</td>
</tr>
<tr>
<td>N - y</td>
<td>152</td>
<td>0.1565 ± 0.0018</td>
</tr>
<tr>
<td>N - v</td>
<td>145</td>
<td>0.1460 ± 0.0016</td>
</tr>
<tr>
<td>w - y</td>
<td>204</td>
<td>0.2020 ± 0.0026</td>
</tr>
<tr>
<td>w - v</td>
<td>93</td>
<td>0.1032 ± 0.0101</td>
</tr>
<tr>
<td>y - v</td>
<td>152</td>
<td>0.2934 ± 0.0059</td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Est - mah</td>
<td>9</td>
<td>0.0700 ± 0.0016</td>
</tr>
<tr>
<td>Pept-2 - se</td>
<td>7</td>
<td>0.1129 ± 0.0045</td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adh - fo</td>
<td>103</td>
<td>0.3355 ± 0.0090</td>
</tr>
<tr>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pgm - 4s</td>
<td>10</td>
<td>0.1562 ± 0.0071</td>
</tr>
<tr>
<td>Pgm - st</td>
<td>17</td>
<td>0.0934 ± 0.0020</td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>bw - vs</td>
<td>275</td>
<td>0.5198</td>
</tr>
</tbody>
</table>

* Fum, -Est, Pgm and Adh stands for Fumarase, -Esterase, Phosphoglucomutase and Alcohol dehydrogenase. 4s refers to inversion breakpoints of chromosome 4.

The biological interest of *D. buzzatii* as a species to perform evolutionary studies has been emphasized several times (Fontdevila 1982). However, design of genetic experiments with this species has been hampered in many cases by the lack of appropriate morphological and biochemical markers. We report here the first list of morphological mutants of *D. buzzatii* obtained from natural populations and laboratory strains. Initially, chromosomal rearrangements, known to be located in specific chromosomes (Pla et al. 1984; Ruiz et al. 1985). Mutant naming and morphological description analogous to those of *D. melanogaster* have been adopted only when homologies are well established (Stone 1955; Zouros 1976). In other cases, appropriate names have been adopted. Genetic naming follows that one by Lindsley & Grell (1968).

### Description of mutants


- **ey**: *eyeless*. Eye more reduced to normal area. Recessive. Could be homologous to eyeless of *D. melanogaster*.

### Mutant names

- **D.m.** (chromosome 4). Originated from a natural population from Mazarron (Murcia, Spain).

- **fo**: *folded*. Wings remain unexpanded. Recessive. Located in chromosome 3. Induced by X-ray in a laboratory stock.

- **mah**: *mahogany*. Eye color brown (or maroon) and darkening with age. Recessive. Located in chromosome 2. Probably homologous to mahogany of *D. melanogaster* (chromosome 3R). Originated from a natural population from Sitges (Barcelona, Spain).

- **N**: *Notch*. Wings incised at tips and often along edges. Terminal part of vein L5 often opened. Variable expression. Dominant. Located in chromosome X. Male and homozygous female lethal. Probably homologous to Notch of *D. melanogaster* but we have not detected any deficiency. Induced by X-ray in a laboratory stock.


- **v**: *vermilion*. Eye color bright red. Recessive. Located in chromosome X. Probably homologous to vermilion of *D. melanogaster* (chromosome X). Originated from a natural population from Sitges (Barcelona, Spain). With sepia produces orange eyes.

- **vs**: *vesiculated*. Wings warped, wrinkled, blistered and rough texture. Variable expression. Penetration of 90%. Recessive. Located in chromosome 5. Induced by X-ray in a laboratory stock.

- **w**: *white*. Eye color white. Recessive. Located in chromosome X. Probably homologous to white of *D. melanogaster* (chromosome X). Originated spontaneously from a laboratory stock.

- **y**: *yellow*. Body color yellow. Recessive. Located in chromosome X. Probably homologous to yellow of *D. melanogaster* (chromosome X). Originated spontaneously from a laboratory stock.

### References

Department of Genetics, University of California, Davis, Calif. 95616

We here report the identification of a new locus in *D. melanogaster*. The recessive phenotype is identical with that of "eagle", e.g., 3-47.3, but is linked to chromosome 2 mapping 26.5 ± 0.9 centimorgans to the left of B1, and so is located at position 28.3 ± 0.9. We propose the name "nesher" (Hebrew for "eagle"), symbol "nr". Nesher and eagle combined exhibit the eagle phenotype. Nesher was isolated from 8 independent isofemale lines derived from two natural populations; one Apple Hill in northern California and the other Furnace Creek in Death Valley, California. Nesher occurred among the much more numerous independent appearances of eagles found by J. Bundgaard in isofemale lines from the above localities as well as several others in California.

Biological Sciences, University of Sydney, Australia.

Eyes Absent (eya)

This mutation was uncovered several years ago during screening of a winery population using chromosome homozygosis for chromosomes II via the Cy/Pm technique. Attempts to map the mutation were unsuccessful, and it was found to be associated with an inversion with breakpoints at 22D and 34B.

The phenotype is characterised by complete absence of eye facets. Other aspects of head development appear normal, although the head size is considerably reduced, and the pattern of bristles surrounding the residual eye socket is variable. The mutation is fully recessive. The viability in pure culture is high, although reduced when measured in competitive viability tests.
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Davies, J PhD Visiting Fellow molecular developmental neurobiology
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Higginbotham, J Curator of Stocks
Gray, B Molecular technical assistant
Dept. of Neurobiology: De Couet, HG PhD Res Fellow vision, muscle struct & function, immunology, mol neurobiol.

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Ghysen, A lecturer, research associate
Deceased: Ms Altorfer
Retired: Prof. Brachet

COLOMBIA: Bogota: Universidad de Los Andes, Inst. de Genetica Tel: 2824066 x 129 [REPLACE OLD LISTING]
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Rodriguez, J del C PhD theoretical (mathematical) population genetics and biophysics
de Polanco, MME MSC cytotgenetics of Drosophila pseudobscura of the cundiboyacense altiplano from Columbia
Ordoñez, M MSC electrophor analysis in natural pop of D.pseudoobscura of cundiboyacense altiplano from Columbia
Montaño, D4 Misc electrophoretic analysis in natural populations of D.reperta group in South America
Delalcáz V., MHC quantitative genetics of diateclic analysis and measurement of distances in natural populations of D.pseudoobscura of the cundiboyacense altiplano from Colombia
Cárdenas, H MSC multivariate anal in natural pop of D.pseudoobscura of cundiboyacense altiplano (Colombia)
Riveros, A3 MSC mutation rate in natural pop of D.pseudoobscura of cundiboyacense altiplano (Colombia)
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Calcagno, AM MSC evolutionary biology of cactaceas family in South America
de Romero, IF technician competition problems
Bustos Parra, E tech cytogenetics of IIIrd chromosome of D.pseudoobscura & D.reperta group (S America)
Ordoñez, M L Misc technician competition problems
Laguna, M A tech multivariate anal in natural pop of D.pseudoobscura of cundiboyacense altiplano (Colombia)
Niño, J I tech cytogenetics of IIIrd chrom of D.pseudoobscura of cundiboyacense altiplano (Colombia)
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Rodriguez, R D technician curator preparing cultures

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Ish-Horowicz, D senior scientific staff molecular developmental genetics of pattern formation
Pinchin, S M senior research officer molecular developmental genetics of pattern formation
Taylor, A M molecular biology of development

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White, D C S DPhil lecturer mechanical studies of mutant flight muscle 0904-430000

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Dey, L Senior Res Fellow genotoxicology Patnaik, K Junior Res Fellow chemical mutagen.
Majhi, B Senior Res Fellow chemical mutagenesis Tripathy, N K Reader chemical mutagen.

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Chatterjee, S MSc SRF behaviour genetics
Kumar, A MSc Res Fellow cytogenetics & biochem genetics
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Singh, A K MSc Res Fellow crossing over
Singh, B N PhD Reader pop & behav genetics

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Fuyama, Y Dr Instr isolation mechanisms & parthenogenesis
Goni, B MSc grad student cytogenetics
Kaneko, A Dr Guest Investigator ecology & taxonomy
Kim, B K MSc grad stu mtDNA polymorphisms
Kitagawa, O Dr Prof evol & behav genetics
Matasabayashi, H grad stu cytogenetics & mutation
Matsuda, M Dr Instr cytogenetics of male recomb
Moriwaki, D Dr Prof Emer genetics of male recomb mating behavior
Oba, S Dr Prof enzyme & mtDNA polymorphisms, longevity & aging

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Han, Y J Instructor behavior genetics
Huh, M K Instructor population genetics
Ju, G J Research Asst population genetics
Kim, N W Research Asst population genetics
Lee, T J Dr Prof population genetics, Drosophila taxonomy
Suh, M S Research Asst Curator of stocks

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Cho(Shin), I S MS Res Asst milk phosphoglucomutase
Chung, Y J PhD Prof population genetics
Kang(Song) S J PhD Assoc Prof amylose polymorphism
Kim, E Y grad student electrophoresis
Kim, M S grad student cytogenetics
Kim, S S grad student animal genetics
Lee(Ahn), M S MS Instructor cytogenetics
Lee(Lee), Y R MS Instructor electrophoresis
Park, M K grad student cytogenetics
Park, S BS technician
Shin, S M MSc Res Asst tissue & cell culture

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Kim, Y MD grad stu in vitro cell culture, biochem genetics
Lyu, M MD Instr cytogenetics, biochem genetics
Paik, Y PhD Prof Head of Dept pop & evol genetics
Park, H S MS grad stu cytogenetics

KOREA: Suweon 170: Sung Kyun Kwan University, Biology Dept., Genetics Lab Tel (0331) 44-2161 x 306.
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Kim, D I MSc grad student chromosomal polymorphism
Kim, S J BS grad student hybrid dysgenesis
Kim, W S MSc grad student enzyme polymorphism
Kim, Y S MSc grad student enzyme polymorphism
Kwon, Y S MS grad student enzyme polymorphism

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Kamping, A Res Asst pop genetics

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Frayne, E Postdoc Fellow mol genetics, caudal O’Kane, C Postdoc Fellow devel genetics,
Gehring, W J Prof developmental genetics transposable elements
Hironi, Y Res Assoc devel genetics, homeotic genes Pick, L Postdoc Fellow devel genetics,
Kloter, U Curator of stocks transcription
Krause, H Postdoc Fellow mol genetics, DNA Walldorf, U Postdoc Fellow devel genetics,
LeMotte, P Res Assoc molecular genetics, Scr binding of ftz protein
Mlodzik, M grad stu molecular genetics, caudal Wirz, J grad stu mol genetics, homeotic genes
Muller, M grad stu devel genetics, ftz

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Finnerty, V M PhD AssocProf gene expression -4201
Langley, S D MS research specialist gene expression -4201
Warner, C K PhD research assoc gene expression -4231

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Lande, R S PhD Assoc Prof quantitative genetics -1078
Lindquist, S PhD Assoc Prof heat shock proteins -8049
Potter, R V PhD Lect virilis group ADH locus -8034
Sporoff, J B PhD Assoc Prof position effect, duplications -8941
Swift, H S PhD Prof cytogenetics, molecular genetics -8041
Throckmorton, L H PhD Prof Dros phylogeny, systematics, biogeography, reprod biology & evolution -8945
VanValen, L PhD Prof evolution -9475

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Dotson, R L MS res asst mutagenesis -2006 Reardon, J T MS grad stu DNA repair -2385
Dusenberg, R L PhD Asst Prof DNA repair -2729 Smith, P D PhD Prof genetics -2231
Golden, C A L PhD res assoc mol genetics -2385

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Held, L Postdoctoral scholar -6997 Sponaugle, D grad student -6997
Langelaan, R Postdoctoral scholar -6997 Jo Wen Wu grad student -6997
Woods, D Postdoctoral Scholar -6997 Hsei, B research assoc -6997
Jursnich, V grad student -6997 Wilson, L research assoc -6997

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delete: Sederoff, R R

USNA: St. Paul, Minnesota 55112: Bethel College, Biology Dept, 3900 Bethel Drive Tel 612-638-6400
Jones, C W PhD structure & function of ecdysone-responsive genes in Drosophila [NEW LISTING]

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add: Sederoff, R R PhD molecular genetics

USNA: Stony Brook, New York 11794: State Univ of New York, Biochemistry Dept Tel 516-245-5043 [UPDATE]
add: Bingham, P M PhD physiological genetics, transposons Erk, F C PhD developmental genetics
Carlson, E A PhD mutagenesis, mosaicism

NOTE: The Alphabetical Directory is being omitted in this issue; it will appear in volume 65 (1987).
THE MOLECULAR GENOME OF DROSOPHILA MELANOGASTER

CATALOGS OF CLONED DNA, BREAKPOINTS AND TRANSFORMED INSERTS
BY CHROMOSOME LOCATION

John Merriam, Susan L. Smalley, Andrew Merriam and Bronwyn Dawson
Department of Biology, University of California
Los Angeles, CA 90024

The force of Drosophila cytogenetics has been strong in identifying specific genes as mutant alleles. With the advent of molecular cloning and in situ hybridization, this cytogenetic approach can be extended, so far uniquely to Drosophila, to cloning genes, or identifying cloned genes, starting from chromosome locations. To do so via chromosome walks, chromosome jumps or transposon tagging requires preexisting clones or breakpoints or other genes inserted by transformation in a location. Sources of knowledge of these preexisting starting points are critical for experimental design. The community of Drosophila workers is cooperative about sharing this information. Beginning in 1983 with the National Annual meeting and with extensions to the international community we have sought to collect and summarize what is known about the molecular genome of Drosophila. As these reports in the D.I.S. have grown, the format of presenting the information has also changed. The present report consists of four tables: MASTER LIST, TABLE 1, TABLE 2, and REFERENCES. A suggested report form is appended to copy in order to send in more recent additions.

MASTER LIST contains data by chromosome location with the following information listed:

<table>
<thead>
<tr>
<th>LABEL</th>
<th>DESCRIPTION OF INFORMATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>CLONE NAME</td>
<td>Name or number of cloned sequence</td>
</tr>
<tr>
<td>GENES</td>
<td>Genes contained in cloned sequence</td>
</tr>
<tr>
<td>BREAKPOINT</td>
<td>Name of rearrangement with breakpoint(s) in cloned region</td>
</tr>
<tr>
<td>CROSS LIST</td>
<td>Cross listing of other breakpoints of rearrangement</td>
</tr>
<tr>
<td>INSERTION</td>
<td>Transformants inserted at this location</td>
</tr>
<tr>
<td>CROSS LIST</td>
<td>Cross listing of chromosome location of genes contained in the insert</td>
</tr>
<tr>
<td>REF</td>
<td>Literature citation for information on clones, breakpoints, and inserts.</td>
</tr>
</tbody>
</table>

TABLE 1 contains other information on clones listed by chromosome location. Information on stock of origin, K8 size, DNA library, reference to mapping sequence, and miscellaneous information is provided when available under the heading OTHER INFORMATION.

TABLE 2 contains information on inserts listed in MASTER LIST. Inserts are listed by chromosome location with gene markers contained in the inserts listed under the heading MARKERS.

REFERENCES are listed in the last table by reference number used in the MASTER LIST.

This work was supported by NIH Grant LM04344 from the National Library of Medicine.
1A

CLONE NAME: unn
GENES: yellow
BREAKPOINT: In(1)y4
CROSS LIST: 18A3, 4

1A8B1

CLONE NAME: unn
GENES: yellow
BREAKPOINT: In(1)y4
CROSS LIST: 18A3, 4

1A

CLONE NAME: lambda T-A
GENES: telomeres
CROSS LIST: 21A, 60F, 61A, 100F, 102F

1B

INSERTION: 27X-F
CROSS LIST: 67B, 87D12
CLONE NAME: unn
GENES: scute
CLONE NAME: lambda y1
GENES: yellow
CLONE NAME: y+p13-2, yp3A2
GENES: yellow
CLONE NAME: adm134E8
GENES:
BREAKPOINT: In(1)sc29
CROSS LIST: 13A2-5
INSERTION: g71:1
CROSS LIST: 68C3-5, 87D12

1B1, 2

CLONE NAME: unn
GENES: yellow, achete
BREAKPOINT: In(1)y3p
CROSS LIST: 20D1

1B1, 2-B4, 5

CLONE NAME: sc64-sc133
GENES: y, ac, sc-alpha and sc-beta

1B2, 3

CLONE NAME: unn
GENES: achete, scute
BREAKPOINT: In(1)ac3, In(1)sc260-22, In(1)sc8, In(1)sc9
In(1)sc260-14
CROSS LIST: 1B14C1, 1E2, 3, 20D1, 18B8, 9, 11D3, 8

1B3, 4

CLONE NAME: unn
GENES: scute
BREAKPOINT: In(1)sc4, In(1)scL8, In(1)scL1
CROSS LIST: 20C1, 20C, 20D1

1B4, 5

CLONE NAME: unn
GENES: scute
BREAKPOINT: T(1;3)scKA8, T(1;3)sc260-15
CROSS LIST: 98 (+ OR -), 71CD
1B4,6
CLONE NAME: unn
GENES: scute
BREAKPOINT: In(1)sc7
CROSS LIST: 5D3,6
REF: 105

1B4,7
CLONE NAME: unn
GENES: scute
BREAKPOINT: T(1;2)sc19, T(1;2)scS2
CROSS LIST: 25A, 60C1,3
REF: 105

1B4-C6
CLONE NAME: unn
GENES: scute
BREAKPOINT: T(1;4)scH
CROSS LIST: 101-102
REF: 105

1B4,5-1B8,9
CLONE NAME: elav
GENES:
BREAKPOINT: In(1)63
CROSS LIST: 2E
REF: 206

1B5,8
CLONE NAME: cos 4P
GENES:
CROSS LIST: 2E
REF: 57

1B11-13
CLONE NAME: unn
GENES: suppressor of sable
REF: 15

1B1,2-4,5
CLONE NAME: unn
GENES: yellow, achete, scute
REF: 57

1B14C1
BREAKPOINT: In(1)ac3
CROSS LIST: 1B2,3
REF: 105

1CD
INSERTION: Bs2.71-2
CROSS LIST: 66D11-15, 87D12
REF: 115

1E 2-3
INSERTION: Strain 23-2
CROSS LIST: 87D12, 5S ribosomal genes
REF:

1E2,3
BREAKPOINT: In(1)sc260-22
CROSS LIST: 1B2,3
REF: 105

1F
BREAKPOINT: T(1;3)Uab5
CROSS LIST: 89E1-4
INSERTION: R704.2, R702.1
CROSS LIST: 87D12
INSERTION: DA24-14
CROSS LIST: 35B2-3, 37C1,2
REF: 110, 115, 114
2A
INSERTION:  P[(w,ry)E]5
CROSS LIST:  3C1,2; 87D
INSERTION:  27P X/X-F
CROSS LIST:  67B, 87D12

2B
INSERTION:  P(ry,hsp0-1)8
CROSS LIST:  87D12
INSERTION:  Tf(1)Gr304-1
CROSS LIST:  87D12

2B1,2
CLONE NAME:  unn
GENES:  1(1)BA11
BREAKPOINT:  Dp(1;f)101
CROSS LIST:  20

2B1,2-5,6
CLONE NAME:  unn
GENES:  occ

2B1,2
CLONE NAME:  Dm156, Dm159, Dm160
GENES:

2B3,4
CLONE NAME:  unn
GENES:  sta
BREAKPOINT:  T(1;3)sta
CROSS LIST:  89B21
CLONE NAME:  unn
GENES:  sta
BREAKPOINT:  T(1;3)sta
CROSS LIST:  89B21
CLONE NAME:  Dm174, Dm 340
GENES:

2B13-18
INSERTION:  g711:2
CROSS LIST:  68C3-5,87D12

2C
INSERTION:  Pc[ry(delta0-1)]48
CROSS LIST:  87D12

2C1-2E3
CLONE NAME:
GENES:  vsp, csw, ph, Pgd, kz, 11 lethals

2D2-3
BREAKPOINT:  Df(1)pu3; Df(1)Pgd-kz;Df(1)JA52;Dp(1)dorY18T

2EF
CLONE NAME:  unn
GENES:  fs(1)pecanex,pcxl

2E
CLONE NAME:  cos 4P
GENES:
CROSS LIST:  1B5,8
2E2-F3
CLONE NAME: unn
GENES: fs(1)K10, crooked neck, pcx, kurz
REF: 98

2F5-6
CLONE NAME: draf1
GENES: raf oncog. homo.; pos. ser/thr kinase
CROSS LIST: minor homology to 43A2-5
REF: 129

3
CLONE NAME: S24
GENES:
REF: 16

3A
INSERTION: 7-1;7-2
CROSS LIST: 3C11,12;87D; 3C11,12;ry+
REF: 140

3A1-4
CLONE NAME:
GENES: zeste
BREAKPOINT: In(1)e(bx); Df(1)64C4; Df(1)w258-11; Df(1)wrj
REF: 166
CROSS LIST: 4F

3A6-8
BREAKPOINT: Df(1)64j4
CROSS LIST: 3C1-4
REF: 107

3B
CLONE NAME: lambda DT2,5
GENES:
REF: 217
CLONE NAME: mDm112 C 10
GENES:
INSERION: Tf(1)GR420-3
REF: 111
CROSS LIST: 87D12

3B1,2
CLONE NAME:
GENES: per, L(1)BA11
BREAKPOINT: Df(1)w~64D, In(1)3B1, 2-20F, Df(1)3B1,2-3C2,3
Dp(1;f)101, Df(1)62d18, T(1;4)JC43
REF: 107
CROSS LIST: 20F, 20, 3C6, 102
CLONE NAME: unn
GENES: per
REF: 9

3B2-3C2
CLONE NAME: unn
GENES: white
REF: 62

3BC
CLONE NAME: M187
GENES:
REF: 84

3C
CLONE NAME: unn
GENES: distal to white
REF: 95

3C1-4
BREAKPOINT: Df(1)64j4
CROSS LIST: 3A6-8
REF: 107
3C1,2
CLONE NAME: lambda ml.2
GENES: white
BREAKPOINT: Df(1)wNfm20
CROSS LIST: 3C11-12

3C2-3
BREAKPOINT: Df(1)Nfm21, Df(1)N10
CROSS LIST: 3C11-12

3C6-8
CLONE NAME: unn
GENES: Notch
BREAKPOINT: Df(1)62d18
CROSS LIST: 3B1,2

3C6
BREAKPOINT: Df(1)N541g
CROSS LIST: 3C11-12

3C7,8
CLONE NAME: unn
GENES: Notch
BREAKPOINT: In(1)N76b8, Df(1)N62b1
CROSS LIST: 3C9,10, 3D5,6

3C7
CLONE NAME: pKdm 6B3
GENES: intermolt I RNA
CROSS LIST: 3D1

3C9,10
BREAKPOINT: In(1)N76b8
CROSS LIST: 3C7,8

3C11,12
CLONE NAME: unn
GENES: Sgs 4
BREAKPOINT: Df(1)wNfm20, Df(1)Nfm21, Df(1)N10, Df(1)N541g
CROSS LIST: 3C1-2, 3C2-3, 3C6, 3E4

3C11
CLONE NAME: pOM3, pOW3
GENES: Sgs-4
CROSS LIST: pSME3, pSWE3
CLONE NAME: pOM3,pOW3
GENES: Sgs-4
CROSS LIST: pSME3, pSWE3
3D1
CLONE NAME: pKdm 6B3
GENES: intermolt I RNA
CROSS LIST: 3C7

3D5,6
BREAKPOINT: Df(1)N62b1
CROSS LIST: 3C7,8

3E4
BREAKPOINT: Df(1)dm75elg
CROSS LIST: 3C11,12

3F
INSERTION: g71dx:2
CROSS LIST: 68C3-5,87D12

3,4
CLONE NAME: adm 136G5
GENES:

4C5-6
INSERTION: Strain 23-2
CROSS LIST: 87D12, 5S ribosomal genes

4B
INSERTION: chB delta -59
CROSS LIST: 87, lacZ, 87D12

4BC
CLONE NAME: mDm 109A7
GENES:

4C
CLONE NAME: M97
GENES:

4D
BREAKPOINT: T(1;3)bxd111
CROSS LIST: 89E1,4
INSERTION: R405.1
CROSS LIST: 87D12

4F5A
CLONE NAME: pKdm 35D12
GENES: late IV RNA
CLONE NAME: adm 139C12
GENES:

5AB
CLONE NAME: adm 126D6
GENES:

5B
BREAKPOINT: T(1;3)Ubx21560.8A complex
CROSS LIST: 89E1,4
5C
CLONE NAME: pDmA z
GENES: actin
CLONE NAME: unn
GENES:

5D
CLONE NAME: unn
GENES: ribosomal protein 7/8

5D3-6
BREAKPOINT: In(1)sc7
CROSS LIST: 1B4,6

5EF
CLONE NAME: adm 140C11
GENES:
CROSS LIST: 63F64A

5F (prox)
CLONE NAME: B70
GENES: maternal restricted transcript

6AB
INSERTION: P[(w,ry)H]4
CROSS LIST: 3C1,2; 87D

6F
INSERTION: S6.9-2
CROSS LIST: 66D11-15, lacZ, 87D12

6F5
CLONE NAME: unn
GENES: sex lethal
CLONE NAME: S1, S2A, S2B
GENES: Sex-lethal (sxl)
BREAKPOINT: In(1)sxl-af; Df(1)sxl-ra
REF: 223

7A
CLONE NAME: unn
GENES:

7B3,4
CLONE NAME: unn
GENES: cut

7D
CLONE NAME: m58
GENES:
INSERTION: R403.1
CROSS LIST: 87D12

7D5
CLONE NAME: unn
GENES: fs(1)homeotic, l(1)myospheroid

7D5-6
CLONE NAME:
GENES: fs(1) homeotic; l(1) B104 leth. myospher.
BREAKPOINT: T(1;3) N72
CROSS LIST: 7D1,2
7E6
CLONE NAME: unn
GENES: CROSS LIST: 7F1,2

7E11-7F1,2
CLONE NAME: unn
GENES: s36, s38
CROSS LIST: see Table 2

7F1,2
CROSS LIST: 7E6

7F1
CLONE NAME: CROSS LIST: 7E6
GENES: ovarian tumor (otu)

7,8
CLONE NAME: adm 132H10
GENES:

8
CLONE NAME: 56
GENES:

8A
CLONE NAME:unn
GENES:

8BC
INSERTION: tAP-25,3.2
CROSS LIST: 35B2-3

8D
CLONE NAME: PLZ-p
GENES: lozenge

8E
INSERTION: cHB delta-59
CROSS LIST: 87,lacZ,87D12

8F9A
CLONE NAME: PYP2
GENES: yolk protein 2
CLONE NAME: PYP1
GENES: Yolk protein 1

9E3-4
CLONE NAME: lambda EMBL4-LF1
GENES: raspberry, P-element

9A-D
INSERTION: R404.2
CROSS LIST: 87D12

9B
INSERTION: unn
CROSS LIST: 87D12
INSERTION: tAP-24B,3.2
CROSS LIST: 35B2-3
INSERTION: SB2.1-5
CROSS LIST: 66D11-15,87D12
9C
INSERTION: S6.9-9
CROSS LIST: 66D11-15, lacZ, 87D12
REF: 115

9D
INSERTION: P(ry,hsp0-1)22
CROSS LIST: 87D12
REF: 137

9E
INSERTION: R701.1
CROSS LIST: 87D12
REF: 115

INSERTION: cHB lambda -23
CROSS LIST: 87-lacZ, 87D12
REF: 118

10A
CLONE NAME: unn
GENES: vermilion: tryptophan oxygenase
REF: 79

10A1
CLONE NAME:
GENES: vermilion
REF: 186

10A1,2
CLONE NAME: lambda vDT1
GENES: vermilion (tryp oxygenase)
REF: 217

10A2
CLONE NAME: sevenless
GENES: photoreceptor cell development
REF: 173

10B
CLONE NAME: unn
GENES:
REF: 91

10BC
INSERTION: tAP-20,3.2
CROSS LIST: 35B2-3
REF: 112

10C1,2
CLONE NAME: lambda DmRpl11-1
GENES: RNA polymerase II largest subunit
BREAKPOINT: Df(1) GA112; Df(1) HA85; M259-4
CROSS LIST: 10B1,2; 11A1,2; 10E1,2
REF: 147

10D
INSERTION: AR4-038
CROSS LIST: 3C1,2
REF: 126

10EF
CLONE NAME: adm134A3, adm130E12
GENES: late V RNA
REF: 34

10F
CLONE NAME: adm 10F.1
GENES: minor heat shock cDNA from Kc cells
REF: 39

11A
CLONE NAME: unn
GENES: gastrulation defective
CLONE NAME: lambda Dm (Can 6) LSP1 alpha: 1
GENES: LSP1 alpha
REF: 130
<table>
<thead>
<tr>
<th>Clone Name</th>
<th>Genes</th>
<th>Reference</th>
</tr>
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<tbody>
<tr>
<td>11A2-4</td>
<td>CLONE NAME:</td>
<td>REF: 174</td>
</tr>
<tr>
<td>Gastrulation defective</td>
<td></td>
<td></td>
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<tr>
<td>11D3-8</td>
<td>BREAKPOINT: In(1)sc260-14</td>
<td>REF: 105</td>
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<td>CROSS LIST: 1B2,3</td>
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<td>12</td>
<td>CLONE NAME: S21b</td>
<td>REF: 16</td>
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<tr>
<td>Genes:</td>
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<tr>
<td>12A</td>
<td>INSERTION: tAP-17.4.8</td>
<td>REF: 112</td>
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<td>CROSS LIST: 35B2-3</td>
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<tr>
<td>12B</td>
<td>INSERTION: P[(w,ry)E]2</td>
<td>REF: 176</td>
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<td>CROSS LIST: 3C1,2; 87D</td>
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<tr>
<td>12B,C</td>
<td>CLONE NAME: PYP3</td>
<td>REF: 2</td>
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<tr>
<td>Genes:</td>
<td>yolk protein 3</td>
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<tr>
<td>INSERTION: SRS3.9-1</td>
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<td>REF: 115</td>
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<td>CROSS LIST: 66D11-15, 87D12</td>
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<td>12BC</td>
<td>INSERTION: AR4-032(X)</td>
<td>REF: 126</td>
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<tr>
<td>CROSS LIST: 3C1,2</td>
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<tr>
<td>12D</td>
<td>INSERTION: R301.2</td>
<td>REF: 115</td>
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<td>CROSS LIST: 87D12</td>
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<tr>
<td>12DE</td>
<td>CLONE NAME: pDt17R, pDt27, pDt73</td>
<td>REF: 27</td>
</tr>
<tr>
<td>Genes:</td>
<td>ser 7,4,4-7 tRNA, respectively</td>
<td></td>
</tr>
<tr>
<td>12E</td>
<td>INSERTION: E 7-10</td>
<td>REF: 134</td>
</tr>
<tr>
<td>CROSS LIST: 3C1-2</td>
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<tr>
<td>12F</td>
<td>CLONE NAME: lambda 32-10</td>
<td>REF: 58</td>
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<tr>
<td>Genes:</td>
<td>tRNA</td>
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<tr>
<td>12F1,2</td>
<td>CLONE NAME: Dm2L1</td>
<td>REF: 75</td>
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<tr>
<td>GENES: tandem repeated 2L1 sequence</td>
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<tr>
<td>12</td>
<td>CLONE NAME: adm 136F10</td>
<td>REF: 34</td>
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<tr>
<td>Genes:</td>
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<tr>
<td>13A2-5</td>
<td>BREAKPOINT: In(1)sc29</td>
<td>REF: 105</td>
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<tr>
<td>CROSS LIST: 1B</td>
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</tbody>
</table>
13A-C
INSERTION: BS.27-5
CROSS LIST: 66D11-15,87D12
REF: 115

13CD
INSERTION: SB2.1-6
CROSS LIST: 66D11-15,87D12
REF: 115

13EF
INSERTION: P[(w,ry)E]3g;P[(w,ry)G]4
CROSS LIST: 3C1,2; 87D; 3C1,2; 87D
CLONE NAME: M3,6,8,10
GENES: c-myb
REF: 178

13F
CLONE NAME: G2
GENES: Glyceraldehyde-3-phosphate dehydrogenase
REF: 135

14A
INSERTION: 21-1
CROSS LIST: 3C11,12;87D
REF: 140

14BC
CLONE NAME: adm 132B8
GENES:
REF: 34

14D
CLONE NAME: M75
GENES:
REF: 84

15A1
CLONE NAME: unn
GENES: rudimentary
REF: 30

15A,B
CLONE NAME: 548
GENES: head specific RNA
REF: 31

15B
CLONE NAME: unn
GENES: ribosomal protein S 18
REF: 85

15DE
INSERTION: BS2.7-10
CROSS LIST: 66D11-15,87D12
REF: 115

15E
INSERTION: P15-1
REF:

15F
CLONE NAME: Dmf3
GENES: forked
REF: 158
CLONE NAME: lambda f1
GENES: forked
REF: 77

16B3-5
CLONE NAME: PTE-1
GENES:
REF: 2
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16BC
INSERTION: S6.9-11
CROSS LIST: 66D11-15, lacZ, 87D12
REF: 145

16C
INSERTION: H1
CROSS LIST: 87D12
REF: 221

16D
INSERTION: unn
CROSS LIST: 87D12
REF: 117

16E
INSERTION: 27 N/P-A
CROSS LIST: 67B, 87D12
REF: 200

16EF
CLONE NAME: unn
GENES: shaker
REF: 40

16F
INSERTION: 27C X/X-A
CROSS LIST: 67B, 87D12
REF: 200

16F17
CLONE NAME: adm 135H4
GENES:
REF: 34

17AB
CLONE NAME: lambda dmpt 61
GENES:
REF: 58

17C
INSERTION: tAP-5
CROSS LIST: 35B3-5
REF: 181

17DE
INSERTION: B1-2
CROSS LIST: 3C1-2
REF: 113

18A
INSERTION: R704.3
CROSS LIST: 87D12
REF: 115

18A3,4
BREAKPOINT: In(1)y4
CROSS LIST: 1A8B1
REF: 105

18B8,9
BREAKPOINT: In(1)sc9
CROSS LIST: 1B2,3
REF: 105

18CD
CLONE NAME: A57
GENES: maternal restricted transcript
REF: 69

18D
INSERTION: 16-3
CROSS LIST: 3C11,12;87D
CLONE NAME: lambda DmG21
GENES: G6PD
REF: 28
INSERTION: BS2.7-3
CROSS LIST: 66D11-15, 87D12
CLONE NAME: trc 18
GENES:

18E
CLONE NAME: Dm14
GENES: G6PD
CLONE NAME: lambda Dm G6PD 14
GENES: G6PD

19A
INSERTION: cHB delta-89

19E
INSERTION: +65
INSERTION: tAP-1
CROSS LIST: 35B3-5

19E8
CLONE NAME: PP95
GENES: unc

19EF
CLONE NAME: DCg2
GENES: collagen-like gene
CROSS LIST: 20AB

19F
INSERTION: P[(w,ry)E]1
CROSS LIST: 3C1,2; 87D
CLONE NAME: pDt67R
GENES: Arg rTNA locus

20
BREAKPOINT: Dp(1;f)101
CROSS LIST: 2B1,2
BREAKPOINT: T(1;3)CbxrvR17.49A, T(1;3)P115, Dp(3;1)P68
CROSS LIST: 89E,89F

20A
INSERTION: 2
CROSS LIST: 88F
INSERTION: Adh hs20A
CROSS LIST: 35B2-3,87

20AB
CLONE NAME: DCg2
GENES: collagen-like gene
CROSS LIST: 19EF

20C
BREAKPOINT: In(1)scL8
CROSS LIST: 1B3,4

20C1
BREAKPOINT: In(1)sc4
CROSS LIST: 1B3,4
20CD
INSERTION: P(ry,HsAFP)
CROSS LIST: 87D12
REF: 141

20D
INSERTION: AR4-024
CROSS LIST: 3C1,2
REF: 126

20D1
BREAKPOINT: In(1)scS1
CROSS LIST: 1B3,4
REF: 105
BREAKPOINT: In(1)sc8, In(1)y3P
CROSS LIST: 1B2,3, 1B1,2
REF: 103

20F
BREAKPOINT: Df(1)w-64D= In(1)3B1,2-20F+,
Df(1)3B1,2-3C2,3
CROSS LIST: 3B1,2
REF: 107

21 tip
BREAKPOINT: T(3;2)bx-d-D36 complex
CROSS LIST: 89E
REF: 110

21A
CLONE NAME: lambda alpha 8; lambda 8001 to 8014
GENES: lethal(2) giant larvae
REF: 131
CLONE NAME: alpha 8
GENES: l(2)gl
REF: 154
CLONE NAME: lambda T-A
GENES: telomeres
REF: 156
CROSS LIST: 1A, 60F, 61A, 100F, 102F

21A1,2
BREAKPOINT: T(2;3)Ubx16160.18
CROSS LIST: 89E
REF: 110

21B
INSERTION: P[(w,ry)G]1
CROSS LIST: 3C1,2, 87D
REF: 176
CLONE NAME: adm 142G5
GENES: 
REF: 34

21C
CLONE NAME: unn
GENES: double sex cognate
REF: 88

21D
CLONE NAME: pDm6A65
GENES: U1 snRNA
CROSS LIST: 82E, 95C
REF: 151
INSERTION: P[(w,ry)F]4-2
CROSS LIST: 3C1,2; 87D
REF: 176
CLONE NAME: lambda Dm9
GENES: LSP-1 beta
REF: 219
CLONE NAME: pD957
GENES: 
REF: 3
INSERTION: R602.1
CROSS LIST: 87D12
REF: 115
CLONE NAME: lambda Dm (Can S) LSP1 beta: 1
GENES: LSP1 beta
REF: 130
21DE
INSERTION: tAP-10, 4.8
CROSS LIST: 35B2-3
REF: 112

21F
CLONE NAME: adm 123D12, 123H3, 128B8
GENES:
CROSS LIST: 21F
REF: 34

21F
CLONE NAME: adm 123D12, 123H3, 128B8
GENES:
CROSS LIST: 21F
REF: 34

22A
CLONE NAME: adm 123D12, 123H3, 128B8
GENES:
CROSS LIST: 21F
REF: 34
INSERTION: R604.1
CROSS LIST: 87D12
REF: 115

22AC
CLONE NAME: adm 123D12, 123H3, 128B8
GENES: 4S RNA
REF: 193

22B
CLONE NAME: mDm103H10, mDm108C7
GENES:
CROSS LIST: 45A
REF: 1
INSERTION: w20.2, w20.10
CROSS LIST: 3C1-2
REF: 120

22B1,2
BREAKPOINT: T(2;3)Cbx rVR17.175
CROSS LIST: 89E
REF: 110

22B, C
CLONE NAME: adm 129E7
GENES:
REF: 34

22F1,2
CLONE NAME: unn
GENES: decapentaplegic complex
CLONE NAME: pDm22F Ya, Yb
GENES: tyr tRNA
REF: 189

22F-23A
INSERTION: P13-1
CROSS LIST: 87D12
REF:

23A
INSERTION: +65
CROSS LIST: 87, 87D12
REF: 118

23A3-7
CLONE NAME: unn
GENES:
REF: 59

23BC
CLONE NAME: B13
GENES: maternal restricted transcript
INSERTION: q5:2
CROSS LIST: 68C3-5, 87D12
REF: 124

23E
CLONE NAME: pDt5
GENES: ser 7 tRNA
REF: 27
24AB
INSERTION: $P[(w,ry)D]_4$
CROSS LIST: 3C1,2; 87D
CLONE NAME: M98
GENES: 0-1 hrs.
CROSS LIST: 67C4,5
REF: 176

24C
CLONE NAME: mDm101A10
GENES:
REF: 1

24CD
INSERTION: AR4-24
CROSS LIST: 3C1,2
REF: 126

25A
BREAKPOINT: T(1;2)sc19
CROSS LIST: 1B4-7
INSERTION: 6-1
CROSS LIST: 3C11,12; 87D
REF: 105

25B
INSERTION: alpha T3.1
CROSS LIST: 11A
INSERTION: alpha T3.21
CROSS LIST: 11A
REF: 82

25BC
CLONE NAME: mDm109D3
GENES:
REF: 1

25C
INSERTION: $P[(w,ry)D]_1$
CROSS LIST: 3C1,2; 87D
CLONE NAME: DCg-1
GENES: collagen-like gene
INSERTION: R401.3
CROSS LIST: 87D12
REF: 25

25D
CLONE NAME: 150-3(lambda)
GENES: blastoderm-specific poly(A) RNA
REF: 47

25D1-4
CLONE NAME: MH5
GENES:
REF: 6

25F
INSERTION: 27P X/X-B
CROSS LIST: 67B, 87D12
REF: 200

25F5
CLONE NAME: Gpdh 411
GENES: glycerol-3-phosphate dehydrogenase
REF: 139

26A
CLONE NAME: DmcMM115, LS1
GENES: vitelline
REF: 72
26A3-5
CLONE NAME: Gpdh 411
GENES: glycerol-3-phosphate dehydrogenase

26A7-9
CLONE NAME: unn
GENES: beta galactosidase

26AB
CLONE NAME: A20
GENES:

26B
INSERTION: D4
CROSS LIST: 99D, 87D12
INSERTION: D1
CROSS LIST: 99D, 87D12

27A-C
INSERTION: icarus-neo
CROSS LIST: heatshock, neo

27C
INSERTION: 33-4
CROSS LIST: 3C11, 12; 87D
CLONE NAME: Pupal cuticle protein
GENES: within GAR transformylase intron
CLONE NAME: unn
GENES: Gar transformylase, synthetase, AIR synth.

27D
CLONE NAME: lambda 39-1
GENES:
CROSS LIST: 43A, 91C

27F
CLONE NAME: adm 125G11
GENES:

28A
CLONE NAME: 551
GENES: head specific RNA
INSERTION: BS2.7-11
CROSS LIST: 66D11-15, lacZ, 87D12

28C
CLONE NAME: 538
GENES: head specific RNA

28D5
INSERTION: E 7-1
CROSS LIST: 3C1-2

28D9-12
CLONE NAME: unn
GENES: CDNA, Kc cells

29
BREAKPOINT: T(2;3)Hm complex
CROSS LIST: 89E
29A
CLONE NAME: pDt59R
GENES: lys 5 tRNA locus
REF: 27

29A-C
BREAKPOINT: T(2;3)P10
CROSS LIST: 89E
REF: 110

29B
INSERTION: R308.1
CROSS LIST: 87D12
REF: 115

29B1-4
CLONE NAME: unn
GENES: CDNA, Kc cells
REF: 8

29C
CLONE NAME: unn
GENES: SRC homologous
REF: 61

30A
INSERTION: 28C-B
CROSS LIST: 67B, 87D
INSERTION: 28C-B
CROSS LIST: 87D12, 67B
INSERTION: cHB delta-89
CROSS LIST: 87-lacZ, 87D12
REF: 180
REF: 121
REF: 118

30A9-30B1,2
CLONE NAME: lambda Dm65
GENES: P6
REF: 219

30B
CLONE NAME: lambda dmpt 75
GENES:
REF: 58

30C
INSERTION: S6.9-3
CROSS LIST: 66D11-15, lacZ, 87D12
INSERTION: A4-N22
CROSS LIST: 3C1,2
REF: 115
REF: 126

30DE
CLONE NAME: adm 136D3
GENES:
REF: 34

30EF
CLONE NAME: lambda dmpt 104
GENES:
REF: 58

31
BREAKPOINT: T(2;3) Ubx18264.1
CROSS LIST: 89E
REF: 110

31A
CLONE NAME: mDm 106A10
GENES:
CLONE NAME: M35
GENES:
REF: 1
REF: 84
31B
INSERTION: cHB delta-89
CROSS LIST: 87, lacZ, 87D12
REF: 118

31C
CLONE NAME: adm 134G6
GENES:
CLONE NAME: adm 142H
GENES:
CROSS LIST: 33B
REF: 34

31F
CLONE NAME: adm 142F4
GENES:
CROSS LIST: 39F
REF: 34

32
BREAKPOINT: T(2;3)Hm complex
CROSS LIST: 89E
REF: 110

32AB
CLONE NAME: 503
GENES: head specific RNA
REF: 31

32BC
INSERTION: BS2.7
CROSS LIST: 66D11-15, 87D12
REF: 115

32CD
INSERTION: cp70 delta B
CROSS LIST: 87, lacZ, 87D12
CLONE NAME: 231
GENES: myogenic cell RNA
REF: 63

32EF
CLONE NAME: Dmc MM99
GENES: vitelline
REF: 72

32F
INSERTION: g6:5
CROSS LIST: 68C3-5, 87D12
REF: 124

32F-33E
CLONE NAME:
GENES: paired
REF: 218

33AB
CLONE NAME: unn
GENES: extra sex combs
REF: 70

33B
CLONE NAME: adm 142H
GENES:
CROSS LIST: 31C
REF: 34
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CLONE NAME: adm 124D9  GENES:  REF: 34

CLONE NAME: M146  GENES: 0-1,2-5,3-5  REF: 84

33B1,2  CLONE NAME: esc  GENES:  REF: 218

34  BREAKPOINT: T(2;3) Ubx4.3 (madrid)  CROSS LIST: 89E  REF: 110

34AB  CLONE NAME: A34  GENES:  REF: 69

34C  CLONE NAME: DmcMM99  GENES: vitelline, Oregon R ovaries, cDNA  REF: 72

34D  INSERTION: g7:4  CROSS LIST: 68C3-5,87D12  REF: 124

34E  INSERTION: P(w)11P  CROSS LIST: 3C1,2  REF: 136

34EF  INSERTION: g711:1  CROSS LIST: 68C3-5,87D12  REF: 124

34F  CLONE NAME: 527  GENES: head specific RNA  REF: 31

35A  INSERTION: 15-1;15-2  CROSS LIST: 3C11,12;87D; 3C11,12;87D  REF: 140

35A4-35B1  CLONE NAME: Adh, outspread, no ocelli  REF: 157

35AB  CLONE NAME: lambda CHD1  GENES:  REF: 133

35B1-3  CLONE NAME: lambda ob 5  GENES: Adh, noc, osp, 1(2)br22  REF: 132

BREAKPOINT: Tp(2;2)ScoR+12;In(2LR)ScoR+1;In(2L)noc2; Df(2L)A267;Df(2L)A379  REF: 132

CROSS LIST: 34A8-B1; 44C3-5; 36D3; 35B9-10; 57A8-10-60

CLONE NAME: lambda w4.04  GENES: Adh, noc, osp, 1(2)br22  REF: 132

BREAKPOINT: Df(2L)osp29  REF: 132

CROSS LIST: 35E6
CLONE NAME: lambda w3.13
GENES: Adh, noc, osp, 1(2)br22
BREAKPOINT: T(2;3)osp90
CROSS LIST: 89B9-11
CLONE NAME: lambda ob 9.04
GENES: Adh, noc, osp, 1(2)br22
BREAKPOINT: Df(2L)A446;In(2LR)noc4;In(2L)fr27;T(2;3)Mpe
CROSS LIST: 49B1-3; 41; 35D1-2; 86C1-2
CLONE NAME: lambda ob 2.01
GENES: Adh, noc, osp, 1(2)br22
BREAKPOINT: In(2L)ScoR+11;Df(2L)A220;Df(2L)A264
CROSS LIST: 35D1-2; 35B9; 35B8-9

35B1,2
CLONE NAME: ob 9.04
GENES: no-ocelli

35B1-3
CLONE NAME: lambda ob 1.12
GENES: Adh, noc, osp, 1(2)br22
BREAKPOINT: Df(2L)TE146(Z)GW8
CROSS LIST: 35A2-4 to B1-2
CLONE NAME: lambda gAC2
GENES: Adh, noc, osp, 1(2)br22
BREAKPOINT: Df(2L)A48;Df(2L)A72
CROSS LIST: 35D5-7; 35B7-8
CLONE NAME: lambda ob 4.01
GENES: Adh, noc, osp, 1(2)br22
BREAKPOINT: Df(2L)b84a8
CROSS LIST: 35B3-4
CLONE NAME: lambda ob 7.06
GENES: Adh, noc, osp, 1(2)br22
BREAKPOINT: In(2L)ScoR+8;Df(2L)b81a1;Df(2L)TE146(Z)GW4;
Df(2L)TE146(Z)GW10
CROSS LIST: 35D1-2; 34D3; 34F1-2; 34F1
CLONE NAME: lambda ob 8.10
GENES: Adh, noc, osp, 1(2)br22
BREAKPOINT: In(2LR)ScoR+9;In(2LR)noc7;Df(2L)nNxF2
CROSS LIST: 41; 46B1-2; 35B10
CLONE NAME: lambda gAC3
GENES: Adh, noc, osp, 1(2)br22
BREAKPOINT: In(2L)osp59
CROSS LIST: 38B3-6
CLONE NAME: lambda w2.0
GENES: Adh, noc, osp, 1(2)br22
BREAKPOINT: Df(2L)noc20
CROSS LIST: 35B2
CLONE NAME: lambda ob 10.02
GENES: Adh, noc, osp, 1(2)br22
BREAKPOINT: Df(2L)n7813
CROSS LIST: 35D5-7

35B3-5
CLONE NAME: AC
GENES: alcohol dehydrogenase
35B9-C1
  CLONE NAME: cln 149
  GENES: crinkled locus
  INSERTION: TE 36
  CROSS LIST: 3C1,2-3,4; FB elements
  REF: 149

35C36
  CLONE NAME: adin 125E7
  GENES:
  REF: 34

35DE
  INSERTION: S11.4-1
  CROSS LIST: 66D11-15
  REF: 115

36A
  INSERTION: tAP-3
  CROSS LIST: 35B3-5
  INSERTION: unn
  CROSS LIST: 87,lacZ,87D12
  REF: 118

36B
  CLONE NAME: unn
  GENES: myosin heavy chain
  CLONE NAME: unn
  GENES: walked from myosin heavy chain
  REF: 20

36C
  CLONE NAME: iH-6
  GENES: dorsal
  BREAKPOINT: In(2L)dlT; In(2L)dlH
  CROSS LIST: 21E,F; 37C1,2
  INSERTION: tAP-8C,4.8
  CROSS LIST: 35B2-3
  REF: 172

36D1-E1
  BREAKPOINT: Df(2L)VA18
  CROSS LIST: 37C1,3
  REF: 35

36E4-6
  BREAKPOINT: Df(2L)hk18
  CROSS LIST: 37B9-C1,2
  REF: 35

36F
  CLONE NAME: mdm 103D5
  GENES:
  REF: 1

37A
  INSERTION: P[(w,ry)G]2
  CROSS LIST: 3C1,2; 87D
  INSERTION: unn
  CROSS LIST: 67B,lacZ,87D12
  REF: 118

37B9-C1,2
  CLONE NAME: unn
  GENES: dopa decarboxylase
  BREAKPOINT: Df(2L)hk18
  CROSS LIST: 36E4,6
  REF: 35

37B13-C5
  CLONE NAME: lambda Ddc-1 thru-20
  GENES: dopa decarboxylase
  REF: 35
37BC
CLONE NAME: DmcMM109
GENES: Oregon R ovaries
INSERTION: unn
CROSS LIST: 35B2-3,63BC1

37C1,5
BREAKPOINT: Df(2L)TE42-1
CROSS LIST: 38F4
CLONE NAME: unn
GENES: Ddc
CROSS LIST: see Table 2
BREAKPOINT: Df(2L)VA18, Df(2L)VA17, Df(2L)TE42-1
Df(2L)VA12
CROSS LIST: 36D1-E1, 37F5-38A1, 38C1, 2D1,2, 38B1,2-C1,2

37F5-38A1
BREAKPOINT: Df(2L)VA17
CROSS LIST: 37C1,5

38A6
CLONE NAME: 2E2
GENES:

38B
INSERTION: P(ry, HsAFP)2
CROSS LIST: 87D12
INSERTION: cHB delta-59
CROSS LIST: 87, lacZ, 87D12

38B1,2-C1,2
BREAKPOINT: Df(2L)VA12
CROSS LIST: 37C1-5

38BC
INSERTION: tAP-19,4.8
CROSS LIST: 35B2-3

38C1,2-D1,2
BREAKPOINT: Df(2L)TE42-1
CROSS LIST: 37C1-5

38D
INSERTION: 1
CROSS LIST: 88F

38E
CLONE NAME: F33
GENES: caudal
INSERTION: unn
CROSS LIST: 87D12

38F4
BREAKPOINT: Df(2L)TE42-1
CROSS LIST: 37C1-5

39
BREAKPOINT: T(2;3)Ubx19286.8m
CROSS LIST: 89E
39B

INSERTION: 28P-C
CROSS LIST: 67B, 87D
INSERTION: 28P-C
CROSS LIST: 87D12, 67B

39BC

INSERTION: E 5-5
CROSS LIST: 3C1-2
INSERTION: S6.9-8
CROSS LIST: 66D11-15, lacZ, 87D12

39DE

INSERTION: H5
CROSS LIST: 87D12
CLONE NAME: unn
GENES: histone

39E

INSERTION: AR4-3
CROSS LIST: 3C1-2, 87D12
CLONE NAME: adm 136D9
GENES: chromocenter
INSERTION: B4
CROSS LIST: 99D 87D12

39EF

INSERTION: AR4-2
CROSS LIST: 3C1-2, 87D12

39F

CLONE NAME: adm 142F4
GENES: chromocenter
CROSS LIST: 31F

40

CLONE NAME: adm 106H5, 123C3, adm136D9
GENES: chromocenter

40F

INSERTION: AR4-3
CROSS LIST: 3C1-2, 87D12

41

CLONE NAME: adm 130B2
GENES: chromocenter
CROSS LIST: 80
BREAKPOINT: T(2;3)AntpNS+RC8
CROSS LIST: 84B2

41A

BREAKPOINT: T(2;3)bxd22044D
CROSS LIST: 89E
BREAKPOINT: T(2;3)Ubx17756.180, T(2;3)Ubx18136.147
T(2;3)Ubx19649.18, T(2;3)UbxD1, T(2;3)bxdB231
T(2;3)CbxrV17.22X, T(2;3)rvR17.34
CROSS LIST: 89E

41F

BREAKPOINT: T(2;3)Ubx16160.36
CROSS LIST: 89E
42A
CLONE NAME: 4 tRNA genes
GENES: cluster of asn, arg, lys, ile tRNAs
CLONE NAME: mDm 106F8
GENES:
INSERTION: R301.1
CROSS LIST: 87D12
INSERTION: +411
CROSS LIST: 87, 87D12
INSERTION: tAP-13,4.8
CROSS LIST: 35B2-3

42AB
INSERTION: R303.1
CROSS LIST: 87D12

42BC
BREAKPOINT: T(2;3)bxd x22290.11x
CROSS LIST: 89E
CLONE NAME: lambda st11-205.16
GENES:

42DE
INSERTION: unn
CROSS LIST: 87D12

42E
CLONE NAME: pDt 61
GENES: tRNA-lys-2
INSERTION: R305.1
CROSS LIST: 66D11-15, lacZ, 87D12

42EF
CLONE NAME: adm 126F7, 127A10
GENES:

42F
INSERTION: 28X-C
CROSS LIST: 67B, 87D
INSERTION: S6.9-4
CROSS LIST: 66D11-15, lacZ, 87D12

43
INSERTION: R704.1
CROSS LIST: 87D12

43A
CLONE NAME: lambda 39-1
GENES:
CROSS LIST: 27D, 91C

43A2-5
CLONE NAME: draf2
GENES: raf oncogene homologous
CROSS LIST: minor homology to 2F5-6

43AB
CLONE NAME: 555
GENES: head specific RNA
43BC
CLONE NAME: B17
GENES: maternal restricted transcript

43C
INSERTION: R304.1
CROSS LIST: 87D12

43CD
INSERTION: +411
CROSS LIST: 87, 87D12

43DE
CLONE NAME: B45
GENES: maternal restricted transcript

43E
CLONE NAME: G3
GENES: Glyceraldehyde-3-phosphate dehydrogenase
INSERTION: S6.9-7
CROSS LIST: 66D11-15, lacZ, 87D12
INSERTION: g7:7
CROSS LIST: 68C3-5, 87D12

44A
BREAKPOINT: T(Y;2;3)Mcp rvc10 complex
CROSS LIST: 89E

44C
CLONE NAME: L10
GENES:

44C
CLONE NAME: lambda e8 e9
GENES:
INSERTION: S6.9-7
CROSS LIST: 66D11-15, lacZ, 87D12

44C4
BREAKPOINT: T(2;3)P75Ubx 5T17.14-17
CROSS LIST: 89E

44CD
CLONE NAME: 536
GENES: head specific RNA
INSERTION: S6.9-7
CROSS LIST: 66D11-15, lacZ, 87D12

44D
CLONE NAME: lambda DmLCP1-13
GENES: larval cuticle protein

44E
INSERTION: R3.9-4
CROSS LIST: 66D11-15, 87D12

44EF
CLONE NAME: M51
GENES:
CROSS LIST: 64B
44F
CLONE NAME: 129E7
GENES: 
INSERTION: cp70ZT
CROSS LIST: 87-lacZ, 87D12

45A
CLONE NAME: mDm103H10, mDm108C7
GENES: 
CROSS LIST: 22B
INSERTION: S11.4-1
CROSS LIST: 66D11-15

45AB
INSERTION: F4
CROSS LIST: 99D, 87D12

45B
CLONE NAME: M199
GENES: 0-3.5 hrs

45D
CLONE NAME: mDm108A8
GENES: 
INSERTION: AR4-020(11)
CROSS LIST: 3C1, 2

45E
INSERTION: DR-18
CROSS LIST: 87D12, 37C1, 2
INSERTION: cHB delta-73
CROSS LIST: 87, lacZ, 87D12

46B
CLONE NAME: B41
GENES: maternal restricted transcript

46C
INSERTION: 27 X/A-2-A; 27 X/A-2-B; 27 X/A-2-C; 27 X/A-2-D
CROSS LIST: 67B, 87D12
CLONE NAME: S72
GENES: eve
INSERTION: A4-N21
CROSS LIST: 3C1, 2

46DF
CLONE NAME: 236
GENES: myogenic cell RNA

46E
CLONE NAME: 549
GENES: head specific RNA

47A
INSERTION: tAP-18, 4.8
CROSS LIST: 35B2-3
INSERTION: A1-1
CROSS LIST: 3C1-2, 87D12
47C
INSERTION: chB delta-89
CROSS LIST: 87, lacZ, 87D12
REF: 118

47D
INSERTION: P[(w,ry)E]8
CROSS LIST: 3C1,2; 87D
INSERTION: 27 N/P-B
CROSS LIST: 67B, 87D12
REF: 200

47E
CLONE NAME: 528
GENES: head specific RNA
REF: 31

47F
CLONE NAME: unn
GENES:
REF: 14

47F48D
CLONE NAME: 217
GENES: myogenic cell RNA
REF: 63

48A
CLONE NAME: unn
GENES: engrailed
REF: 14

48AB
INSERTION: g7:3
CROSS LIST: 68C3-5, 87D12
REF: 124

48B
INSERTION: hsp26-lacZ
INSERTION: tAP-6
CROSS LIST: 35B3-5
CLONE NAME: pDt74
GENES: Met 2 tRNA
REF: 27

48C
CLONE NAME: adm 132A7
GENES:
REF: 34

48D
CLONE NAME: Dm-15
GENES: F1, female enriched RNA (all stages)
CROSS LIST: homology to F2 at 100E
INSERTION: DR-9
CROSS LIST: 87D12, 37C1,2
REF: 114

48E
CLONE NAME: adm 135E10
GENES:
REF: 34

48E-F
INSERTION: tAP-4
CROSS LIST: 35B3-5
REF: 181

48EF
CLONE NAME: aDms7
GENES: Deb-A, Deb-B
REF: 198
48F
CLONE NAME: 38B10
GENES: his tRNA genes
CLONE NAME: 543
GENES: head specific RNA

49A
CLONE NAME: p500, p2.2
GENES: calmodulin

49A12B3
CLONE NAME: unn
GENES: possible site aristapedioid

49B
INSERTION: unn
CROSS LIST: 67B, lacZ, 87D12

49C
INSERTION: P(w)24S
CROSS LIST: 3C1, 2
CLONE NAME: mDm101D3
GENES:

49CD
CLONE NAME: mDm101D12
GENES:

49D
INSERTION: -51
CROSS LIST: 87, 87D12
INSERTION: A1
CROSS LIST: 99D, 87D12
INSERTION: S38M-5
CROSS LIST: 7E11
INSERTION: AR4-042
CROSS LIST: 3C1, 2

49DE
CLONE NAME: adm 140D1
GENES:

49DEF
CLONE NAME: lambda 120
GENES: Vestigial

49E5F1
CLONE NAME: unn
GENES: possible site aristapedioid

49EF
INSERTION: S3.8-6
CROSS LIST: 66D11-15, 87D12

49F
CLONE NAME: Dm1606
GENES: muscle specific Troponin C(ca++)
INSERTION: tAP-9, 4.8
CROSS LIST: 35B2-3
50A
INSERTION: P[(w,ry)F]1
CROSS LIST: 3C1,2, 87D
REF: 176

50
CLONE NAME: L6
GENES:
REF: 16

50AB
CLONE NAME: 7 tRNA genes
GENES: cluster of leu and ile tRNAs
REF: 191

50B
CLONE NAME: adm 142E9
GENES:
INSERTION: R306.1, S6.9-6
CROSS LIST: 87D12, lacZ, 66D11-15
REF: 115

50BC
CLONE NAME: 7 tRNA genes
GENES: cluster of lys tRNAs
REF: 192

50C
CLONE NAME: mDm3021
GENES:
REF: 1

50CD
CLONE NAME: adm 133H7,136F9,138G8,130H8
GENES:
REF: 34

50F
CLONE NAME: unn
GENES: double sex cognate
REF: 88

51A
CLONE NAME: S34
GENES:
REF: 16

51B
CLONE NAME: S14
GENES: head specific RNA
REF: 31

51CD
CLONE NAME: A19
GENES: maternal restricted transcript
REF: 69

51D
CLONE NAME: adm 134E2
GENES:
REF: 34

51DE
CLONE NAME: mDm102F11, mDm102B6
GENES:
REF: 1

51E
BREAKPOINT: T(2;3)Cbx rVR17.6F
CROSS LIST: 89E
REF: 110

52A1,2
CLONE NAME: M144
GENES:
REF: 84
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<td>T(2;3)Ubx X6000.78A4</td>
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53CD
CLONE NAME: rpA1
GENES: rpA1
REF: 199
CLONE NAME: rp A1
GENES: ribosomal protein
REF: 212

39CD
CLONE NAME: unn
GENES: ribosomal protein A1
REF: 17

52CD
CLONE NAME: lambda Dm32 (class A)
GENES: amy pseudogene
REF: 33

53E
INSERTION: R3,9-1
CROSS LIST: 66D11-15,87D12
REF: 115

53EF
INSERTION: SB2.1-1
CROSS LIST: 66D11-15,87D12
REF: 115

53F
CLONE NAME: lambda dmpt116
GENES:
INSERTION: g4:1
CROSS LIST: 68C3-5,87D12
REF: 58

54A
INSERTION: 2
CROSS LIST: 88F
INSERTION: 2
CROSS LIST: 88F
REF: 123

54A1B1
CLONE NAME: lambda Dm 65 (class B)
GENES: amylase duplication
REF: 33

54C
INSERTION: Adh,hs54c
CROSS LIST: 35B2-3,87
REF: 116

54E
CLONE NAME: adm 54E.1
GENES: minor heat shock cDNA
BREAKPOINT: In(3LR)89/75+T(2;3)54/75,Ubx 6-26 Madrid
CROSS LIST: 89E
REF: 39

54F55A
CLONE NAME: adm 110A4,132C9,132E11, 132E12
GENES:
CLONE NAME: adm 132G5, 134A4, 135D12
GENES:
REF: 34
REF: 34

55BCD
CLONE NAME: adm 110G1,110H1,132D6
GENES:
REF: 34

55F
CLONE NAME: B32
GENES: maternal restricted transcript
REF: 69
56AB
INSERTION: H2
CROSS LIST: 87D12
REF: 221

56C
CLONE NAME: DTB2
GENES: B tubulin
REF: 25

56D
CLONE NAME: pTu56
GENES: B 1 tubulin
INSERTION: R3.9-6
CROSS LIST: 66D11-15,87D12
REF: 115

56D412
CLONE NAME: KV 2-70A
GENES: B tubulin
REF: 22

56EF
CLONE NAME: adm 135H8
GENES:
REF: 34

56F
CLONE NAME:
GENES: 5S RNA
CLONE NAME: lambda Dmt 56-6
GENES: tRNA Gly
INSERTION: DR-15, DR-5
CROSS LIST: 87D12,37C1,2
REF: 194

57
INSERTION: HB4/Scl
CROSS LIST: 21D
REF: 82

57A
INSERTION: alpha T3.21
CROSS LIST: 11A
REF: 82

57AB
INSERTION: 27P X/X-A;27P X/X-E
CROSS LIST: 67B, 87D12
REF: 200

57B
INSERTION: P[(w,ry)E]6;P[(w,ry)F]4-1
CROSS LIST: 3C1,2; 87D; 3C1,2; 87D
INSERTION: 27S-B
CROSS LIST: 67B, 87D12
CLONE NAME: unn
GENES:
REF: 176

57C
CLONE NAME: unn
GENES: punch
CLONE NAME: 525
GENES: head specific RNA
CLONE NAME: unn
GENES: tudor
REF: 92

57F
INSERTION: unn
CROSS LIST: 87D12
REF: 117
CLONE NAME: unn
GENES: c-erbB, EGF receptor protein

58C
CLONE NAME:
GENES: en-like homeobox
CROSS LIST: 95A-B

58D
INSERTION: 27 X/A-1-B
CROSS LIST: 67B, 87D12
INSERTION: AR4-043
CROSS LIST: 3C1,2

58EF
INSERTION: R3.9-5
CROSS LIST: 87D12,37C1,2

58F
CLONE NAME: adm 132A3,135D10,135E6
GENES:
INSERTION: cp70 delta B
CROSS LIST: 87,1acZ,87D12
INSERTION: tAP-7A,4.8
CROSS LIST: 35B2-3

59-60
BREAKPOINT: T(2;3)Mcp rvC1 complex
CROSS LIST: 89E

59B
INSERTION: A3-1
CROSS LIST: 3C1-2,87D12

59C
INSERTION: tAP-7A,4.8
CROSS LIST: 35B2-3
BREAKPOINT: T(2;3)bxd 29315.46
CROSS LIST: 89E

59D
INSERTION: 27C X/X-B
CROSS LIST: 67B, 87D12

59E
INSERTION: S11.4-1
CROSS LIST: 66D11-15
CLONE NAME: trc 59
GENES:

60A
INSERTION: R302.1
CROSS LIST: 87D12
CLONE NAME: adm 125C2
GENES:
CLONE NAME: A8
GENES:
CLONE NAME: adm 106H6
GENES:
CLONE NAME: B6
GENES: maternal restricted transcript
60AB
INSERTION: g1
CROSS LIST: 68C3-5, 87D12
REF: 124

60B
CLONE NAME: pTu60
GENES: B 3 tubulin locus
BREAKPOINT: T(2;3) Mcp rv26105A
CROSS LIST: 89E
INSERTION: BS2.7-4
CROSS LIST: 66D11-15, 87D12
REF: 110
REF: 115

60BC
INSERTION: 27P X/X-B
CROSS LIST: 67B, 87D12
CLONE NAME: B50
GENES: maternal restricted transcript
REF: 200
REF: 69

60C
INSERTION: C2
CROSS LIST: 99D, 87D12
CLONE NAME: DTB3
GENES: B tubulin locus
REF: 119
REF: 25

60C1-3
BREAKPOINT: T(1;2)scS2
CROSS LIST: 1B4-7

60C
INSERTION: unn
CROSS LIST: 87D12
REF: 117

60C6-8
CLONE NAME: KV 1-11
GENES: B tubulin locus
REF: 22

60E
INSERTION: S38Z-1
CROSS LIST: 7E11-7F1, 2, lacZ, 87D12
INSERTION: tAP-15A, 4.8
CROSS LIST: 35B2-3
REF: 115
REF: 112

60F
CLONE NAME: lambda T-A
GENES: telomeres
CROSS LIST: 1A, 21A, 61A, 100F, 102F
INSERTION: S3.8-4
CROSS LIST: 66D11-15, 87D12
REF: 115

60F3
CLONE NAME:
GENES: Kruppel
REF: 97

61A
CLONE NAME: lambda T-A
GENES: telomeres
CROSS LIST: 1A, 21A, 60F, 100F, 102F
INSERTION: 27C+G-B
CROSS LIST: 67B, 87D12
CLONE NAME: lambda Dm60
GENES: LSP-1 gamma
REF: 200
REF: 219
INSERTION: Bg61  
CROSS LIST: 87D12, 87, lacZ  
REF: 118

INSERTION: S38Z-5  
CROSS LIST: 7E11-7F1,2,lacZ,87D12  
CLONE NAME: lambda Dm (Can S) LSP1 gamma: 1  
GENES: LSP1 gamma  
REF: 130

61A1-3  
CLONE NAME: mDm105F3  
GENES:  
REF: 1

61A-B  
INSERTION: tAP-2  
CROSS LIST: 35B3-5  
REF: 181

61C  
INSERTION: Adh hs61C  
CROSS LIST: 35B2-3, 87  
REF: 116

61D  
INSERTION: 28P-A  
CROSS LIST: 67B, 87D  
INSERTION: cp70 delta B  
CROSS LIST: 87-lacZ, 87D12  
INSERTION: 28P-A  
CROSS LIST: 87D12, 67B  
REF: 180

61E  
INSERTION: SRS3.9-4  
CROSS LIST: 66D11-15,87D12  
REF: 115

61F  
INSERTION: P(ry,HsAFP)3  
CROSS LIST: 87D12  
CLONE NAME:  
GENES: double sex cognate  
REF: 88

61F-62A  
BREAKPOINT: In(3LR)Ubx 300  
CROSS LIST: 89E  
REF: 110

62A  
INSERTION: P[(w,ry)D]2  
CROSS LIST: 3C1,2; 87D  
CLONE NAME: 5 tRNA genes  
GENES: cluster of glu tRNAs  
CLONE NAME: adm 112C10  
GENES:  
CLONE NAME: lambda 48-9  
GENES: tRNA locus  
REF: 195

62AB  
INSERTION: tAP-27  
CROSS LIST: 35B2-3  
CLONE NAME: adm 140F12  
GENES:  
CROSS LIST: 97C  
REF: 34

REF: 176  
REF: 112

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<td>62CD</td>
<td>REF: 63</td>
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<td>Genes: myogenic cell RNA</td>
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<td>62D</td>
<td>REF: 34</td>
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<td>REF: 85</td>
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<td>Genes: ribosomal protein L12 locus</td>
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<td>Genes: minor hsp</td>
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65AB
INSERTION: w47.1 N8  
CROSS LIST: 3C1-2  
REF: 120

65B
CLONE NAME: IMP-L3  
GENES: 20-hydroxyecdysone inducible  
REF: 164

65C
CLONE NAME: adm 111F10  
GENES:  
REF: 34

65D
INSERTION: 28A-B  
CROSS LIST: 67B, 87D  
REF: 180  
INSERTION: 28A-B  
CROSS LIST: 87D12, 67B  
REF: 120

65D-66B
INSERTION: S38Z-7  
CROSS LIST: 7E11, lacZ, 87D12  
REF: 145

65F
INSERTION: g711:3  
CROSS LIST: 68C3-5, 87D12  
REF: 124

66A
INSERTION: 28X-D  
CROSS LIST: 67B, 87D  
REF: 180  
INSERTION: 28X-D  
CROSS LIST: 87D12, 67B  
REF: 121

66B
CLONE NAME:  
GENES: leu tRNA  
BREAKPOINT: Tp(3)P47  
CROSS LIST: 89E  
REF: 216

66C
CLONE NAME: IMP-E1  
GENES: 20-hydroxyecdysone inducible  
CLONE NAME: adm 126B4  
GENES:  
CROSS LIST: 64F  
BREAKPOINT: Tp(3)bxd 100  
CROSS LIST: 89E  
REF: 164  
REF: 34  
REF: 110

66CD
CLONE NAME: adm 106E3  
GENES:  
REF: 34

66D
CLONE NAME: 507, 547  
GENES: head specific RNA  
INSERTION: DR-17  
CROSS LIST: 87D12, 37C1, 2  
REF: 31  
REF: 114

66D9-10
CLONE NAME: .8247, 30152, 3019  
GENES:  
REF: 32
June 1986

Master List of Clones, etc.

66D10-15
CLONE NAME: unn
GENES:

66D11-15
CLONE NAME: unn
GENES: chorion protein genes
CROSS LIST: see Table 2

66D12-15
CLONE NAME: AB
GENES: g5es 18-1, s15-1, s19-1

66E1,2
INSERTION: Tt(3L)Ga6.0-1
CROSS LIST: 35B2-3

66E
INSERTION: AR4-012
CROSS LIST: 3C1,2

66F
CLONE NAME: lambda dmpt 121
GENES:

67A5-7-67B1,2
CLONE NAME: unn
GENES:

67B
CLONE NAME: overlapping
GENES: hsp22, hsp23, hsp26, hsp27, 1, 2, 3
CLONE NAME: lambda 88
GENES: loci of hsp 22, 23, 26, and 28
CLONE NAME: lambda Dmp 67
GENES: hsp and flanking transcripts
CLONE NAME: J1
GENES: includes hsp 28, 23, 26

67BC
INSERTION: 27P X/X-D
CROSS LIST: 67B, 87D12

67C
CLONE NAME: DTA2
GENES: delta tubulin

67C4,5
CLONE NAME: M98
GENES: 0-1 hrs.
CROSS LIST: 24AB

67DE
CLONE NAME: gt11.205.23
GENES:

67E
INSERTION: 27 C X/X-A
CROSS LIST: 67B, 87D12
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<td>68A</td>
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<td>pkdmH2, adm 134C10, pkdm2C1, 2G6</td>
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<td>GENES: intermolt IV, III, II RNA</td>
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<td>68C3-7</td>
<td>Sgs proteins</td>
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<td>GENES: Sgs protein genes</td>
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CROSS LIST: 89E  
INSERTION: \( g6:4 \)  
CROSS LIST: 68C3-5, 87D12  

68E3,4  
BREAKPOINT: \( \text{Df(3L)vin 3} \)  
CROSS LIST: 68C3-7  

68EF  
CLONE NAME: adm 133H1  
GENES:  

69  
CLONE NAME: L3g  
GENES:  

69C3-4  
BREAKPOINT: \( \text{In(3LR)bxd 113} \)  
CROSS LIST: 89E  

69CD  
INSERTION: \( \text{tAP-12,4.8} \)  
CROSS LIST: 35B2-3  

69D  
CLONE NAME: M37  
GENES:  

69F  
CLONE NAME: 270  
GENES: myogenic cell RNA  

70A  
CLONE NAME: IMP-L1  
GENES: 20-hydroxyecdysone inducible  
CLONE NAME: 63H5  
GENES: asp tRNA genes  
CLONE NAME: adm 107A4  
GENES:  

70AB  
INSERTION: \( \text{S3.8-3} \)  
CROSS LIST: 66D11-15, 87D12  
CLONE NAME: adm 128C11, 132B3  
GENES:  

70BC  
CLONE NAME: B20  
GENES: maternal restricted transcription  
CLONE NAME: pdT 55  
GENES: Val 4 tRNA locus  

70C  
CLONE NAME: adm 29D11  
GENES:  
INSERTION: \( \text{S11.4-2} \)  
CROSS LIST: 66D11-15  

REF: 196  
REF: 34  
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REF: 27  
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70D  
BREAKPOINT: In(3LR)Cbx rvR17.42  
CROSS LIST: 89E  
REF: 110

70D1-3  
CLONE NAME: lambda Dm117  
GENES: P1  
REF: 219

70D4,5  
CLONE NAME: Q111,Q131,Q112  
GENES: frizzled locus  
REF: 81

71A  
CLONE NAME: 2-5 (lambda)  
GENES: gastrula differential poly(A) RNA  
REF: 47

71AB  
INSERTION: -51  
CROSS LIST: 87,87D12  
CLONE NAME: adm 123C4  
GENES:  
REF: 118

71C  
INSERTION: S38M-1  
CROSS LIST: 7E11-7F1,2, M13+, 87D12  
REF: 115

71C3,4D1,2  
CLONE NAME:  
GENES: EIP 28/29 locus  
REF: 26

71CD  
BREAKPOINT: T(1;3)sc260-15  
CROSS LIST: 1B4,5  
REF: 105

71CE  
CLONE NAME: lambda cDm 20-24  
GENES: ecdysone induced late puff  
REF: 24

71DE  
CLONE NAME: adm 134A9,134A11,134C11,pkdm 46B7  
GENES: late I RNA  
CLONE NAME: pkdm 38C9, 38C4  
GENES: late II,III RNA  
REF: 34

71F  
INSERTION: tAP-11,4.8  
CROSS LIST: 35B2-3  
BREAKPOINT: In(3LR)AntpPW  
CROSS LIST: 84B2  
INSERTION: g7:2  
CROSS LIST: 68C3-5,87D12  
REF: 112

72BC  
CLONE NAME: 557  
GENES: head specific RNA  
REF: 31

72D11-72E1  
BREAKPOINT: In(3LR)bx4 106  
CROSS LIST: 89E  
REF: 110
72DE
CLONE NAME: lambda dmpt 115
GENES:

73A
CLONE NAME:
GENES: double sex cognate, not transformer
CLONE NAME:
GENES: transformer

73A3-4
CLONE NAME: lambda str4
GENES: scarlet

73A3-B1,2
CLONE NAME: stdb-st tra DIS-5 cABL std5
GENES:

73A3,4-73B
CLONE NAME:
GENES: tra

73B
INSERTION: Pc[ry(delta0-1)]2
CROSS LIST: 87D12
INSERTION: 275-D
CROSS LIST: 67B, 87D12
CLONE NAME: Dash
GENES: Abelson SRC homologous

73D
CLONE NAME: adm 73D.1
GENES: minor heat shock locus

73DEF
CLONE NAME: 521
GENES: head specific RNA

73E
INSERTION: P(ry,HsAFP)6
CROSS LIST: 87D12

74
BREAKPOINT: In(3)Ubx 130 (TM2) complex
CROSS LIST: 89E

74EF
CLONE NAME:
GENES: early ecdysone responding puff

74F-75A
BREAKPOINT: In(3LR)Antp NS+RC4
CROSS LIST: 84B2

75
CLONE NAME: S39
GENES:

75C
INSERTION: P[(w,ry)H]2-2
CROSS LIST: 3C1,2; 87D
BREAKPOINT: In(3LR)89/75+T(2;3)54/75, Ubx6.26 Madrid
CROSS LIST: 89E
CLONE NAME: adm 135F3
GENES:

75CD
INSERTION: R502.1
CROSS LIST: 87D12

75D
CLONE NAME: trc 75
GENES:
INSERTION: R706.1
CROSS LIST: 87D12

76A
CLONE NAME: adm 132D11
GENES:
INSERTION: A38M-4
CROSS LIST: 7E11-7F1,2, M13+, 87D12

76DE
CLONE NAME: B48
GENES: maternal restricted transcript

76F
CLONE NAME: mDm 104G3
GENES:

77A
INSERTION: cHB delta-73
CROSS LIST: 87, lacZ, 87D12

77DE
INSERTION: P(ry, HsAFP)1
CROSS LIST: 87D12

78BC
INSERTION: R603.1
CROSS LIST: 87D12

78CD
INSERTION: P[(w,ry)H]1
CROSS LIST: 3C1,2; 87D

78D
INSERTION: cHB delta-89
CROSS LIST: 87-lacZ, 87D12
INSERTION: cHB delta-89
CROSS LIST: 87-lacZ, 87D12

78D7,8
CLONE NAME: lambda Dm-208B
GENES: polycomb

79-80
CLONE NAME: ribosomal protein-21

79B
CLONE NAME: unn
GENES:
<table>
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<tr>
<th>Clone Name</th>
<th>Genes</th>
<th>Breakpoint</th>
<th>Cross List</th>
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<td>79C-E</td>
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<td>In(3LR)Cbx rVR17.44V</td>
<td>89E</td>
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<td>79E</td>
<td></td>
<td>+411</td>
<td>87,87D12</td>
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<td>79E1,2</td>
<td>13E5</td>
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<td>79F</td>
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<td>8-1</td>
<td>3C11,12,87D</td>
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<tr>
<td>80A</td>
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<td>B1,F2</td>
<td>99D,87D12</td>
</tr>
<tr>
<td>80B</td>
<td></td>
<td>In(3LR)Cbx rVR17.16R</td>
<td>89E</td>
</tr>
<tr>
<td>80C</td>
<td></td>
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</tr>
<tr>
<td>80F</td>
<td></td>
<td>adm 130B2</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>chromocenter</td>
<td></td>
</tr>
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<td>81</td>
<td></td>
<td>adm 128F12</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>chromocenter</td>
<td></td>
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<td></td>
<td></td>
<td>In(3)Hab rVC51, In(3)Ubx 125</td>
<td>89E</td>
</tr>
<tr>
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<td>In(3)Mcp rVC1 complex</td>
<td>87D</td>
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<tr>
<td>82A</td>
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<td>S6-7</td>
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<td>w47.4L</td>
<td>3C1-2</td>
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<td>82B</td>
<td></td>
<td>Adh hs82b</td>
<td>35B2-3,87</td>
</tr>
</tbody>
</table>
82BC
INSERTION: tAP-8B, 4.8
CROSS LIST: 35B2-3
INSERTION: g4:3
CROSS LIST: 68C3-5, 87D12

82C
INSERTION: 28X-E
CROSS LIST: 67B, 87D
INSERTION: 28X-E
CROSS LIST: 87D12, 67B

82E
CLONE NAME: pDm6A65, Dm 525
GENES: U1 snRNA
CROSS LIST: 21D, 95C
CLONE NAME: lambda Dm525
GENES: 2 SnRNA U1 pseudogenes (inverted)

82F
INSERTION: 5-1
CROSS LIST: 3C11, 12; 87D
CLONE NAME: 506
GENES: head specific RNA
INSERTION: AR4-025
CROSS LIST: 3C1, 2

83A
CLONE NAME: adm 136E4
GENES:
INSERTION: unn
CROSS LIST: 87D12

83A, B
CLONE NAME: pDt 66R2
GENES: Lys 5 tRNA locus

83AB
CLONE NAME: adm 140E12
GENES:

83B
INSERTION: 27S-E
CROSS LIST: 67B, 87D12
CLONE NAME: adm 123G4
GENES:

83BC
INSERTION: SB2.1-2
CROSS LIST: 66D11-15, 87D12

83C
CLONE NAME: mDm 105 B9
GENES:

83CD
CLONE NAME: B21, B31
GENES: maternal restricted transcript
83D4,5
CLONE NAME: B31
GENES:
BREAKPOINT: Tp(3;3)Dfd, Tp(3;3)DfdTRX1
CROSS LIST: 84A4,5, 98F1,2
REF: 69

83F
CLONE NAME: adm 140C1
GENES:
INSERTION: R3.9-3,9/ S38M-2
CROSS LIST: 87D12/ 7E11-7F1,2, M13+, 87D12
REF: 34, 115

84A1
BREAKPOINT: Df(3R)Scr
CROSS LIST: 84B2
REF: 102

84A1,2
BREAKPOINT: Df(3R)JA99
CROSS LIST: 84B2
REF: 102

84A4,5
BREAKPOINT: Tp(3;3)Dfd
CROSS LIST: 83D4,5
REF: 69

84A1
CLONE NAME:
GENES: zen
REF: 213

84A4,5-84C1,2
CLONE NAME:
GENES: antennapedia complex
REF: 49

84A,B
CLONE NAME: pDt 12, pDt 39
GENES: Lys 5 tRNA Ylocus
REF: 27

84B
INSERTION: 27P X/X-E
CROSS LIST: 67B, 87D12
REF: 200

84B1
BREAKPOINT: Df(3R)ASCB
CROSS LIST: 84B2
REF: 102

84B
CLONE NAME: DTA 1
GENES: delta tubulin
REF: 25

84B2
CLONE NAME: unn
GENES: Antp
BREAKPOINT: T(2;3)AntpNS+RC8, Df(3R)Scr
CROSS LIST: 41, 84A1
BREAKPOINT: In(3R)Antp LC, IN(3LR)AntpPW
In(3LR)Antp NS+RC4, Df(3R)JA99, In(3R)Antp73B
CROSS LIST: 91F-92A, 71F, 74F-75A, 84A1,2, 84D1,2
BREAKPOINT: Df(3R)ASCB, In(3R)Hu, Df(3R)Antp NS+RC7
In(3R)AntpB
CROSS LIST: 84B1, 84F2,3, 85F, 84D, 85E
REF: 102
84B2-C1

CLONE NAME: unn
GENES: hu
BREAKPOINT: In(3R)Hu
CROSS LIST: 84F2, 3, 86B4-C1

84B3-6

CLONE NAME: lambda Dm 2.55a
GENES: delta tubulin

84B3-C1,2

CLONE NAME: double sex
GENES: double sex

84BC

CLONE NAME: adm 123D11
GENES: delta tubulin
INSERTION: S3.8-1
CROSS LIST: 66D11-15, 87D12

84C

INSERTION: unn
CROSS LIST: 87D12

84C8

CLONE NAME: DmA 3a, 4a, 4b, 5a, 5b
GENES: glucose dehydrogen., eclosion genes A-D

84D

INSERTION: 28X-B
CROSS LIST: 67B, 87D
CLONE NAME: lambda 49-4
GENES: tRNA
CROSS LIST: 85C, 90C
INSERTION: unn
CROSS LIST: 67B-lacZ, 87D12
CLONE NAME: mDm 104H7
GENES: delta tubulin locus
CLONE NAME: pDt 78 RC
GENES: Val 3b tRNA locus
CLONE NAME: DTA 4
GENES: delta tubulin locus
BREAKPOINT: T(2;3)bxd DB6
CROSS LIST: 89E
INSERTION: 28X-B
CROSS LIST: 87D12, 67B
BREAKPOINT: Df(3R)Antp NS+RC7
CROSS LIST: 84B2

84D1,2

BREAKPOINT: In(3R)Antp 73b
CROSS LIST: 84B2

84D3,4

CLONE NAME: 1, 3, 6, 10
GENES: overlaps Val 3b tRNA

84D4-8

CLONE NAME: lambda Dm 5-1
GENES: delta tubulin
84E
CLONE NAME: IMP-E3
GENES: 20-hydroxyecdysone inducible
INSERTION: chB delta-194, g6:2
CROSS LIST: 87, lacZ, 87D12
INSERTION: g6:2
CROSS LIST: 68C3-5, 87D12

84E1,2
CLONE NAME:
GENES: double sex locus and flanking

84E11-12 - F4-5
CLONE NAME: unn
GENES:

84F
CLONE NAME: 14C4
GENES: cluster of arg and asn tRNAs
CLONE NAME: B34
GENES: maternal restricted transcript
BREAKPOINT: In(3)Mcp rv29175.10
CROSS LIST: 89E
INSERTION: 1
CROSS LIST: 88F
INSERTION: g71:3
CROSS LIST: 68C3-5, 87D12
INSERTION: A3
CROSS LIST: 99D, 87D12

84F2,3
CLONE NAME: unn
GENES:
BREAKPOINT: In(3R)Hu
CROSS LIST: 84B2-C1

85
CLONE NAME: adm 123B10
GENES:

85A
INSERTION: 27 X/A-1-A
CROSS LIST: 67B, 87D12
CLONE NAME: lambda 50-8
GENES: tRNA locus
INSERTION: R309.1
CROSS LIST: 87D12

85AB
INSERTION: 814, CH8
CROSS LIST: 99D, 87D12

85BC
INSERTION: 24-1
CROSS LIST: 3C11, 12; 87D

85C
CLONE NAME: lambda 49-4
GENES: tRNA
CROSS LIST: 84D, 90C
CLONE NAME: p85C
GENES: Arg tRNA
CLONE NAME: lambda m 1:2
GENES:

85D
INSERTION: P[(w,ry)H]3
CROSS LIST: 3C1,2; 87D
INSERTION: P(ry,HsAFP)5
CROSS LIST: 87D12
INSERTION: BS2.7-7
CROSS LIST: 66D11-15, 87D12
CLONE NAME: KV 1-22
GENES: delta tubulin locus

CLONE NAME: 542
GENES:
CLONE NAME: DHSV 7
GENES: RAS homologous
CLONE NAME:
GENES: delta 2 tubulin locus
INSERTION: g6:1
CROSS LIST: 68C3-5, 87D12

85D,E
CLONE NAME:
GENES: double sex cognate

85D6-12
CLONE NAME: DTB 4
GENES: delta tubulin locus

85E
CLONE NAME: M253
GENES: 0-1 hr.
CLONE NAME: DTA 3
GENES: delta tubulin
CLONE NAME: mDm 3008
GENES:
BREAKPOINT: In(3R)AntpB
CROSS LIST: 84B2

85E6-10
CLONE NAME: lambda Dm 5-22
GENES: delta tubulin locus

85E10-15
CLONE NAME: cDm51
GENES: metallothionein
CLONE NAME: lambda Dm13
GENES: metallothionein
CLONE NAME: lambda Dm13
GENES: metallothionein (Mtn)

85F
INSERTION: P[(w,ry)H]2-1
CROSS LIST: 3C1,2; 87D
INSERTION: tAP-15B,4.8
CROSS LIST: 35B2-3
BREAKPOINT: In(3R)Hu
CROSS LIST: 84B2
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86

CLONE NAME: adm 35E6
GENES: 
REF: 34

CLONE NAME: S35g
GENES: 
REF: 16

86A

INSERTION: 20-1
CROSS LIST: 3C11,12;87D
REF: 140

86B4-C1

BREAKPOINT: In(3R)Hu
CROSS LIST: 84B2-C1
REF: 50

86C

INSERTION: P[(w,ry)F]2
CROSS LIST: 3C1,2; 87D
REF: 176

86D

INSERTION: R311.1
CROSS LIST: 87D12
REF: 115

INSERTION: g7:6
CROSS LIST: 68C3-5,87D12
REF: 124

86E

INSERTION: S38Z-2
CROSS LIST: 7E11-7F1,2, lacZ,87D12
REF: 115

87A

CLONE NAME: GB
GENES: Hsp 70, Sn cell DNA
CLONE NAME: 56H8
GENES: hsp 70 and flanking
INSERTION: R307.1
CROSS LIST: 87D12
REF: 115

87A7

CLONE NAME: pPw 223
GENES: hsp 70
CLONE NAME: 
GENES: hsp 70 subclone
REF: 10

87AB

INSERTION: 28A-C
CROSS LIST: 67B, 87D
CLONE NAME: 540
GENES: 
REF: 16

INSERTION: 28A-C
CROSS LIST: 87D12,67B
REF: 121

87B

BREAKPOINT: In(3)Ubx 882
CROSS LIST: 89E
REF: 109

87C

CLONE NAME: G3
GENES: hsp 70, Sn cell DNA
REF: 42

87C1

CLONE NAME: 132E3
GENES: hsp 70 and flanking
REF: 41
87C1-3
BREAKPOINT: Df(3R)ry 81
CROSS LIST: 87E1,2
REF: 110

87C7-8
BREAKPOINT: Df(3R)kar SZ11
CROSS LIST: 87E1,2
REF: 110

87C10
CLONE NAME: pPW232, pPW229
GENES: hsp 70
REF: 10

87CD
INSERTION: C1-1
CROSS LIST: 3C1-2
REF: 113

87CF
CLONE NAME: adm 125G5
GENES:
CROSS LIST: 94D
REF: 34

87D
CLONE NAME: mG31
GENES: Hsc 70
CLONE NAME: unn
GENES: rosy ace, 1512
BREAKPOINT: In(3LR)3 ry64, In(3R)ry54, In(3R)ryPS11136
CROSS LIST: 64E, 81
REF: 42

87D1,2
BREAKPOINT: Df(3R)ry75
CROSS LIST: 87D14
REF: 144

87D2-4
BREAKPOINT: Df(3R)ry614, Df(3R)ry1301, Df(3R)ry1402
CROSS LIST: 87D14, 87E1,2
REF: 144

87D3,4
BREAKPOINT: Df(3R)ry 1607
CROSS LIST: 87E1,2
REF: 144

87D5-87E5
CLONE NAME: rosy and Ace
GENES: rosy and Ace
REF: 5

87D5,6
BREAKPOINT: Df(3R)ry 1608
CROSS LIST: 87E1,2
REF: 144

87D6-8
CLONE NAME: unn
GENES: rosy ace
BREAKPOINT: Df(3R)ry 619
CROSS LIST: 87E12,F1
REF: 144

87D8-10
CLONE NAME: unn
GENES: rosy ace
BREAKPOINT: Df+In(3R)kar lg27
CROSS LIST: 99E1-F1
REF: 144
87D12-13

CLONE NAME: unn
GENES: rosy ace
CROSS LIST: see Table 2
BREAKPOINT: T(3;4)ryP51149
CROSS LIST: 4th het

87D12-14

BREAKPOINT: In(3)Cbx rv21988B
CROSS LIST: 89

87D14

CLONE NAME: unn
GENES: rosy ace
BREAKPOINT: Df(3R)ry75
CROSS LIST: 87D12, 87D2,4

87D17

CLONE NAME: unn
GENES: rosy ace

87E

INSERTION: unn
CROSS LIST: 87D12
CLONE NAME: unn
GENES:
INSERTION: unn
CROSS LIST: 87D12

87E1,2

CLONE NAME: unn
GENES: rosy ace
BREAKPOINT: Df(3R)ry 81, Df(3R)karSZ11,In(3)Cbx+R1
Df(3R)ry1402, Df(3R)ry1301
Df(3R)ry1607, Df(3R)ry1608
CROSS LIST: 87C1,3, 87C7,8, 89E, 87D2,4, 87D3,4, 87D5,6
BREAKPOINT: Df(3R)126c
CROSS LIST: 87F11,12

87E5,6

BREAKPOINT: Df(3R)1C4a
CROSS LIST: 87E11,F1

87E11,F1

BREAKPOINT: Df(3R)1C4a
CROSS LIST: 87E5,6

87E12,F1

BREAKPOINT: Df(3R)ry 619
CROSS LIST: 87D6,8

87E-F

BREAKPOINT: In(3)Cbx rv21987A
CROSS LIST: 89
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87F
BREAKPOINT: In(3)Cbx wt
CROSS LIST: 89E
BREAKPOINT: T(2;3)Cbx rvR17.6F
CROSS LIST: 89E
INSERTION: R308.2, R404.1
CROSS LIST: 87D12
INSERTION: R404.1
CROSS LIST: 87D12
INSERTION: DRI-15
CROSS LIST: 87D12, 37C1, 2

87F11,12
BREAKPOINT: Df(3R)126c
CROSS LIST: 87E12

87F-88A
BREAKPOINT: In(3)Ubx80
CROSS LIST: 89E

88
CLONE NAME: S32
GENES:

88A
INSERTION: cHB delta-23
CROSS LIST: 87-lacZ, 87D12

88B
CLONE NAME: adm 88B.1
GENES: minor heat shock cDNA
BREAKPOINT: In(e)Ubx 12.5 (Madrid)
CROSS LIST: 89E

88C
CLONE NAME: mDm 104D12
GENES:
INSERTION: S6.9-5
CROSS LIST: 66D11-15, lacZ, 87D12

88D
CLONE NAME: double sex cognate

88E
CLONE NAME: mG34
GENES: hsc 70
INSERTION: BS2.7-9, R401.2
CROSS LIST: 66D11-15, 87D12/ 87D12

88F
CLONE NAME: muscle specific tropomyosin
CLONE NAME: actin
CLONE NAME: unn

REF: 110
REF: 110
REF: 115
REF: 115
REF: 114
REF: 110
REF: 16
REF: 118
REF: 39
REF: 110
REF: 1
REF: 115
REF: 110
REF: 88
REF: 42
REF: 115
REF: 169
REF: 170
REF: 11
GENES: actin
CLONE NAME: lambda dmpt 73
GENES: tropomyosin
INSERTION: cp70 delta B
CROSS LIST: 87-lacZ, 87D12
INSERTION: S6.9-1
CROSS LIST: 66D11-15, lacZ, 87D12

88F2-5
CLONE NAME: lambda DM 85
GENES: 3 tropomyosin loci

89
CLONE NAME: unn
GENES: pic
BREAKPOINT: In(3)Cbx rv21987A, In(3)Cbx rv21988B
CROSS LIST: 87E-F, 87D12-14

89A-B
BREAKPOINT: In(3)bdx27830.C5A
CROSS LIST: 89E

89A
BREAKPOINT: In(3)Cbx 3 (Cbx-like)
CROSS LIST: 89E
INSERTION: +204
CROSS LIST: 87, 87D12
INSERTION: Bl-1
CROSS LIST: 3C1-2

89B
INSERTION: 28-2
CROSS LIST: 3C11, 12, 87D
INSERTION: 27P X/X-C
CROSS LIST: 67B, 87D12
CLONE NAME: pDd 14
GENES: Val 4 Phe 2 tRNA
BREAKPOINT: In(3)Camel
CROSS LIST: 89E
INSERTION: BS2.7-6
CROSS LIST: 66D11-15, 87D12
INSERTION: g7:8
CROSS LIST: 68C3-5, 87D12

89B4
CLONE NAME: sb
GENES: sb

89B21
BREAKPOINT: T(1;3)sta
CROSS LIST: 2B3, 4

89BC
CLONE NAME: EV27
GENES: EV27

89C
BREAKPOINT: In(3)bdx 183
CROSS LIST: 89E
89C1,2

BREAKPOINT: T(2;3)P10
CROSS LIST: 89E

REF: 110

89E

BREAKPOINT: T(2;3)bxdB23, T(2;3) P75Ubx5T17.14-17
T(2;3) CbxrvR17.6F, T(2;3) UbxX6000.78A4
T(2;3) Ubx105

CROSS LIST: 41A, 44C4, 51E, 52A-C, 53C
BREAKPOINT: In(3) 89E/97F-97A, Cbx rv21560.60
Tp(3) Vno, Tp(3) abx, In(3) Ubx
In(3) Mcp rvB315

CROSS LIST: 90A

BREAKPOINT: T(2;3) Mcp rvC1 complex, T(2;3) bxd29315.46,
Tp(3) bxd100, Tp(3) Ubx2P20, Tp(3) Ubx7P20,
Tp(3) P47, In(3LR) bxd113, In(3LR) CbxrvR17.42

BREAKPOINT: In(3) Ubx 882

CROSS LIST: 87B

BREAKPOINT: In(3) Ubx 130 (TM2) complex
CROSS LIST: 74

BREAKPOINT: In(3) Cbx rvR17.5E, Tp(3) P47,
In(3) Ubx130 (TM2) complex, Tp(e) Vno,
Tp(3) McpB277, In(3) Ubx19286.76

CROSS LIST: 96F-97A, 96F-97A, 97CD, 97D, 98D-F
BREAKPOINT: In(3LR) Ubx130 TM2 complex
In(3LR) bxd106, In(3LR) bxd19409.2x

CROSS LIST: 74, 72D11-72E1, 80F
BREAKPOINT: In(3) Hab rvFC51, In(3) Ubx125,
In(3) Mcp rvC1 complex, In(3LR) Cbx rvR17.44V
In(3LR) Cbx rvR17.16R

CROSS LIST: 81, 79C-E, 80B, 84D
BREAKPOINT: In(3) bxd27830.5CA, In(3) Ubx5.12 Madrid,
T(1;3) bxd111, In(3) Camel, In(3) bxd183
In(e) 54A62 (szedged)

CROSS LIST: 89A, B, 90A, 90B2, 89B, 89C, 88C4
BREAKPOINT: In(3LR) 89/75+T(2;3) 54/75, Ubx 6-26 Madrid
T(1;3) Ubx21560.8A Complx, T(1;3) Uab5
T(1;3) bxd111, Dp(3;1) P68, T(1;3) P115

CROSS LIST: 54E, 5B, 1F, 4D, 20
BREAKPOINT: In(3) Ubx 1928.16N, T(3;4) bxd101,
T(3;4) UbxA complex, T(3;4) Cbx rv R17.40R

CROSS LIST: 99, 101F, 102, 4
BREAKPOINT: T(2;3) CbxrvR17.34, T(2;3) rvR17.22x
T(2;3) bxd22044D, 5(2;3) Ubx16160.36
T(y;2;3) Mcpvc10 complex, T(2;3) bxd x22920.11x

CROSS LIST: 41A, 41F, 44A, 42BC
BREAKPOINT: In(3) Cbx +R1
CROSS LIST: 87E1, 2
BREAKPOINT: T(2;3) P10, In(3) Ubx961.29, In(3) Tab,
In(3) Ubx3966.30, In(3) cbx2, Tp(3) bxd110,
Tp(3) bxd110

CROSS LIST: 89C1,2, 90C, 90E, 91B, 91C, 91D1,2, 92A1,2
BREAKPOINT: T(2;3) bxd36 complex, T(2;3) Ubx18136.147
T(2;3) Ubx19286.8m, T(2;3) Ubx17756.180
T(2;3) Ubx19649.18, T(2;3) Ubx1

CROSS LIST: 2Lt1p, 41A, 39
BREAKPOINT: T(2;3) Cbx rvR17.6F
CROSS LIST: 87F
BREAKPOINT: In(3) Sab Mcp rv29340.8
CROSS LIST: 90A
BREAKPOINT: T(1;3)CbxrvRl7.49A, T(2;3)Ubx16160.18  REF: 110
T(2;3)Ubx18264.1, T(2;3)Ubx4.3, T(2;3)Ubx4.3 Mø
T(2;3)CbxrvRl7.175, T(2;3)Hm complx, T(2;3)Pl0
CROSS LIST: 20, 31, 21L12, 34, 32, 29A-C, 22B1, 2, 2L tip
BREAKPOINT: In(3)Mcp rv29175.10, In(3)Cbx wt
In(3)Ubx80, In(e)Ubx12.5 Madrid,
In(3)Cbx3 (Cbx-like)
CROSS LIST: 84F, 87F, 87F-88A, 88B, 89A
CLONE NAME: unn  REF: 110
GENES:
BREAKPOINT: T(2;3) Mcp rv26105A
CROSS LIST: 60B

89E1-4
CLONE NAME: unn  REF: 18
GENES: bithorax complex
BREAKPOINT: T(1;3)Uab5, T(1;3)bxd111,
T(1;3)Ubx21560.8A complex
CROSS LIST: 1F, 4D, 5B

89F
BREAKPOINT: T(1;3) P115, Dp(3;1) P68
CROSS LIST: 20

90A
BREAKPOINT: In(3)Ubx 5.12 (madrid)
In(3)Sab Mcp rv29340.8
CROSS LIST: 89E, 89E

90B2
BREAKPOINT: T(1;3)bxd 111
CROSS LIST: 89E

90BC
CLONE NAME: pkdm 7E5  REF: 34
GENES: Intermolt V RNA, sgs
CLONE NAME: pDt 48  REF: 27
GENES: val 3b, pro tRNA
CLONE NAME: lambda bDm 1508  REF: 24
GENES:
CLONE NAME: pDt 92RC, pDt 120RC, pDt 41 RC4
GENES: Val tRNA, val 4 tRNA, Cal 3b, pro tRNA

90C
CLONE NAME: lambda 49-4  REF: 58
GENES: tRNA
CROSS LIST: 84D, 85C
BREAKPOINT: In(3)Ubx 961.29
CROSS LIST: 89E

90CD
INSERTION: S3.8-2  REF: 115
CROSS LIST: 66D11-15, 87D12

90E
INSERTION: P[(w,ry)D]3  REF: 176
CROSS LIST: 3Cl, 2; 87D
BREAKPOINT: In(3)Tab
CROSS LIST: 89E
90EF
INSERTION: DR-1
CROSS LIST: 87D12,37C1,2

91
CLONE NAME: S24
GENES:

91AB
INSERTION: AR4-01
CROSS LIST: 3C1,2

91B
BREAKPOINT: In(3)Ubx 3966.30
CROSS LIST: 89E
INSERTION: chB delta-194
CROSS LIST: 87-lacZ,87D12

91C
CLONE NAME: lambda 39-1
GENES:
CROSS LIST: 27D, 43A
BREAKPOINT: In(3)cbx 2
CROSS LIST: 89E
INSERTION: A2-1
CROSS LIST: 3C1-2,87D12

91D
CLONE NAME: mDm 103G4
GENES:
INSERTION: g4:2
CROSS LIST: 68C3-5,87D12

91D1,2
BREAKPOINT: Tp(3) bxd 110
CROSS LIST: 89E

91F-92A
BREAKPOINT: In(3R)Antp LC
CROSS LIST: 84B2
INSERTION: S3.8-5
CROSS LIST: 66D11-15,87D12

92
BREAKPOINT: Tp(3)P47
CROSS LIST: 89E
CLONE NAME: S12g
GENES:

92A
BREAKPOINT: In(3) Cbx rvR17.5E
CROSS LIST: 89E
CLONE NAME: mDm 101F8
GENES:
INSERTION: R3.9-2
CROSS LIST: 66D11-15,87D12

92A1,2
BREAKPOINT: Tp(3) bxd 110
CROSS LIST: 89E
92A2-3
CLONE NAME: HP-clones
GENES: delta
BREAKPOINT: T(3.3) nII13

92B
INSERTION: AR4-032(111);AR4-01(111)
CROSS LIST: 3C1,2

92B8-11
CLONE NAME: DmRh1
GENES: opsin

92BC
INSERTION: H4
CROSS LIST: 87D12
INSERTION: B2-1
CROSS LIST: 3C1-2

92CD
CLONE NAME: 512
GENES: head specific RNA

92E
CLONE NAME: adm 124B10
GENES:

92F
INSERTION: BS2.7-2
CROSS LIST: 66D11-15,87D12

93AB
INSERTION: R310.1
CROSS LIST: 87D12

93B
BREAKPOINT: In(3)Ubx 130 (TM2) complex
CROSS LIST: 89E
CLONE NAME: unn
GENES:

93CD
CLONE NAME: unn
GENES: ebony, 93D heat shock locus

93D
CLONE NAME: cos7, p5A, cDm El
GENES: heat-shock
INSERTION: SB2.1-4
CROSS LIST: 66D11-15,87D12
CLONE NAME: adm 129F5
GENES: heat shock

93D6,7
INSERTION: g5:1
CROSS LIST: 68C3-5,87D12
94A
CLONE NAME: adm 134C5, 135D2
GENES:
BREAKPOINT: Tp(3)Vno, Tp(3)McpB277
CROSS LIST: 89E

94B
INSERTION: 28P-D
CROSS LIST: 67B, 87D
INSERTION: 28P-D
CROSS LIST: 87D12, 37C1, 2

94D
INSERTION: P[(w,ry)E]7
CROSS LIST: 3C1, 2; 87D
CLONE NAME: adm 125G5
GENES:
CROSS LIST: 87CF

94E
CLONE NAME: lambda dmpt 123
GENES:
INSERTION: AR4-020
CROSS LIST: 3C1, 2

94F
CLONE NAME: 0018
GENES: oocyte RNA

95A
INSERTION: R601.1
CROSS LIST: 87D12

95A-B
CLONE NAME:
GENES: en-like homeobox
CROSS LIST: 58C

95AB
INSERTION: 27S-C
CROSS LIST: 67B, 87D12

95B
CLONE NAME: mDm 108E11
GENES:

95C
CLONE NAME: pDm6A65
GENES: U1 snRNA
CROSS LIST: 82E, 21D
CLONE NAME: 156-1(lambda)
GENES: blastoderm-differential poly(A) RNA
INSERTION: D1
CROSS LIST: 99D, 87D12

95D
INSERTION: 27S-A
CROSS LIST: 67B, 87D12
CLONE NAME: pFW227, lambda 15
GENES: hsp 68

REF: 34
REF: 110
REF: 180
REF: 121
REF: 176
REF: 34
REF: 58
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REF: 41
REF: 115
REF: 208
REF: 200
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REF: 47
REF: 119
REF: 10
REF: 200
REF: 10
INSERTION: BS2.7-8
CROSS LIST: 66D11-15, 87D12
INSERTION: unn
CROSS LIST: 87D12

REF: 115
REF: 117

INSERTION: 28A-A
CROSS LIST: 67B, 87D
INSERTION: 28A-A
CROSS LIST: 87D12, 67B

REF: 180
REF: 121

INSERTION: DR-2
CROSS LIST: 87D12, 37C1,2

REF: 114

BREAKPOINT: In(3)Ubx 19286.76
CROSS LIST: 89E
CLONE NAME: adm 137A2
GENES:

REF: 110
REF: 34

INSERTION: P[(w,ry)G]3
CROSS LIST: 3C1,2; 87D

REF: 176

INSERTION: S3.8-1
CROSS LIST: 66D11-15, 87D12
INSERTION: S38M-3
CROSS LIST: 7E11-7F1,2,87D12

REF: 116
REF: 115

CLONE NAME: mDm 107D4
GENES:

REF: 1

BREAKPOINT: Tp(3)Mcp B277
CROSS LIST: 89E

REF: 110

BREAKPOINT: Tp(3)Vno
CROSS LIST: In(3)89E/97F-97A, Cbx rv21560.60
CLONE NAME: adm 126D12
GENES:

REF: 110
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CLONE NAME: adm 132C4, 132E7, 132H4
GENES:

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INSERTION: 28N
CROSS LIST: 67B, 87D12
INSERTION: tAP-16,4.8
CROSS LIST: 35B2-3
CLONE NAME: lambda 50
GENES:

REF: 200
REF: 112
REF: 58
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97AB
INSERTION: +65  REF: 118
CROSS LIST: 87, 87D12
INSERTION: 28-term  REF: 121
CROSS LIST: 87D12, 67B

97B
INSERTION: P[(w,ry)F]4-3  REF: 176
CROSS LIST: 3C1,2; 87D
INSERTION: 28P-B  REF: 180
CROSS LIST: 67B, 87D
INSERTION: 28P-B  REF: 121
CROSS LIST: 87D12, 67B

97C
CLONE NAME: adm 140F12  REF: 34
GENES:
CROSS LIST: 62AB

97CD
BREAKPOINT: In(3)Ubx 3798.68  REF: 110
CROSS LIST: 89E

97D
BREAKPOINT: Tp(3)abx  REF: 110
CROSS LIST: 89E

97D1,2
CLONE NAME:
GENES: toll  REF: 203

97EF
CLONE NAME: DTB1  REF: 25
GENES: delta tubulin

97F
INSERTION: unn  REF: 117
CROSS LIST: 87D12
CLONE NAME: KV 3-12  REF: 22
GENES: delta tubulin

98
CLONE NAME: L2  REF: 16
GENES:
CROSS LIST: 99

98 (+ or -)
BREAKPOINT: T(1;3)scKA8  REF: 105
CROSS LIST: 1B4,5

98A
INSERTION: BS.27-1  REF: 115
CROSS LIST: 66D11-15, 87D12

98B-C
BREAKPOINT: In(3)Ubx-x  REF: 110
CROSS LIST: 89E

98B
INSERTION: cHB delta-89  REF: 118
CROSS LIST: 87-lacZ, 87D12
98C
INSERTION: R602.1, R705.1
CROSS LIST: 87D12
REF: 115

98D-F
BREAKPOINT: In(3)Mcp rvl315
CROSS LIST: 89E
REF: 110

98E
CLONE NAME: B8
GENES: Maternal restricted transcript
REF: 69

98F
CLONE NAME: b11
GENES: maternal RNA
CLONE NAME: myosin alkali light chain
CLONE NAME: M55
GENES: 0-2.5 hrs.
REF: 84

98F1,2
BREAKPOINT: Tp(3;3)DfdTRX1
CROSS LIST: 83D4,5
REF: 69

99
CLONE NAME: L2
GENES:
CROSS LIST: 98
BREAKPOINT: In(3)Ubx 1928.16N
CROSS LIST: 89E
REF: 110

99A
INSERTION: g6:3
CROSS LIST: 68C3-5, 87D12
REF: 124

99AB
INSERTION: DA24-44
CROSS LIST: 35B2-3, 37C1,2
REF: 114

99B
CLONE NAME:
GENES: Homeo box
REF: 50

99C
CLONE NAME: 559
GENES: Head specific RNA
REF: 31

99C5-6
CLONE NAME: 559
GENES: transient receptor potential
REF: 68

99CF
CLONE NAME: adm 129B8
GENES:
REF: 34

99D
CLONE NAME: EH8
GENES: serendipity alpha, beta, delta
INSERTION: SRS3.9-3
CROSS LIST: 66D11-15, 87D12
CLONE NAME: rpro 49
GENES: ribosomal protein, Minute
REF: 53
REF: 115
REF: 142
CLONE NAME: 153-1 (lambda) REF: 47
GENES: blastoderm-specific poly(A) RNA

99E
CLONE NAME: REF: 47
GENES: myosin light chain 2
CLONE NAME: dmpT57 REF: 
GENES: myosin light chain-2
INSERTION: B 25 REF: 134
CROSS LIST: 3C1-2
CLONE NAME: lambda Dm 11-9 REF: 22
GENES: myosin light chain
CLONE NAME: adm 132G9 REF: 34
GENES:

99E1-3
CLONE NAME: 36-1 (lambda) REF: 47
GENES: blastoderm differential poly(A)

99E1-F1
BREAKPOINT: Df+In(3R)kar 1g27 REF: 110
CROSS LIST: 87D8-10

99F
CLONE NAME: adm 142D9 REF: 34
GENES:

100
CLONE NAME: S2 REF: 16
GENES:

100AB
CLONE NAME: 5D7 REF: 16
GENES:

100B
CLONE NAME: DM 24B10 REF: 150
GENES: Ag24B10
CLONE NAME: mDm 103 F1 REF: 1
GENES:
CLONE NAME: lambda dmpt 31 REF: 58
GENES:
CLONE NAME: 516 REF: 31
GENES: head specific RNA

100C
CLONE NAME: double sex cognate REF: 88
GENES:

100C1-7
CLONE NAME: mDm 102A3 REF: 1
GENES:

100CD
CLONE NAME: 11B2 REF: 94
GENES: cell death-5 locus

100D
INSERTION: unn REF: 117
CROSS LIST: 87D12
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CLONE NAME: mDm 105 H1
GENES:
REF: 1

100E
CLONE NAME: Dm-17
GENES: F2, pupal mRNA in both males and females
CROSS LIST: homology to F1 at 48D
REF: 188

100F
CLONE NAME: lambda T-A
GENES: telomeres
CROSS LIST: 1A, 21A, 60F, 61A, 102F
CLONE NAME: lambda 5c, lm.R15, lm.gt11.20516
GENES: microtubule associated protein locus
INSERTION: A4-4
CROSS LIST: 3C1-2, 87D12
REF: 83
REF: 113

100G
BREAKPOINT: T(3;4)ryP51149
CROSS LIST: 87D12-13
BREAKPOINT: T(3;4)Cbx rvR17.40R
CROSS LIST: 89E
REF: 110
REF: 110

101-102
BREAKPOINT: T(1;4)scH
CROSS LIST: 1B4-C6
REF: 105

101F
BREAKPOINT: T(3;4)bxdl101
CROSS LIST: 89E
REF: 110

102
BREAKPOINT: T(3;4)Ubx A complex
CROSS LIST: 89E
BREAKPOINT: T(1;4)JC43
CROSS LIST: 3B1,2
REF: 110
REF: 107

102C
CLONE NAME: mDm 108D1
GENES:
REF: 1

102CD
CLONE NAME: 116H2
GENES:
REF: 3

102EF
CLONE NAME: lambda dmpt101
GENES:
REF: 58

102F
CLONE NAME: lambda T-A
GENES: telomeres
CROSS LIST: 1A, 21A, 60F, 61A, 100F
REF: 156
### TABLE 1: OTHER INFORMATION ON CLONES

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REFERENCES

4 D. Cavener, Vanderbilt U, Nashville TN 37235.
5 Bender, Spierer & Hogness, J Mol Biol 168:17-33.
7 S. Henikoff, J.A. Sloan & J.D. Kelly, Hutchinson Cancer Res Ctr, Seattle WA 98104.
12 S.L. Tobin, Dept Mol Biol, U of Oklahoma, Hlth Sci Ctr, PO Box 26901, Oklahoma City OK 73190.
14 Poole et al. 1985, Cell 40:37.
19 S. Kerridge & R. Griffeen-Shea, LGBC CERS Case 907, Univ Luminy, 70 Route Leon Lachamp, 13288 Marseille, France.
20 E.L. George & C.P. Emerson Jr., Biol Dept, U of Virginia, Charlottesville VA 22901.
23 J.E. Natsle & J.W. Fristrom, Dept Genetics, U of Calif, Berkeley, CA 94720.
30 D.I. Horowicz, R. Howard, P. Ingham, A. Leigh-Brown & S. Pinchin, Imperial Cancer Research Fund, Burton Hole Lane, Mill Hill, London UK NW7 1AD.
35 J.W. Fristrom & D. Kimble, Dept Genetics, U of Calif, Berkeley CA 94720.
36 band 11A cut out of chrom. by tech of Scalangle, Tunco, Edstrom, Pirotta & Melli 1981, Chrom. 82:205.
38 C.V. Cabrera, J. Casal & A. Ferrus, Centro de Biol Mol, Consejo Sup. de Invest, Cint. & Univ Auto de Madrid, Spain.
41 R. Devlin & V. Finerty, Biol Dept, Emory U, Atlanta GA 30322.
43 J.E. Natsle & J.W. Fristrom, Dept Genetics, U of Calif, Berkeley, CA 94720.
45 S. Hayashi, D.L. Cribbs, J.A. Sloan & J.D. Kelly, Hutchinson Cancer Res Ctr, Seattle WA 98104.
49 J.W. Fristrom & D. Kimble, Dept Genetics, U of Calif, Berkeley CA 94720.
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52 C.V. Cabrera, J. Casal & A. Ferrus, Centro de Biol Mol, Consejo Sup. de Invest, Cint. & Univ Auto de Madrid, Spain.
55 R. Devlin & V. Finerty, Biol Dept, Emory U, Atlanta GA 30322.
57 J.E. Natsle & J.W. Fristrom, Dept Genetics, U of Calif, Berkeley, CA 94720.
59 S. Hayashi, D.L. Cribbs, J.A. Sloan & J.D. Kelly, Hutchinson Cancer Res Ctr, Seattle WA 98104.
61 S. Hayashi, D.L. Cribbs, J.A. Sloan & J.D. Kelly, Hutchinson Cancer Res Ctr, Seattle WA 98104.


Livak 1984, Genetics 107:611.

Pirrotta et al. 1983, EMBOJ 2:927.


E. Stephenson, Biol Dept, U of Rochester, Rochester NY 14627.


Renkawitz-Pohl & Bialojan 1984, Chromosoma 89:206; Bialojan et al., EMBOJ in press.


P. Adler & B. Brunl, Genetcs Lab, U of Wisconsin, Madison WI 53706.

W. Eggleson, Genetics Lab, U of Wisconsin, Madison WI 53706.


Henikoff, Sloan & Kelly 1983, Cell 34:405-414.

J. Belote, B. Baker, Biol Dept, U of Calif-San Diego, La Jolla CA 92093.

D. Andrew & B. Baker, Biol Dept, U of Calif-San Diego, La Jolla CA 92093.

T. Kaufman, Biol Dept, Indiana U, Bloomington IN 47401.

R. Brodberg & W. Pak, Genetics, U of Calif, Davis CA 95616 & Biol Sci, Purdue U, W. Lafayette IN 47907.

D. Sponaugle & Woods, Dev Biol Ctr, U of Calif, Irvine CA 92717.

H. Biesmann, Biochem & Biophys, U Calif, San Francisco CA 94143.


C. Dearolf, Biology, John Hopkins U, Baltimore MD 21218.

M. Goldberg, Genetcs & Devel, Cornell U, Ithaca NY 14850.


W. Bender- BXC rearrangements from E. Lewis at Cal Tech (except when designated Madrid).

E. Meyerowitz, Biol Dept, Calif Inst Tech, Pasadena CA 91125.


W. Gehring, Gehring et al. 1984, EMBOJ 3:2077.

E. Hoffman & V. Corces, Biology, John Hopkins U, Baltimore MD 21218.


G. Mark, R. MacIntyre & M. Digan, (in prep.).


Chia, Karp, McGill & Ashburner, J Molecular Biology (in press).

Pirotta, Devel Biol 86:438.

Hoffmann, Korge (in prep.).


Levis (unpubl.), Biochem Dept, U of Calif, Berkeley CA 94720.

T. Laverty (unpubl.), Biochem Dept, U of Calif, Berkeley CA 94720.

Nash (unpubl.).

von Kalm (unpubl.).

Krumm, Roth & Korge 1985, PNAS USA 82:5055-5059.

Rancourt, Walker & Davies, unpublished.


Gubb, Shelton, Roote, McGill & Ashburner 1984, Chromosoma (Berl) 91:54-64.


Parker, Falkenthal & Davidson 1985, Mol & Cell Biology 3058-3068.

Young, Pession, Traverse, French & Pardue 1983, Cell 34:85-94.


Zuker, Cowman & Rubin 1985, Cell 40:851-858.


Brock (unpubl.), Biol Dept, U of Brit Columbia, 6270 University Blvd, Vancouver BC Canada.


Mariani, Pirrotta & Manet 1985, EMBOJ 4(8):2045-2052.


Karl1k & Fyberg 1985, Cell 41:57-65.


Hafner et al. (unpubl.).

Goralski, Konrad & Mahowald (unpubl.).

Alit-Ahmed (unpubl.).


W. Gehring, Gehring et al. 1984, EMBOJ 3:2077.

E. Hoffman & V. Corces, Biology, John Hopkins U, Baltimore MD 21218.


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<td>184</td>
<td>Blessmann, Biochem Dept, U of Calif, San Francisco CA 94143.</td>
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<tr>
<td>185</td>
<td>Stellar (unpubl.).</td>
</tr>
<tr>
<td>186</td>
<td>Walker et al., MGG (January 1986).</td>
</tr>
<tr>
<td>187</td>
<td>Levine (unpubl.).</td>
</tr>
<tr>
<td>188</td>
<td>Walldorf, Hovemann &amp; Bautz 1985, PNAS 82:5795-5799.</td>
</tr>
<tr>
<td>189</td>
<td>Gelbart (personal communication).</td>
</tr>
<tr>
<td>193</td>
<td>Schedl &amp; Donelson 1978, Biochimica et Biophysica Acta 520:539-554.</td>
</tr>
<tr>
<td>197</td>
<td>Howells (in prep.).</td>
</tr>
<tr>
<td>199</td>
<td>Qian (personal communication).</td>
</tr>
<tr>
<td>200</td>
<td>Hoffman &amp; Corces, EMBOJ (submitted).</td>
</tr>
<tr>
<td>203</td>
<td>Hudson &amp; Anderson (personal communication).</td>
</tr>
<tr>
<td>204</td>
<td>Champe &amp; Laird (personal communication).</td>
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<tr>
<td>205</td>
<td>Garbe &amp; Pardue (personal communication).</td>
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<tr>
<td>206</td>
<td>Campos, Rosen &amp; White (personal communication).</td>
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<tr>
<td>207</td>
<td>Hammonds &amp; Fristrom (personal communication).</td>
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<tr>
<td>208</td>
<td>Gustafson (personal communication).</td>
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<td>209</td>
<td>Phillips &amp; Shearn (personal communication).</td>
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<tr>
<td>210</td>
<td>Graziani et al. (personal communication).</td>
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<tr>
<td>211</td>
<td>Johnson, Friedman &amp; Edstrom (personal communication).</td>
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<td>212</td>
<td>Qian &amp; Jacobs-Lorena (personal communication).</td>
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<td>213</td>
<td>Doyle et al. (personal communication).</td>
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<td>214</td>
<td>Williams &amp; Bell (personal communication).</td>
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<tr>
<td>215</td>
<td>Butler et al. (personal communication).</td>
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<tr>
<td>216</td>
<td>Glew et al., Gene (in press).</td>
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<tr>
<td>221</td>
<td>Lepesant et al. 1986, EMBOJ 5:583-588.</td>
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<td>Jacobs-Lorena (personal communication).</td>
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Please return completed forms to:

Dr. John Merriam
Department of Biology
University of California
Los Angeles, CA 90024

BITNET ADDRESS:  
IKNONEO@UCLA.MVS

Use this form to report new information on clones, breakpoints known within a cloned sequence, and transformed inserts.

I. Report on Cloned DNA

A. Chromosome Location:

B. Clone Name:

C. Genes in Clone:

D. Other information (strain, size, DNA sequence, restriction map references, misc.):

E. Literature Reference to Clone:

II. Report on Locating Rearrangement Breakpoints

A. Chromosome Location (of previously reported clone):

B. Rearrangement name:

C. Additional Rearrangement Breakpoints (not in clone):

D. New Order (if known):

E. Literature reference to mapping rearrangement within clone:

III. Report on Location of Transformation Inserts

A. List Transformant Name, Insertion sites, and Markers present: Denote gene name and allele symbol (whether + or altered): <continue on back if necessary.>

B. Literature reference to Transformation:

IV. Lab Information: (Names, Lab Address, and Phone #)

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