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Meyer, Helen U. and Michael L. Criswell. Crossover analysis of sex-linked mutations induced in oogonial cells by repeated treatments with 4000r of X-ray

Meyer, Helen U. and Evelyn R. Meyer. Sperm utilization from successive copulations in females of D. melanogaster

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Parker, D. An apparent incompatibility among seemingly normal members of the species D. simulans

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Strangio, V. A. Radiosensitivity to certain breakage aberrations during spermatogenesis in D. melanogaster


Terzaghi, Eric and E. Novitski. An attempt to produce fertile "transformed" males

Tokunaga, Chiyoko. Notes on the sex chromosome constitution of oogonial cells in gynanders

Wolff, M. and A. Coughlin. Tests for meiotic drive in interspecific hybrids

Wurgler, Friedrich E. Modification of X-ray induced embryonic mortality by different anoxia conditions before and during irradiation of uncleaved D. melanogaster eggs

Zimmering, S. and H. J. Muller. Studies on the action of the dominant female-lethal F1 and of a seemingly less extreme allele, F1s

Technical Notes

Browning, Luolin S. and Edgar Altenburg. Weighting of dehydrated Drosophila as a counting method

Mickey, G. H. Nigrosine as an aid for staining brain and salivary gland chromosomes


Personal and Laboratory News

Materials Requested or Available

Announcements
Wild Stocks

1 Austin, Texas
2 Canton-S
3 Espanola, New Mexico
4 Oregon-R

Chromosome 1

amx/In(1)dl-49, m²g
amx 1z²v/y f:=
Bx² ma-1
1(1)7dl-49, y Hw m²g
1(1)7e, 1(1)7dl-49, y Hw
1(1) 55a B/In(1)sc², S,
w²/y sc²
l Z²v/y f:=
m
m a 1
p n²
Qd
sc cho
sc eq 1g²v g f/FM3,
y²sc²dm B l
sn 1z²v/y f:=
sl n m a 1
su²/s n²
v 58k
v m
w m
w²sn²
w a rb
w a²sp ec
w b f²
w bf²
w b l
w b²sp sn²/y f:=
w B b x²a sp sn²/y f:=
w c w a²sp sn³/y f:=
w e w sp sn³
w
w

Chromosome 2

al dp b px bl³ by²/
Cy, al²lt² L² sp²
al dp b pr c px sp/Cy
al S ast ho/Cy³k E-S
al ²ho/Pm dp²
ast³ dp cl
b
b pr c px sp
b vg
Bla/SM5, al²lt² Cy sp²
Ba/Ig (2LR), dp
B L²/SM5
br²
b s
b w
b w ba
cn b w

Attached X-Y

V f B X-Y/x² su-w² a²wb
X Y³ y² y²,( 3d/v f² y²)² X Y² y² y² ( 108-9 Parker) y² su-w² a²
(bb²)² y² y²/bb O/V f B XY; pol
y Y² X² In(1)EN -49, y v f car/
y² a²bb² O

Inversions

In(1)FM6, FM6, Yu² In(1)EN -49, y v f car/

Modified Y

Yb²
y²/y² sc² Y
y²f²/y² sc² v f:=
s c² y²/In(1)dl-49, y v f y²

Yc²/y² B²/y² f:=
Y² y² ct² f²/y² f:=
Y² y² #²/y² v f Y²/y² f:=

Y Mu l-5, In(1)sc² S, y w² B sc²
y v Muller 5, In(1)sc² S, y w² v B sc²
In(1)y², 1z²v/y v car

Attached Y

Y²
y²/y² sc² .Y
Y³/y² sc² Y
Y²f²/y² sc² v f:=
s c² y²/In(1)dl-49, y v f y²

Yc²/y² B²/y² f:=
Y² y² ct² f²/y² f:=
Y² y² #²/y² v f Y²/y² f:=

Y Mu l-5, In(1)sc² S, y w² B sc²
y v Muller 5, In(1)sc² S, y w² v B sc²
In(1)y², 1z²v/y v car
July 1961

Melanogaster - Stocks - Austin

**Duplications**

\[ \text{Dw}^{51b7} \]

**Translocations**

\[
T(1;4)_{E}^{S} y \quad \text{bw; e; cl ey}^{R} \\
T(1;4)_{N}^{m} \quad \text{p' /In(1)} \quad -49, \ w \ 1z^{s} \\
T(1;4)_{N}^{m} \quad \text{258} \quad -18, \ D \\
T(1;4)_{N}^{m} \quad \text{258} \quad -21, \ ey \\
T(1;4)_{N}^{m} \quad \text{w/Ins(1)} \quad -49, \ \text{sc}^{8}, \ y^{31d} \\
\]

**Triploid**

\[ y^{2} \quad \text{sc}^{8}, \ y^{31d} \]

**Note:** Additions and corrections to the list in DIS-33 (p. 12).

**Additions:**

- c9 w m f
- c9a w m f (containing XXX y2)
- c9b y2 ch2 w
- c31 In(1) X2 w w y l2 \& y l2 /sc 8 y Do
- d7a cn Su-Pm/Cy cn vg Pm
- d7b cn Su-Pm Tac/Pm (dp b c ?)
- d9a l(2) m e
- d10a m/Pm
- d11c px slt sp
- e3a l(3) tr/M6 Sb
- e6a red
- g1e cn b y; e
- g1f ct 152 v; b w; e; (ey2)+
- g3c Cy tu bw; st su-tu
- g9a y; b w; e
- g9b y2 v f; bw
- g11a T(Y;2) J/px bw sp

**Discarded:**

- c24 car 1B3+1/Ins(1)sc S1 8 B w
- c25 car 1B5+2/Ins(1)sc 8 w/ sc, B w

**Wild Stocks**

- 106 In(1) dl-49, y Hw
- 120 wco su2/ FM
- 131 y sc m f5
- 133 y w
- 134 In(1)y, In(1)w
- 135 y w spl sn3/y
- 139 y w spl sn3/y

**Chromosome 1**

- 106 In(1) dl-49, y Hw
- 120 wco su2/ FM
- 131 y sc m f5
- 133 y w
- 134 In(1)y, In(1)w
- 135 y w spl sn3/y
- 139 y w spl sn3/y

**Chromosome 2**

- 128 y ac Dp w(a2)/ w(a2)/
- 129 y ac Dp w(a2)/ w(a2)/
- 130 y sc
- 200 a px sp
35:8  Melanogaster - Stocks - Berkeley

July 1961

201 al b c sp²/In(2LR)Cy, al² lt³ L² sp²  408 cl eyR12 ey²
202 al b pr cn vg c sp²/In(2LR)Cy, L² sp²  420 M-4/ey³
204 al dp b pr blt bw/Cy, al² lt³ L² sp²  Multichromosomal
205 al dp b pr c px sp/Cy pr
206 al dp b pr cn vg c a px bw mr sp²/Sc² Cy
   lt³ pr+ Bl cn² L² sp²
208 b
212 bw
214 c
215 cg-c/U
216 cl
218 cn bw
220 esc c sp/SM5, al² Cy lt⁵ sp²
225 l(2) gl cn bw/Cy, al² lt³ L² sp²
226 L⁴
228 pr cn ix/SM5, al² lt⁵ Cy sp²
229 pr en
232 vg
233 vg

Chromosome 3

301 cu
303 cv-c sbd²
308 Gl Sb/LVM
310 h
312 Ly/D²
314 p²
315 ru h st p² ss es
316 ru h th st cu sr es ca M/Cu.
319 se
320 se h
323 ss
324 ss²
325 ss²-B
340 In(3LR)TM1, M²/In(3LR)Rbx³ Es³
350 Pe/T(2,3)²M²

Chromosome 4

402 bt eyR sy²
403 bt²/ci¹
404 ci
405 ciW

BUFFALO, NEW YORK: STATE UNIVERSITY
College of Education
(Active M. Lang, Assoc. Prof. of Science)

Wild Stocks
Sex Linked
eosin eye
white eye
Chromosome 2
black body
brown eye
curved wing
vestigial wing
Chromosome 3
scarlet eye
Chromosome 4
eyless
### Wild Stocks

**1 Oregon-R**: isogenics, mixed

<table>
<thead>
<tr>
<th>Chromosome 1</th>
<th>Chromosome 2</th>
<th>Chromosome 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 f5 su-f</td>
<td>10 al</td>
<td>44 M(3)y G/L/5B Ubx</td>
</tr>
<tr>
<td>3 v f BB</td>
<td>11 al b lt c sp</td>
<td>45 M(3)y G/L Inv(3R)LVM/</td>
</tr>
<tr>
<td>4 v f BB/v v f car</td>
<td>12 al b pr stw c px</td>
<td>Inv(3L)LVM Sb Ubx</td>
</tr>
<tr>
<td>5 v f ++: reverted</td>
<td>13 al b pr stw c px</td>
<td>46 M(3)y G/L p3 cu/p3</td>
</tr>
<tr>
<td></td>
<td>14 al b pr stw c sp</td>
<td>cu Sb Ubx</td>
</tr>
<tr>
<td></td>
<td>15 al b pr stw px</td>
<td>47 M(3)y G/L Sb Ubx/LVM</td>
</tr>
<tr>
<td></td>
<td>16 al b pr stw sp</td>
<td>48 M(3)y G/L Sb/GU Ubx</td>
</tr>
<tr>
<td></td>
<td>17 al Cy L sp/al dp b pr</td>
<td>49 M(3)y G/L Sb/GU Ubx</td>
</tr>
<tr>
<td></td>
<td>lt stw c px sp</td>
<td>50 Mz cu sr e5 ca/ru h th</td>
</tr>
<tr>
<td></td>
<td></td>
<td>st cu sr e5 ca</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(&quot;ruca&quot;)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>51 ru h th st cu su e5 ca</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(&quot;ruca&quot;)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>52 ru h th st sr e5 Pr ca</td>
</tr>
</tbody>
</table>

### Wild-type

**1 Chicago wild-type**

<table>
<thead>
<tr>
<th>Chromosome 1</th>
<th>Chromosome 2</th>
<th>Chromosome 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 f57a/FM-1</td>
<td>11 In(2R)bw34, Cy/al dp b Bl c px sp</td>
<td>25 In(1)EN3, y/y f=</td>
</tr>
<tr>
<td>3 l(1)J-i, sc1J-i/Del</td>
<td>18 In(2LR)lac3</td>
<td>25 Ins(1)sc1EN3, y sc4 car y/y f=</td>
</tr>
<tr>
<td></td>
<td>(1)D4</td>
<td>26 Ins(1)sc1EN3, y sc4 car n w3 sc8/In(1)dl-49, y w la5</td>
</tr>
<tr>
<td>4 lxx/y w</td>
<td>19 T(2;3)lt+29</td>
<td>27 In(1)y4, y4</td>
</tr>
<tr>
<td>5 sc2 p3</td>
<td>20 pr ltd</td>
<td>28 Deficiencies - Duplications - X Chromosome</td>
</tr>
<tr>
<td>6 sc10-1/y Hw</td>
<td></td>
<td>28 w-5Sk13 spl; Dp(1;3)w5co</td>
</tr>
<tr>
<td>7 sc zn</td>
<td></td>
<td>29 Df(1)w555, y w spl dm; Dp(1;3)w5co/y w f</td>
</tr>
<tr>
<td>8 webb/y f :=</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 y ac z ec ct</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 y z4</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Multichromosomal

<table>
<thead>
<tr>
<th>Chromosome 2</th>
<th>Chromosome 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>11 b pr lt stw3</td>
<td>44 M(3)y G/L/5B Ubx</td>
</tr>
<tr>
<td>12 bwD</td>
<td>45 M(3)y G/L Inv(3R)LVM/</td>
</tr>
<tr>
<td>13 bw59</td>
<td>Inv(3L)LVM Sb Ubx</td>
</tr>
<tr>
<td>14 bw75</td>
<td>46 M(3)y G/L p3 cu/p3</td>
</tr>
<tr>
<td>15 bw81</td>
<td>cu Sb Ubx</td>
</tr>
<tr>
<td>16 bw5/cy cn2</td>
<td>47 M(3)y G/L Sb Ubx/LVM</td>
</tr>
<tr>
<td></td>
<td>48 M(3)y G/L Sb/GU Ubx</td>
</tr>
<tr>
<td></td>
<td>49 M(3)y G/L Sb/GU Ubx</td>
</tr>
<tr>
<td></td>
<td>50 Mz cu sr e5 ca/ru h th</td>
</tr>
<tr>
<td></td>
<td>st cu sr e5 ca</td>
</tr>
<tr>
<td></td>
<td>(&quot;ruca&quot;)</td>
</tr>
<tr>
<td></td>
<td>51 ru h th st cu su e5 ca</td>
</tr>
<tr>
<td></td>
<td>(&quot;ruca&quot;)</td>
</tr>
<tr>
<td></td>
<td>52 ru h th st sr e5 Pr ca</td>
</tr>
</tbody>
</table>
Melanogaster - Stocks - Chicago

30 Dp(1)w258-48, y w-; Dp(1;3)wco/yw
31 y w- rst2; Dp(1;2R)w158
32 Dp(1)w260; y w- f Y.w55f10/yw
33 Dp(1;3)w258-50a/4; y w f Y.w55f10/y w w sc8.Y

Ring-X
34 X01, Y/sc8.Y/v v f car
35 X02, Y cv v f car

Reversed Ring
36 RR, In(1)EN, car f v y /In(1)sc8, Y- ac- sc- m/sc8.Y

Tandem Metacentric
37 TM(Hw f), originally Y Hw v f oy+

Reversed Acrocentric
38 RA, Y ac sc pn -- In(1)sc8/In(1)sc8
(C.O.J-3), Y- ac- sc- w a f/sc8.Y, Y

X with Y Fragments Attached
39 Fr1, Y8 Y cv v f/y f:=
40 Y2 su-wa w8 Y.w BS/Ins(1)sc8
41 Y w- Dp BS/Ins(1)sc8 Y w- dL-49, v
42 y w f Y.w8/sc8.Y/or w- w:=
43 y Hw.YS Y/f T/Y 8/Y w- w:=

Attached X-Y; no free X
44 Y 8 B f v y Y.w8/Y f:=0/y v bb
45 Y 8 Y B-Y.w/y v f/0 with sc 4
46 Y 8 su-wa w8 Y.w8/Y w bb
47 Y 8 su-wa w8 Y.w8/Y w bb

Y S Fragments
51 Y.w8: ac+ 8 B Y L/y v w:=
52 Y 8: ac+ 8 B Y L/y v w:=
54 Y.w8: ac+ 8 B Y L/y w:=
55 Y.w8: ac+ 8 B Y L/y v w:=
56 Y.w8: ac+ 8 B Y L/y w:=
57 Y.w8: ac+ 8 B Y L/y w:=
58 Y.w8: ac+ 8 B Y L/y w:=
59 Y.w8: ac+ 8 B Y L/y w:=

Y L Fragments
60 Y.w8: ac+ 8 B Y L/y w:=
61 Y.w8: ac+ 8 B Y L/y w:=
62 Y CL/y w:=
63 Y.w8: ac+ 8 B Y L/y w:=
64 Y CL/y w:=
65 Y CL/y w:=

Multichromosomal
66 In(3LR)Ubx130, Ubx130 e3/Xa
67 SM1, al Cy sp2/In(2LR)102 dsW sp2;
68 Y/sc8.Y; ru h th st pC cu sr e3
69 Y; Cy/Pm; CxD/Sb, In(3R)Mo
70 Y; In(3R)Mo
71 Y; In(3R)Mo
72 Y.; In(3R)Mo
73 Y.; In(3R)Mo

CLEVELAND, OHIO: WESTERN RESERVE UNIVERSITY

Chromosome 1
1 ec dx 26 pr
2 sc cv v f 27 vg
3 v 25 net
4 w 26 pr
5 y sc v w x f:= 27 vg
6 y w sn3 26 pr

Chromosome 2
21 b 46 ru6 jy se by
22 dp 47 se
23 ho 248 st
24 ltd

Chromosome 3
41 cd
42 cu
43 e
44 g13
45 h

Chromosome 4
60 ci eyR
61 eyD ciD

Chromosomes 2, 4
76 cn; st
77 Pm dp b/cy sp2;

Chromosomes 1, 2
78 pr; Mal

Chromosomes 1, 2, 3
79 v; cn; st

Translocation
80 b pr tk

July 1961
Note: Stock list is the same as given in DIS-33 except:

Delete

C-47 dp b Ex^4/dla
H-59 y f:= and +; 1(2)55/Ins SM1, a^12 Cy sp^2; Sb/Ubx^30 e^4; pol

Add

C-20 da/Ins SM1, a^12 Cy sp^2

LAWRENCE, KANSAS: UNIVERSITY OF KANSAS
Department of Entomology


LEXINGTON, KENTUCKY: UNIVERSITY OF KENTUCKY

Wild Stocks

Big Ridge, Tenn. (single female strain), 1948
Bikini Atoll (mass-inbred strain), 1947
Pine Ridge, Ky. (mass-inbred strain), 1954

MINNEAPOLIS, MINNESOTA: UNIVERSITY OF MINNESOTA
Departments of Zoology and Animal Husbandry

Note: Only unusual stocks are listed.

Equilibrium mutant segregating (EMS) populations: Non-inbred populations in which one or more mutants with visible effects are present and in which there is a near approach to linkage equilibrium with respect to each mutant locus relative to all other loci concerned. Each population was initiated by crossing mutant and wild type stocks and the derived populations have been reproduced by selecting 3:1 or 9:3:3:1 ratios so that the frequencies of the mutants are held at approximately .5. They were at generation 25 on November 25, 1960.

1. e x Wild Gilbert
2. bw x Wild synthetic
3. bw x Falcon Woods wild
4. al x Falcon Woods wild
5. e x bw
6. al x bw
7. al x e
8. th x e

OAK RIDGE, TENNESSEE: OAK RIDGE NATIONAL LABORATORY
Biology Division

Wild Stocks

a-1 Canton-S a-2 Oregon-R a-3 Oregon-R-C a-4 Swedish-c a-5 Samarkand

Normal X Chromosome

b-1 B/y f:=
b-2 car bb
b-3 Co/y x f
b-4 ct^28 t/FM1, y^31d sc^8
b-5 dow/FM6, y^31d sc^8 dm B
b-6 ec dx
b-7 f
b-8 f BB/y f:=
b-9 f fu/CLB
b-10 fa
b-11 fa fa^no sn^3
b-12 fa N^22a/In(1)d1-49, y Hw m^2
b-13 fa N^22c sn^3/In(1)d1-49, y Hw m^2
b-14 fa rb
b-15 fa spl sn^3
b-16 fa^no
b-17 fa^no spl
b-18 fa^no/y f:=

b-19 1(1)J1 sc^j1/Del(1)24
b-20 kz/FM6, y^31d sc^8 dm B
b-21 m f car
b-22 N^264-40/In(1)d1-49, y Hw m^2 g^4
b-23 N^264-109/In(1)d1-49, y Hw m^2 g^4
b-24 Nco/In(1)dl-49, y Hw m2
b-25 nd
b-26 nd rb
b-27 ptg3 v m e2 sd f/y f:=
b-28 ras dy
b-29 rst2/FM1, y31d sg5 w ^2lzg B
b-30 rux/FM6, y31d sg5 dm B
b-31 s
b-32 sc cv v eq
b-33 sc cv v f B/y f:=
b-34 sc cv cy5 v e(1)dl-49, y Hw m2 g
b-35 sc cv ptg3 v/y v f car
b-36 sn5
b-37 spl
b-38 spl cho2
b-39 spl dm/y f:=
b-40 spl rb
b-41 sw
b-42 v
b-43 v f suW-f
b-44 w
b-45 wa
b-46 wa fa
b-47 wa fa rb
b-48 wa fa spl
b-49 wa fano rb
b-50 wa fano spl
b-51 wa fano spl rb/y w f
b-52 wa nd rb
b-53 wa spl
b-54 wa spl rb
b-55 wch rb/y w f:=
\text{b-56 we bbl/y f:=}
\text{b-57 we dy y w f:=}
\text{b-58 we fw}
\text{b-59 y}
\text{b-60 y inbred line A/sc8.Y}
\text{b-61 y inbred line B/sc8.Y}
\text{b-62 y ac sc pn/y f:=}
\text{b-63 y ac sc pn w rb cm ct6 sn3 ras2 v dy g2 f car/Ins(1)scs1,dl-49,scs1 v f car}
\text{b-64 y B/y f:=}
\text{b-65 y bb13a v w/sc8.y}
\text{b-66 y bb174 v w/sc8.y}
\text{b-67 y bb156 v w/sc8.y}
\text{b-68 y bb1452 v sc w-a bb/sc8.y}
\text{b-69 y bb1456 v sc w-a wa bb/sc8.y}
\text{b-70 y cv v f}
\text{b-71 y cv v f car}
\text{b-72 y f30a}
\text{b-73 y fa sn3}
\text{b-74 y 1259/sc8.Y/sc5}
\text{b-75 y 1451/FM6, y31d sc8 dm B}
\text{b-76 y Hw/ins(1)scs1,scs8,scs8r,scs81 wa B sc8}
\text{b-77 y N264-57/In(1)dl-49, y Hw m2 p4}
\text{b-78 y N264-103/In(1)dl-49, y Hw m2 p4}
\text{b-79 y N264-107/In(1)dl-49, y Hw m2 p4}
\text{b-80 y c wool spl f/In(1)rst3, rst3 f}
\text{b-81 y w bb}
\text{b-82 y w fano}
\text{b-83 y w fano sn3}
\text{b-84 y w spl sn3}
\text{b-85 y w}
\text{b-86 y w m f car}
\text{b-87 y w spl rb}
\text{b-88 y2 cho2}
\text{b-89 y2 cv v f}
\text{b-90 y2 spl}
\text{b-91 y2 w w/y f:=}
\text{b-92 No. 1663 (Fahmy)}
\text{b-93 No. 1920 (Fahmy)}

Chromosome II
\text{c-1 ab2/T(Y;2)E}
\text{c-2 ab2 tcm, bw sp2/Ins(2L+2R)Cy, Cy dp^n}
\text{c-3 a1 b c sp2}
\text{c-4 a1 dp b pr c px sp}
\text{c-5 a1 sp b pr El c px sp/SM1, al2 Cy sp2}
\text{c-6 b cn c bw}
\text{c-7 b pr c px sp}
\text{c-8 El L2/SM5, al2 Cy ltv sp2}
\text{c-9 bw}
\text{c10 bwD}
\text{c-11 cn bw}
\text{c-12 fr2 wt/Ins(2L+2R)Cy}
\text{c-13 ho}
\text{c-14 lt stw3}
\text{c-15 M(2)1/In(2R)Cy}
\text{c-16 M(2)S5/Ins(2L+2R)Cy, Cy (L4 sp2?)}
\text{c-17 M(2)S10/Ins(2L+2R)Cy, Cy pr Dp(2;2)412}
\text{c-18 ms cp bw/dp^t1 Cy pr El lt cn2 Ltv sp2}
\text{c-19 net a1 ex ds S ast shv ho rub/SM1, al2 Cy sp2}
\text{c-20 Pin/t/Ins(2L+2R)Cy}
\text{c-21 pr cn ix/SM5, al2 Cy ltv sp2}
\text{c-22 sp2 bs2}
\text{c-23 Sp j L2/Pin/SM5, al2 Cy ltv sp2}
\text{c-24 stw3 c}
\text{c-25 vg}

Chromosome III
\text{d-1 Bd10/In(3R)C, l(3)a}
\text{d-2 bx3u4e}
\text{d-3 C(3)x/tra}
\text{d-4 ca}
\text{d-5 cahd/In(3LR)Ubx130, M(3)1 Ubx130 e8}
\text{d-6 cu kar}
\text{d-7 cy-c sbd2}
\text{d-8 D3 J/In(3L)P, Me}
\text{d-9 DL3/In(3R)C, e}
\text{d-10 e}
\text{d-11 e4 wo ro}
\text{d-12 es}
\text{d-13 es cahd/In(3R)C, Sb e l(3)e}
\text{d-14 M1 Sl/LVM}
\text{d-15 H1/In(3R)Vno, Vno}
\text{d-16 jv1}
\text{d-17 M1 red/MT1, Me ri}
\text{d-18 1(3)tra Sl/In(3LR)Ubx130, Ubx130 e8}
July 1961

Melanogaster - Stocks - Oak Ridge

Chromosome IV

-1 bt
-2 btD/clD
-3 Ce2/spaCat
-4 ci eyR
-5 ci grl eyR svn
-6 ciD/eyD
-7 svn
-8 spa
-9 pol

Multichromosomal

f-1 v/\bb (1;Y)
f-2 br3 dxsT; ed Su2-dx (f;2)
f-3 In(1)smw; E-Var7/Ins(2L+2R)Cy, Cy (f;2)
f-4 In(1)d4-49, Y w; In(2L+2R)Cy, Cy/In(2LR)Pm, al4 ds33k 1t- bw01 (f;2)
f-5 v; In(2R)boWd/Ins(2LR)sm1, al2 Cy sp2 (f;2)
f-6 v; In(2R)boWd/SM1, al2 Cy sp2 (f;2)
f-7 YX+Y", In(1)EN, YS B y f; Y+Y y bb; bw (f;2)
f-8 YX+Y", In(1)EN, Yc+Y y/y2 su-wa bb; cyan bw (f;2)
f-9 In(1)AM, y/PM6, y3id dm B; SM1, al2 Cy sp2/Bl; In(3)Wno, Wno/In(3LR)Ubx130; Ubx130 e8 (f;2)
f-10 Ins(1)d-49, BM1, sc v BM1, SM5, cy2 Cy sp2/Pm; R112 (f;2)
f-11 spl rb; Ins(2LR)sm1, al2 Cy sp2/In(2LR)Pm, al4 ds33k 1t- bw01; C sb/In(3LR)Ubx130, Ubx130 e8 (f;2)
f-12 y; In(2L+2R)Cy, Cy/In(2LR)Pm, al4 ds33k 1t- bw01; Sh/In(3LR)D, D (f;2)
f-13 y; In(2R)boWd/SM1, al2 Cy sp2; M4/eyD (f;2)
f-14 YX+Y", FR1-LC-R, y; bw st (f;2)
f-15 v; In(2R)boWd/SM1, al2 Cy sp2; M4/eyD (f;2)
f-16 y; bw; cId/eyD (f;2)
f-17 y; bw; eyD/simulans (f;2)
f-18 y; bw; cI eyR (f;2)
f-19 y; f; bw; cI eyR (f;2)
f-20 w; e (f;2)
f-21 y; ru h th st pP cu sr es (f;2)
f-22 YX+Y", In(1)EN, Yw y Y x/y2 su-wa wa bb; Sh/In(3LR)Ubx130, Ubx130 e8 (f;2)
f-23 Xy, w/y2 w a bb; Ubx130e8; Sh/In(3LR)Ubx130, Ubx130 e8 (f;2)
f-24 sc cv v f B; ci eyR (f;2)
f-25 w ov v f; svn/svn (f;2)
f-26 y; svn (f;2)
f-27 y; f; cI eyR (f;2)
f-28 YX+Y", In(1)EN, Yw/y2 su-wa wa bb; svn (f;2)
f-29 al; ru (f;2)
f-30 b pr Bl/SM1, al2 Cy sp2; In(3R)Wno, Wno/In(3LR)Ubx130, Ubx130 e8 (f;2)
f-31 bw; st (f;2)
f-32 Ins(2L+2R)Cy, Cy/In(2LR)Pm, al4 ds33k 1t- bw01; Sh/In(3LR)D, D (f;2)
f-33 SM1, al2 Cy sp2/In(2LR)Pm, dp b ds33k; Sh/In(3LR)Ubx130, Ubx130 e8 (f;2)
f-34 stw; c; st (f;2)
f-35 bw; ci snv (f;2)
f-36 bw; cId/eyD (f;2)
f-37 bw; cId/simulans (f;2)

Inverted X Chromosomes

b-9 In(1)Cl, sc l 1 t2 v sl B g-7 In(1)d1-49, y f (ClD)
car/y f:=
g-8 In(1)d1-49, y v f (ClD) g-8 In(1)d1-49, vof f
g-9 In(1)d1-49, ty-1 bb+/y v f car

Inverted X Chromosomes

b-9 In(1)AM/T(1;3)65 y g-7 In(1)d1-49, y f (ClD)
car/y f:=
g-8 In(1)d1-49, y v f (ClD) g-8 In(1)d1-49, vof f
g-9 In(1)d1-49, ty-1 bb+/y v f car
Translocated Chromosomes

<table>
<thead>
<tr>
<th>Chromosome</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>h-1</td>
<td>T(1;2)459, y Y1459/FM6, y 31d se8 dm B</td>
</tr>
<tr>
<td>h-2</td>
<td>T(1;2)6Id/C1B</td>
</tr>
<tr>
<td>h-3</td>
<td>T(1;2)sc19/y f:=; fes sc19 a b pr/ Cy dp TH pr</td>
</tr>
<tr>
<td>h-4</td>
<td>T(1;1H)25(20), y Y125/FM6</td>
</tr>
<tr>
<td>h-5</td>
<td>T(1;1H)150(16-17), y Y1150/FM6</td>
</tr>
<tr>
<td>h-6</td>
<td>T(1;2LH)219(10A), y Y1219/FM6</td>
</tr>
<tr>
<td>h-7</td>
<td>T(1;2RH)75(20), y Y175/FM6</td>
</tr>
<tr>
<td>h-8</td>
<td>T(1;2RH)135(18-19), y Y1135/FM6</td>
</tr>
<tr>
<td>h-9</td>
<td>T(1;2;3)220(14A150A75), y Y1220/ FM6</td>
</tr>
<tr>
<td>h-10</td>
<td>T(1;2;3;4)454, y Y1454</td>
</tr>
<tr>
<td>h-11</td>
<td>T(1;2H)361(20), y Y1361/FM6</td>
</tr>
<tr>
<td>h-12</td>
<td>T(1;3H)53(12D), y Y153/FM6</td>
</tr>
<tr>
<td>h-13</td>
<td>T(1;3H)463(20), y Y1463/FM6</td>
</tr>
<tr>
<td>h-14</td>
<td>T(1;3LH)163(17A-A), x X1163/FM6</td>
</tr>
<tr>
<td>h-15</td>
<td>T(1;3LH)165(53C), y Y155/FM6</td>
</tr>
<tr>
<td>h-16</td>
<td>T(1;3RH)3(3',4'), y Y13/FM6</td>
</tr>
<tr>
<td>h-17</td>
<td>T(1;3RH)129(18B), y Y1129/FM6</td>
</tr>
<tr>
<td>h-18</td>
<td>T(1;4)47, y Y Y Y su-wa n8 bb</td>
</tr>
<tr>
<td>h-19</td>
<td>T(1;4)A13(165C)</td>
</tr>
<tr>
<td>h-20</td>
<td>T(1;4)A17(RA2)/y f:=</td>
</tr>
<tr>
<td>h-21</td>
<td>T(1;4)A17(RA2), y cv/y f:=</td>
</tr>
<tr>
<td>h-22</td>
<td>T(1;4)A19</td>
</tr>
<tr>
<td>h-23</td>
<td>T(1;4)A20/y f:=</td>
</tr>
<tr>
<td>h-24</td>
<td>T(1;4)B8(16A1), B8/y f:=</td>
</tr>
<tr>
<td>h-25</td>
<td>T(1;4)B8(16A1), B8 cv y f:=</td>
</tr>
<tr>
<td>h-26</td>
<td>T(1;4)B8(16A1), B8 cv y f:=</td>
</tr>
<tr>
<td>h-27</td>
<td>T(1;4)B8(16A1), y cv v B8/y f:=</td>
</tr>
<tr>
<td>h-28</td>
<td>T(1;4)B8(16A1), y cv v B8 cv y f:=</td>
</tr>
<tr>
<td>h-29</td>
<td>T(1;4)B11; 11R, y Y Y</td>
</tr>
<tr>
<td>h-30</td>
<td>T(1;4)e15</td>
</tr>
<tr>
<td>h-31</td>
<td>T(1;4)h4</td>
</tr>
<tr>
<td>h-32</td>
<td>T(1;4)h6</td>
</tr>
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</table>

Closed X Chromosomes

<table>
<thead>
<tr>
<th>Chromosome</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>i-1</td>
<td>Xo Y Y f:=</td>
</tr>
<tr>
<td>i-2</td>
<td>Xo2 (ET), +/M-5</td>
</tr>
<tr>
<td>i-3</td>
<td>Xo3 (KOA), +/M-5</td>
</tr>
<tr>
<td>i-4</td>
<td>Xo4, wsc1 491/In(1)dl-49, y Y Y Y</td>
</tr>
<tr>
<td>i-5</td>
<td>Xo2, wpont y f/RM, Ins(1) so5L, S, sc8R sc8 wa sc8/Y</td>
</tr>
<tr>
<td>i-6</td>
<td>Xo2, Y Y</td>
</tr>
<tr>
<td>i-7</td>
<td>Xo2, y f car</td>
</tr>
<tr>
<td>i-8</td>
<td>Xo2, In(1)wvc (stable), wvc/In(1) dl-49, y Y Y Y</td>
</tr>
<tr>
<td>i-9</td>
<td>Xo2, In(1)wvc (stable), wvc/In(1) so8, AM</td>
</tr>
<tr>
<td>i-10</td>
<td>Xo2, In(1)wvc (stable), wvc f/y f:=</td>
</tr>
<tr>
<td>i-11</td>
<td>Xo2, In(1)AB, +/y f:=</td>
</tr>
<tr>
<td>i-12</td>
<td>Xo2, wsc1 (from RR L-26), y cv v f/y f:=; sc8/Y</td>
</tr>
<tr>
<td>i-13</td>
<td>Xo2, wsc1 (from RR L-26), y Y Y /In(1) dl-49, Y Y Y Y</td>
</tr>
</tbody>
</table>

X Chromosomes with a Y Arm Attached

<table>
<thead>
<tr>
<th>Chromosome</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>j-1</td>
<td>X Y (A-2), y Y Y Y</td>
</tr>
<tr>
<td>j-2</td>
<td>X Y (C-2), y Y Y Y</td>
</tr>
<tr>
<td>j-3</td>
<td>X Y (U-8e), sc cv y f Y Y Y</td>
</tr>
<tr>
<td>j-4</td>
<td>X Y (U-8e), sc cv y f Y Y Y</td>
</tr>
<tr>
<td>j-5</td>
<td>X Y (U-8e), sc cv y f Y Y Y</td>
</tr>
<tr>
<td>j-6</td>
<td>X Y (Stern), g Y Y Y</td>
</tr>
<tr>
<td>j-7</td>
<td>X Y, y cv v f car Y Y Y</td>
</tr>
<tr>
<td>j-8</td>
<td>X Y, y v f Y Y Y</td>
</tr>
<tr>
<td>j-9</td>
<td>X Y, In(1)so5L, ENR, y Y Y Y</td>
</tr>
<tr>
<td>j-10</td>
<td>X Y, In(1)so5L, ENR, y Y Y Y</td>
</tr>
</tbody>
</table>
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Attached XY Chromosomes

m-1 XyL.yS (2-10T15 Parker), y² su-wa wa YL.YS/y/Y
m-2 XyL.yS (2-10T15 Parker), y² su-wa wa YL.YS/y/Y
m-3 XyL.yS 108-9 Parker), y² su-wa wa YL.YS/y bb/0
m-4 XyL.yS (112-17 Parker), y² su-wa wa YL.YS/y bb/0
m-5 XyL.yS (127-29 Parker), y² su-wa wa YL.YS/y bb/0
m-6 XyL.yS (129-11 Parker), y² su-wa wa YL.YS/y bb/0
m-7 XyL.yS (1(1)259 w YL.YS/y Dp(1iff)167
m-8 XyS.yL (110-6 Parker), y² su-wa wa YS.yL+x/y v bb/0
m-9 XyS.yL (115-9 Parker), y² su-wa wa YS.yL/y bb/0
m-10 XyS.yL (129-16 Parker), y² su-wa wa YS.yL/y bb/0
m-11 XyS.yL (1296-29 Parker), YS.y bb-.YL/y² su-wa wa bb/0
m-12 XyS.yL (FR-1, U-6dR), YS.y wa cv v f/y² su-wa wa bb/0
m-13 XyS.yL, In(1)EN, YS B f v YL/y² su-wa wa bb/0
m-14 XyS.yL, In(1)EN, YS B f v YL/y² su-wa wa bb/0
m-15 XyS.yL, In(1)EN, YS B f v YL/y² su-wa wa bb/0
m-16 XyS.yL, In(1)EN, YS y.L/y² su-wa wa bb/0
m-17 XyS.yL, In(1)EN, YS y.L/y² su-wa wa bb/0
m-18 XyS.yL, In(1)EN, YS y.L/y² su-wa wa bb/0
m-19 XyS.yL, In(1)EN, YS y.L/y² su-wa wa bb/0
m-20 XyS.yL, Ins(1)EN, 17, YS B f v YL/y² su-wa wa bb/0
m-21 XyS.yL, Ins(1)EN, 18, YS B f v YL/y² su-wa wa bb/0
m-22 XyS.yL, Ins(1)EN, 20, YS B f v YL/y² su-wa wa bb/0
m-23 XyS.yL, Ins(1)EN, 24, YS B f v YL/y² su-wa wa bb/0
m-24 XyS.yL, Ins(1)EN, 24, A, YS B f v YL/y² su-wa wa bb/0
m-25 XyS.yL, Ins(1)EN, 32, YS B f v YL/y² su-wa wa bb/0
m-26 XyS.yL, Ins(1)EN, 39, YS B f v YL/y² su-wa wa bb/0
m-27 XyS.yL, Ins(1)EN, 42, YS B f v YL/y² su-wa wa bb/0
m-28 XyS.yL, Ins(1)EN, 44, YS B f v YL/y² su-wa wa bb/0
m-29 XyS.yL, Ins(1)EN, 46, YS B f v YL/y² su-wa wa bb/0
m-30 XyS.yL, Ins(1)EN, 46, YS B f v YL/y² su-wa wa bb/0
m-31 XyS.yL, Ins(1)EN, 46, YS B f v YL/y² su-wa wa bb/0
m-32 XyS.yL, Ins(1)EN, 46, YS B f v YL/y² su-wa wa bb/0
m-33 XyS.yL, Ins(1)EN, 46, YS B f v YL/y² su-wa wa bb/0
m-34 XyS.yL, Ins(1)EN, 46, YS B f v YL/y² su-wa wa bb/0
m-35 XyS.yL, Ins(1)EN, 46, YS B f v YL/y² su-wa wa bb/0
m-36 XyS.yL, Ins(1)EN, 46, YS B f v YL/y² su-wa wa bb/0

Compound X Chromosomes

k-1 RA, 1(1)Jl stscj1--In(1)sc 6=/XYL.YS, y 1259y YL.YS/y sc 6/y
j-2 RA(ND-27), sc y f=In(1)sc 6=f v sc 6=/XYL.YS, y sc 6/y
n-12 RA, y f=In(1)sc 6=f v sc 6=/XYL.YS, y sc 6/y
g-26 RA, y ac sc pm--In(1)sc 6.
Altered Y Chromosomes

n-1 sc6.Y (y+ ac+ yL' bb+ YS)/y y
n-2 sc6. Yb+x (YL bx' bb+ YS ac+ y+)/y v f: = y v f
n-3 sc6. y (y ac+ yL' bb+ YS)/y y
n-4 Ybb- f (In1) 6w4 y w4
n-5 Ybb- = y eq
n-6 YbS (BS YL Yb+ bb+ YS) /y sm - w4 bb
n-7 Yb3sc (BS YL Yb+ bb+ YS y v)/y v; bw
n-8 YSu-Var- f (In1) 6w4 y w4
n-9 Ybxy (YL bx' bb+ YS)/y v; bw
n-10 Y y3Id 8y y w3Y y cv v f.

n-11 Y: bx (MYR)/y y;

Altered Stocks

Wald Stocks

a3 + Crimea
a4 + Florida-9
a5 + Lausanne-S
a6 + Oregon R
a7 + Samarkand
a8 + Sato, Japan
a9 + Swedish-b-6
a10 + Urbana-S
a11 + Wageningen
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b124 peb v
b125 "(bleached)" pn w rb
cn ct6 sn2 ras2 v dy
gt f car & y f=:
b126 pn,Inh1/y Hw In49
m2 g
b127 pn
b128 ptg
b129 r3/rk f B/InAM
b130 r9 & y f=:

b131 ras dy
b132 ras
b133 ras m
b134 ras m
b135 ras2
b136 ras3 m/CLB
b137 ras
b138 ras
b139 ras2/InAM, y31d
so c w4 lzs B
b140 ras2, In & y f=:
b141 ras3, In n v ct & y f=:
b142 ras2= 3/rst2) /y Hw In49
m2 g
b143 rux/FM6, y31d so c8 dm B
b144 rux2
b145 s
b146 sbr & y f=:
b147 sc
b148 sc cho
b149 sc ct6 car & y f=:
b150 sc cv v dnx/FM6,
y31d so c8 dm B
b151 sc cv ag
b152 sc cv v f
b153 sc cv ct6 f/ FM3, y31d so c8 dm B l
b154 sc In49 snx2 car/sc oc
dtg ad car
b155 sc In49 v Hw
b156 sc ptg sc car/y In49
snx2-BB(select B &

b157 sc pm3 g2 Ex2=(g2
reverted)
b158 sc t2 v f Tu car
& y f=:
b159 sc w B3/Lp YS & y f=:
b160 sc z ec ct5
b161 sc z w702 ec ct6
b162 Sc(Scotched eye)/y so c31
sc ct5
b163 sc pm4 t.
b164 sc flx/y so c8 B f
In49 v
b165 sd (se)
b166 sh2
b167 Sh2/FM1, y31d so c8 wa
iz3 B
b168 sn3
b169 sn3 1z46f24 v & y f=:
b170 sn3 v B31 & y w f=:

b171 sn4
b172 sn34e
b173 sn36a & y f=:
b174 sn3/y In49 m2 g

b175 sp-w
b176 spl
b177 spl rb cx & y f=:
b178 spl rb82
b179 sta & y f=:
b180 sta/FM3, y31d sc8
dg B1

b181 su3-s v
b182 su3-s wa cv t
b183 su3-s cv v f & y f=:
b184 su32-v-pr v & f B=:
b185 su32-v-pr v & y f=:
b186 su3-dx dx
b187 su3-wa wa
b188 su7
b189 su7 sr-wa wa
b190 su7 sr wa
b191 su7 srpol
b192 su7
b193 su7 vb2 sy/InAM
b194 su7
b195 su7 t2 v f
b196 su7 t3
b197 su7 t4

b198 su7 t5
b199 su7 t6
b200 un Bx2 & y f=:
b201 un4
b202 v

v203 v fasw f
v204 v fN3 car
v205 v f
v206 v f
v207 v f
v208 v f
v209 v f
v210 v f
v211 vb
v212 vb13 w
v213 w ec
v214 w ec
v215 w Om49 lzs & y f=:
v216 w f
v217 w f
v218 w f
v219 w f
v220 w f
v221 w f
v222 w f
v223 w f
v224 w f
v225 w f
v226 w f
v227 w f
v228 w f
v229 w f
v230 w f
v231 w f
v232 w f

v233 w f
v234 w f
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v236 w f
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v238 w f
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v245 w f
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v247 w f
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v252 w f
v253 w f
v254 w f
v255 w f
v256 w f
v257 w f
v258 w f
v259 w f
v260 w f
v261 w f
v262 w f
v263 w f
v264 w f
v265 w f
v266 w f
v267 w f
v268 w f
v269 w f
v270 w f
v271 w f
v272 w f
v273 w f
v274 w f
v275 w f
v276 w f
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Scute alleles

(listed alphabetically according to scutes regardless of position of scute in linear order):

c1 sc2

c2 sc2 pn & y f:=

c3 sc2-1 w & y f:=

c4 sc3b

c5 y sc4

c6 y sc4 B f InS &

c7 y sc4 B InS & y f:=

c8 y sc4 B v41b/y w In49

c9 y sc4 InS wA; S sc191

c10 sc5 bb255

c11 y sc5

c12 y sc5 w-258-48 spl; Dp(1;3)wvco; y f:=

c13 sc5 car

c14 sc6 wA

c15 sc7

c16 sc7 InAM car/Df(1)

b263-20

c17 sc7 oc ptg g,Inh &
y f:=

c18 sc7 wA

c19 sc8

c20 sc8 B

c21 sc8 B fX v & y f:=

c22 (w reddish) sc8 B InS

c23 sc9 bb wA

c24 sc9 car f In49 v &
y f:=

c25 sc9 f In49 v & y f:=

c26 sc9 f v cv & y f:=

c27 (w reddish) sc9 B InS

c28 sc9 Tu wA & y f:=

c29 y3id sc9 wA

c30 (y ac) B270 (dappled) sc9

b 258-4 B wA/w In49 l2s 48

c31 y3d sc9

c32 y3d sc9 B f In49 v

c33 y3d sc9 B f In49 v

Partly reverted?)
Combination of scute or similar inversions

d1 $y^{sc^4} B In^c y^{sc^8}$ & $y f$ =

d2 $y^{sc^4} B In^c y^{sc^8} & y f$ =

d3 $y^{sc^4} B In^c y^{sc^8} & y f$ =

d4 $y^{sc^4} B In^c y^{sc^8} & y f$ =

d5 $y^{sc^4} B In^c y^{sc^8} & y f$ =

d6 $y^{sc^4} B In^c y^{sc^8} & y f$ =

d7 $y^{sc^4} B In^c y^{sc^8} & y f$ =

d8 $y^{sc^4} B In^c y^{sc^8} & y f$ =

d9 $y^{sc^4} B In^c y^{sc^8} & y f$ =

d10 $y^{sc^4} B In^c y^{sc^8} & y f$ =

d11 $y^{sc^4} B In^c y^{sc^8} & y f$ =

d12 $y^{sc^4} B In^c y^{sc^8} & y f$ =

d13 $y^{sc^4} B In^c y^{sc^8} & y f$ =

Translocations of X and 4

e1 TX(1B3+)4 $y^{sc^8} B y^{sc^8} & y f$ =

e2 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e3 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e4 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e5 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e6 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e7 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e8 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e9 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e10 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e11 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e12 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e13 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e14 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e15 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =

e16 TX(3C2)4 $y^{sc^8} B y^{sc^8} & y f$ =
Altered Y's sometimes with mutants in X and/or autosomes
(The presence of Y¹ and/or Y² attachments on X·Y chromosomes is uncertain unless they have been freshly tested or are accompanied by markers (bb⁻ for Y¹ and y⁺sc⁸ for Y²) that can be followed.)

f₁ Y² su-wa² wᵃ Y⁸,Y² y⁺ & y v bb⁻= (no free Y)
f₂ Y⁵-DpR y X⁺ bb⁺y⁺ & y² su-wa² wᵃ bb⁻= (no free Y)
f₃ Y⁵-X InEN B y·y⁺ & y² su-wa² wᵃ bb⁻= (no free Y)
f₄ Y⁵-X InEN B y·Y⁸ y⁺ & y¹ su-wa² wᵃ bb⁻; S šes Sp b/(1⁺? ) InCyL b (no free Y)
f₅ Y⁵-X InEN In26 B f+v·Y³ sc⁸ y⁺ & y² su-wa² wᵃ bb⁻= (no free Y)
f₆ ("snoc") Y⁸,X InEN ptg oc sn₅,Y⁸, L & sc ctₙ oc ptg car.
(, no free Y)

f₇ ("snoct") Y⁵,X InEN ptg os sn₅.Y⁸, & sc ctₙ oc ptg car.
(, no free Y)
f₈ Y⁵,X InEN v ptg oc sn₅ w·Y⁸ sc⁸ y⁺ & y sc t² v f car⁻= (no free Y)
f₉ Y⁵,X InEN v y·y⁺(sc³⁺ y) (no free Y)
f₁₀ Y⁵,X InEN v y·y⁺(sc³⁻ y) & sc ctₙ oc ptg car.
(, no free Y)
f₁₁ Y⁵,X InEN v y·y⁺(sc³⁻ y) & sc ctₙ oc ptg car.
(, no free Y)
f₁₂ Y⁵,X InEN v y·Y⁸ sc³⁺ y⁺/v₁; bw/VA/L² l (no free Y)
f₁₃ Y⁵,X InEN v y·Y³ sc³⁺ y⁺ (no free Y)
f₁₄ Y⁵,X InEN v y·Y³ sc³⁺ y⁺ & y² su-wa² wᵃ bb⁻= (no free Y)
f₁₅ Y⁵,X y In49 v f car·Y⁸ (no free Y)
f₁₆ Y bb⁻/w sn bb & y v f=:
ff₁₇ Y bb⁻/w²/sbb²/ (it is uncertain)
f₁₈ Y bb⁻/w² eq
f₁₉ Y bb⁻/w² bb⁻/w² bbb⁻/ & bbb⁻/Y⁺; InsNS px sp/Im²² (Bridges)
f₂₀ Y:bw⁺; net bw crs (iso 1955)
f₂₁ Y:bw⁺/v₁; bw
f₂₂ Y:bw⁺/y v s & sc²·Y/y v; bw (Select)
f₂₃ Y:bw⁺/y v s & sc³·Y/y v; 2 Sp cn bw/dp'tXI Cy cn bw (Select)
f₂₄ ("MYRM") Y:bw⁺/X⁺; bw
f₂₅ ("MYRM") Y:bw⁺/X² oc y f; bw (ring OK 1957)
f₂₆ l₁₁⁺-Y/l₁₁ sc Jo (extra Y in ²)
f₂₇ ("Max") l₁₁⁺-Y/l₁₁ sc Jo (extra Y in ²) In49 ptg oc B¹¹/y scS¹ car ody f g² dy v ras² sn³
cb cm rb ec w pn l sc⁸
f₂₈ ("Max-x") l₁₁⁺-Y/l₁₁ sc Jo (extra Y in ²) In49 v ptg oc B¹¹/y scS¹ car ody f g² dy v ras²
sn³ cb cm rb ec w pn l sc⁸
f₂₉ sc⁸/Y/ac³
f₃₀ sc⁸/Y In(X²) w² oc f & y f=:
f₃₁ sc⁸/Y In49 ptg oc B¹¹ & y f=:
f₃₂ sc⁸/Y/l₁₁ sc Jo & y f=:
f₃₃ sc⁸/Y/l₁₁ sc Jo In49 v B¹¹ & y f=:
f₃₄ sc⁸/Y l (y ac)- B In49 sn²² sc⁸ & y f=; (from X-r ooogonia $24)
f₃₅ ("Max-Tu") sc⁸·Y/l (y ac)- Tu B In49 sn²² sc⁸/y ac pn w rb cm sn³ cb sext oc ras² v
g² f o d car sw
f₃₆ sc⁸/Y oc ptg & y f=; (iso 1956)
f₃₇ sc⁸/Y sc w B·Y² & y f=; Cy,In/S Sp ab² ltd
f₃₈ sc⁸/Y sc w ot f·Y² & y f=; Cy,In/S Sp ab² ltd
f₃₉ sc⁸/Y scV₁ v & sc⁸/Y/y f=; sc19I/Cy lt³
f₄₀ sc⁸/Y/Tam(X;3) & sc²·Y/y f;=:
f₄₁ sc⁸/Y In2² y f & y f=; (ring OK 1957)
f₄₂ sc⁸/Y In2² y v & y f=; (ring OK 1957)
f₄₃ sc⁸/Y ac sc B·Dp(sc⁸/ac⁺ y⁺) & y Nf f=; (N with w⁺)
f₄₄ sc⁸/Y ac sc oc ptg & y f=:
ff₄₅ sc⁸/Y ac sc & y f=:
ff₄₆ sc⁸/Y Be⁺ & y f=; (to cross d by y scS¹ In49 sc⁸; bw; st p² g)
f₄₇ ("multi-d") sc⁸/Y/y In49 B & y f=; bwD
ff₄₈ sc⁸/Y In49 B¹¹
f₄₉ sc⁸/Y In49 v F¹ g & y f=:
f₅₀ sc⁸/Y In49 v F¹ g & y In49 v F¹ g/pn,Inh
f₅₁ sc⁸/Y In49 v f
f₅₂ sc⁸/Y In49 v f B·Y² & y f=:
ff₅₃ sc⁸/Y sc w In49 v g f
<table>
<thead>
<tr>
<th>String</th>
<th>Meaning</th>
</tr>
</thead>
<tbody>
<tr>
<td>f54</td>
<td>sc^6.Y/y sc w In^49 v g f &amp; y f=</td>
</tr>
<tr>
<td>f55</td>
<td>sc^6.Y/y sc^4 B f In^49 &amp; y f=</td>
</tr>
<tr>
<td>f56</td>
<td>sc^6.Y/y sc^4 B In^49 w^4 &amp; y f=</td>
</tr>
<tr>
<td>f57</td>
<td>sc^6.Y/y sc^4 B In^49 &amp; y f=</td>
</tr>
<tr>
<td>f58</td>
<td>sc^6.Y/y sc^4 B In^49 w^4 sc^6 &amp; y f=</td>
</tr>
<tr>
<td>f59</td>
<td>sc^6.Y/y sc^4 f In^49 w^4 &amp; y f=</td>
</tr>
<tr>
<td>f60</td>
<td>sc^6.Y/y sc^4 w sc^6 {sc^6.Y In^5 &amp; 9}</td>
</tr>
<tr>
<td>f61</td>
<td>(&quot;multipare D&quot;) sc^7.Y sc^1 B In^49/y Hw In^49 w^2 g^4; (ci g^4 ey^1 sy^7)</td>
</tr>
<tr>
<td>f62</td>
<td>sc^7.Y sc^7 (rein. sc^4) B.Dp(sc^1 ac^4) &amp; y f=</td>
</tr>
<tr>
<td>f63</td>
<td>sc^7.Y v &amp; y f=</td>
</tr>
<tr>
<td>f64</td>
<td>sc^7.Y/y with*</td>
</tr>
<tr>
<td>f65</td>
<td>sc^7.Y/y w^4 B &amp; y NM f= (N with w)</td>
</tr>
<tr>
<td>f66</td>
<td>sc^7.Ysc^6 sc^6 B f In^49 v</td>
</tr>
<tr>
<td>f67</td>
<td>sc^7.Ysc^1 sc^6 B f In^49 v</td>
</tr>
<tr>
<td>f68</td>
<td>sc^7.Ysc^6 sc^6 B In^49 w &amp; y f= (ho ed cl+/)</td>
</tr>
<tr>
<td>f69</td>
<td>sc^7.Ysc^6 sc^6 y w^4 ras^2</td>
</tr>
<tr>
<td>f70</td>
<td>sc^7.Ysc^6 sc^6 y w^4 dp</td>
</tr>
<tr>
<td>f71</td>
<td>sc^7.Ysc^6 sc^6 y w^4 (ho) ed cl</td>
</tr>
<tr>
<td>f72</td>
<td>sc^7.Ysc^6 sc^6 y ct^6 &amp; y f=</td>
</tr>
<tr>
<td>f73</td>
<td>sc^7.Ysc^6 sc^6 y w^4 &amp; y f=</td>
</tr>
<tr>
<td>f74</td>
<td>sc^7.Ysc^6 sc^6 y w^4 ct^6 &amp; y f=</td>
</tr>
<tr>
<td>f75</td>
<td>sc^7.Ysc^6 sc^6 y w^4 ct^6 &amp; y f=</td>
</tr>
<tr>
<td>f76</td>
<td>sc^7.Ysc^6 sc^6 y w^4 ct^6 f &amp; y f=</td>
</tr>
<tr>
<td>f77</td>
<td>sc^7.Yscw^4/ac^2; bw</td>
</tr>
<tr>
<td>f78</td>
<td>sc^7.Yscw^4/ac^2; cn bw</td>
</tr>
<tr>
<td>f79</td>
<td>sc^7.Yscw^4/ac^2; B In^49 w; bw</td>
</tr>
<tr>
<td>f80</td>
<td>sc^6.Ysc^4/y w^4</td>
</tr>
<tr>
<td>f81</td>
<td>YL/f/Y^2 &amp; sc v f=</td>
</tr>
<tr>
<td>f82</td>
<td>YL/f/Y^2 &amp; Y^2 w^2 g^2 f=</td>
</tr>
<tr>
<td>f83</td>
<td>YL/InEN2/YS &amp; y ct^6 f= (InEN2 from X^2 opened)</td>
</tr>
<tr>
<td>f84</td>
<td>(&quot;YLc snocty&quot;) Y/oc ptg.YS &amp; YLc/Y ct^n oc ptg car</td>
</tr>
<tr>
<td>f85</td>
<td>YLc/oc ptg.YS &amp; y v f=; S Sp ab^2 ltd/Cy,Ins cn^2</td>
</tr>
<tr>
<td>f86</td>
<td>(&quot;YLc snocty; bw&quot;) YLc/oc ptg.YS &amp; YLc/Y ct^b oc ptg car</td>
</tr>
<tr>
<td>f87</td>
<td>YLc/w oc ptg.YS &amp; YLc/y w^2 x^+w^3 In^49 B sc^6 (tandem X-X giving rings)</td>
</tr>
<tr>
<td>Sterilizer (&quot;sz&quot;) stocks (f88-f97)</td>
<td></td>
</tr>
<tr>
<td>f88</td>
<td>(&quot;sz w&quot;) YLc/w YS</td>
</tr>
<tr>
<td>f89</td>
<td>(&quot;sz +w&quot;) YLc/X.YS</td>
</tr>
<tr>
<td>f90</td>
<td>(&quot;sz bw&quot;) YLc/X/YS; bw</td>
</tr>
<tr>
<td>f91</td>
<td>(&quot;sz bw ac&quot;) YLc/X/YS; bw; ac</td>
</tr>
<tr>
<td>f92</td>
<td>(&quot;sz ac&quot;) YLc/X.YS &amp; y v f=; c</td>
</tr>
<tr>
<td>f93</td>
<td>(&quot;sz e&quot;) YLc/X.YS &amp; y v f=; e</td>
</tr>
<tr>
<td>f94</td>
<td>(&quot;sz le x&quot;) YLc/le^2 m f.YS &amp; y v f=</td>
</tr>
<tr>
<td>f95</td>
<td>(&quot;sz le m f&quot;) YLc/le^2 m f.YS &amp; y v f=</td>
</tr>
<tr>
<td>f96</td>
<td>(&quot;sz m f&quot;) YLc/le m f.YS &amp; y v f=</td>
</tr>
<tr>
<td>f97</td>
<td>(&quot;sz y w&quot;) YLc/y w.YS &amp; y ct^6 f=</td>
</tr>
<tr>
<td>f98</td>
<td>YLc/y w.YS &amp; YLc/y w.YS &amp; Y ct^6 f=</td>
</tr>
<tr>
<td>f99</td>
<td>YLc/y w.B &amp; sc^6/ac^2; w^3 ct^6 f=</td>
</tr>
<tr>
<td>f100</td>
<td>(new &quot;facl&quot;, 1959) YLc/y sn oc ptg.YLc/y w^2 oc ptg EM^1/sc^8 In^49 snx^2 sc^8</td>
</tr>
<tr>
<td>f101</td>
<td>(&quot;tjmd&quot;) YLc/y sn oc ptg.YLc/sc^1/pn w rh cm ct^6 oc ras^2 v dy g^2 f od car</td>
</tr>
<tr>
<td>f102</td>
<td>YLc/y sn In^49 v f.Y^2 &amp; y sc t^2 v f car=</td>
</tr>
<tr>
<td>f103</td>
<td>YLc/y sn sc oc ptg.YS &amp; y v f=</td>
</tr>
<tr>
<td>f104</td>
<td>YLc/y oc ptg.YS &amp; YLc/y w^3</td>
</tr>
<tr>
<td>f105</td>
<td>sc^6.Y/sc w ptg.YS &amp; sc^6/Y/y f=; Cy,Ins cn^2/S Sp ab^2 ltd</td>
</tr>
<tr>
<td>f106</td>
<td>sc^6.Y/sc w B/YS &amp; y f=; Cy,In/S Sp ab^2 ltd</td>
</tr>
<tr>
<td>f107</td>
<td>sc^6.Y/sc w B/YS &amp; In/YS &amp; y f=</td>
</tr>
<tr>
<td>f108</td>
<td>sc^6.Y/sc w ct^6 f.YS &amp; y f=; Cy,In/S Sp ab^2 ltd</td>
</tr>
<tr>
<td>f109</td>
<td>sc^6.Y/ac sc ct^6 f.Y^2</td>
</tr>
<tr>
<td>f110</td>
<td>sc^6.Y/y In^49 v f.Y^2</td>
</tr>
</tbody>
</table>
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f111 sc YLS/In49 v f YLS
f112 sc YLS/In49 YLS/reln. sc 8-4 YLS & Y f :=
f113 sc YLS/In49 YLS v InCyR & Y f :=
cn bw (e)
f114 sc YLS/YLS v YLS & Y f :=
f115 sc 2 YLS sc/ct6 f YLS & sc YLS/
y2 X+ sc 6 wa Ins Bg (tandem X-X
giving rings)
f116 YLS/YLS sc w oc f YLS & Y f :=
f117 YLS/YLS sc YLS/oc YLS & Y f :=
f118 YLS/YLS sc YLS/oc YLS & Y f :=
f119 YLS/YLS sc YLS/oc YLS & Y f :=
f120 ("plond") YLS/YLS oc f YLS & Y f :=
f121 YLS/YLS sc w oc f YLS & Y f := (Stern) (dp?)
f122 YLS/YLS sc/ct6 f YLS & Y f :=
f123 YLS/YLS sc YLS/oc YLS & Y f :=
f124 sc YLS/YLS sc In49 YLS v YLS & Y f :=
f125 sc YLS/YLS sc YLS/oc YLS & Y f :=
f126 sc YLS/YLS sc YLS/oc YLS & Y f :=
f127 sc YLS/YLS sc YLS/oc YLS & Y f :=
f128 sc YLS/YLS sc w oc f YLS & Y f :=
f129 TY2O/b pr (tk)
f130 TH3(TyA3) 1/r u h D InsCXYCA (TY3
in and)
f131 Tp4:Y (2 Ys in both & q)
(Transpos. Edmondson)
f132 Tp4:Y/Basc & Y f :=
ci eyR
f133 Tp4:Y/Cat Cat & Cat/M4 q
(un-selected)
f134 Tp4:Y/catD
f135 Tp4:Y/eD or Cat d & eyD/Cat q
f136 Tp4:Y/X+ & Y f :=

Chromosome 2*

*s2 and/or Cy are to be understood always to be accompanied by InCyL and cn2 by
InCyR even where not so designated. When
cn is present InCyR is absent. Ins
following S2 or Cy after a comma refers
to both of these inversions, but InL only
to the left-arm one. If either of these
inversions is designated in a chromosome
without the other, the latter should be
understood to be absent. InMis designates
the long pericentric inversion of Mislove.

g1 a px or

g2 a px sp

g3 a bx

g4 a bx 2 s2 Ins(CyL, CyR) 1/cn2

g5 a bx T(Y;E)

g6 a bx 2 pm1/Cy pr Bl cn2 L4 sp2

g7 a bx 2 bx bw sp2/Cy, dpTh Bl L4 sp2

g8 a bx 2 InCyR L4 sp2 Bl InsNSAR mr

g9 a bx 2 ms ta crs/Cy pr Bl cn2 L4 sp2

g10 a bx 2 SNS5, a12 Cy Ltv sp2

g11 ad

g12 al

g13 a bx c sp

g14 a bx cn sp (iso)

g15 a bx dp b bw l2 ax/SN5, a12 Cy Ltv sp2

g16 a bx dp b pr

g17 a bx dp b pr blt bw/SN5, a12 Cy Ltv sp2

g18 ("apl") a bx dp b pr c px sp

g19 ("twelvepl") a bx dp b pr cn vg a px bw

mr sp/2S Cy Ltv pr + Bl cn2 L4 sp2

g20 a1 S ast ho/Cy, En-S

g21 a12 Cy ab5 b pr Bl cn2 L4 sp2/S sp

g22 a12 Cy, InL l3/b pr Bl l3 cn2 InCyR L4 sp2

g23 a12 Cy pr Bl cn2 InCyR c vg sp2/InsNS px sp

g24 a12 Cy pr Bl cn2 L4 bw sp2/InsNS px sp

g25 a12 InMis dpTI CY cn2 L4 sp2/Sp U,
InLR bw

g26 a12 InMis dpTI Cy pr Bl cn2 L4 sp2/
Sp U,InLR bw

g27 Alu

g28 an/SN5, a12 Cy Ltv sp2

g29 ang

g30 ant; (ro)
g31 ap7/RvD, In2LR

g32 ap7/SN5, a12 Cy Ltv sp2

g33 arch chl/SN5, a12 Cy Ltv sp2

g4 ast ho

g35 ast ho ed dp cl

g36 ast dp cl sp

g37 ast dp cl sp

g38 b

g39 b cn bw

g40 b el rd sp cn

g41 b np

g42 b Go/Gla

g43 b

g44 b (2)Bld pr c px sp/SN5, a12 Cy Ltv sp2

g45 b llt bw

g46 b llt on mi sp/b In(2)bwDel

g47 b llt wxt bw

g48 b nub pr

g49 b pr

g50 b pr Bl tk/s2 Cy cn2 L4 sp2

g51 b pr c px sp

g52 b pr tk

g53 b pr tk/T(Y;E)G

g54 b sf

g55 b vg

g56 El/esc

g57 El/Cy, bx 45a sp2 or 45a

g58 El/In2LR dp

g59 El bx "VA"T(2;3)/Cy, InL L2

g60 El L2/Cy, dp2

g61 El sw3/In2LR dp

g62 El sw45 blt tuf/SN5, a12 Cy Ltv sp2

g63 El/SN5, a12 Cy Ltv sp2

g64 El bx

g65 blt

g66 bran

g67 br1

g68 bs2

g69 bw (iso 2, 1959)

g70 bw ba

g71 bw sp (iso 1954)
g185 InNS1 InNSR/a12 Cy, InN L13 L2

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g292 S/Cy En-5

g293 S dp'/al2 Cy on2 L4 sp2

g294 S fes Atu It/al2 Cy on2 L4 sp2

g295 S fes Sp B/Cy B it2 Cy on2 L4 sp2

g296 S fes Sp ms ta on mr crs/al2

InMs dptxI Cy pr Bl on2 L4 sp2

g297 S Sp ab2 ap1 InNSR px sp/al2 Cy

Bl on2 L4 sp2

g298 S Sp Bl bx'/Cy on2 lc

g299 S Sp Bl lPr bx'/dptxI Cy, Insos pr on2

g300 S Sp Bl lPr bx'/dptxI Cy, Insos pr on2

g301 S Sp Bl lPr bx'/dptxI Cy, Insos pr on2

g302 S Sp cn bw/dptxI Cy on bw

g303 S Sp crs/al2 Cy pr Bl L4 sp2

g304 S Sp InNSR mr/dptxI Cy pr Bl on2

L4 sp2

g305 S Sp (ls?) on/dptxI Cy on

g306 S Sp (ls?') on bw sp/dptxI Cy, InL

on bw sp

g307 S Sp ms ta on crs/al2 InMs dptxI Cy

pr Bl on2 L4 sp2

g308 S Sp ms ta on crs/dptxI Cy, Insos pr

on2

g309 S Sp pr cn Bl rtT23 InNSR mr/dptxI Cy

pr on2

g310 S Sp pr cn2 InCyD/dptxI Cy pr on2

g311 S Sp rtT23/dptxI Cy pr Bl on2 L4

sp2

g312 sg2051b InCyDlp bx L4 Pm1

g313 S Cy lT3 pr Bl on2 L4 sp2/InNSL

InNSR px sp

g314 sg2 dptxI, InCyL/ls Sp b

g315 sg3/dptxI, InCyL/ls Sp b

g316 sca

g317 scg 1(2)c/SM5, al2 Cy lTv sp2

g318 sc2

g319 shr bw2b ab sp/SM5, al2 Cy lTv sp2

g320 shv

g321 shv ho

g322 sn px/SM5, al2 Cy lTv sp2

g323 sm px pd/SM5, al2 Cy lTv sp2

g324 so

g325 so2 b cn

g326 sp2 bs2

g327 Sp/ln(2)Lt, l(2)R

g328 Sp/Sm5, al2 Cy lTv sp2

g329 Sp pur on InNSR px sp/Cy pr Bl on2

L4 bw sp

g330 Sp J/ln(2)Lt-Cy-t, S-S dp2 pr

g331 Sp J/SMD, al2 Cy lTv sp2

g332 Sp J L2 Pin/SM5, al2 Cy lTv sp2

g333 Sp ms on mr crs/Cy pr Bl on2 L4 sp2

g334 spd gt-4/Gla, InLR

g335 sp1e

g336 sp

g337 std/SM5, al2 Cy lTv sp2

g338 stw

g339 stw2

g340 stw3

g341 stw2/T(Y;2)B

g342 stw4

g343 stw48 btl tuf

g344 ta on bw/al2 Cy pr Bl on2 L4 sp2

g345 ta on bw sp/Cy pr Bl on2 L4 sp2

g346 Tfl/Cy

g347 ttk/SM5, al2 Cy lTv sp2

g348 ttk

g349 tri vgMo2/SM5, al2 Cy lTv sp2

g350 tuf ltd

g351 U/cg c

g352 uf

g353 vg (iso 2,3)

g354 vg bw

g355 vg2/SM5, al2 Cy lTv sp2

g356 vgC/Rvd, In2LR

g357 vgC/SM5, al2 Cy lTv sp2

g358 vgD/SM5, al2 Cy lTv sp2

g359 vgD sp2/Cy on2 L4 sp2

g360 vgi

g361 vgsp

g362 vgIw H/A/SM5, al2 Cy lTv sp2

g363 vgIw H/A/T(2;3)SM Cy

g364 vgIw R/vl, bw sp or

g365 vst/SM5, al2 Cy lTv sp2

g366 whd

g367 wt

Chromosomes (containing genes of 2 in a few cases)

h1 a-3
h2 a a h
h3 abd
h4 app
h5 ag
h6 bar-3(Ives)

h7 Bli(3R)C, 1(3)a
h8 Bli(3R)C, Sb e l(3)e
h9 bp/TM1, Me ri
h10 bul

h11 bv
h12 bx3, Cbx Utb bxd pbx/Xa
h13 bx3

h14 bx3 Cbx Utb
h15 ca
h16 ca bv
h17 ca K-pn
h18 ca2

h19 ca3jIIIa3/Me, Ins ri

Sbl
h20 Cbx
h21 od

h22 cmp ca/ln(3R)C, e
h23 cor ru h D InsCXF
h24 cp in ri pB
h25 cp
h26 cu
h27 cu kar
h28 cur
h29 cv-c
h30 cv-c sbd2
h31 cv-d
h32 D/GL
h33 D InsCXF/Tri
h34 D tra/lnLP Dfd InRP ca
h35 D3 H/InsP
h36 D3 Sb ca2/Payne
h37 det
h38 Df(3)MS31/T(2;3)Me
h39 Df(3)sbd105/Xa
h40 Dfd/ln(3LR)C
h41 Dfd
h42 DL H e8 od/ln(3R)spr,
spr
h43 DLl3(3R)C, e
h44 DLl5(3R)C, 1(3)s
h45 DL14(3R)Cyd, Cyd
h46 DLx/Payne
h47 drb
h48 drh/Payne, Dfd ca
h49 e3 P ru h D InsCXF e
h50 e1 wo ro
h51 e11
h52 e5
h53 eg/In(3LR)Cx
h54 eg2/In(3LR)Cx
h55 eyg
h56 f2
h57 g12 e4
h58 g13
h59
h60 Gm bxD/InsLVM
h61 Gm Pb/Payne
h62 gs
h63 h
h64 h ri
h65 h ri ca {iso 1953}
 h66 h ri e5 {iso 1957}
 h67 h2
h68 H/In(3R)hp, hp
h69 H Pr/In(3R)C, e
h70 H2/Xa
h71 H2/In(3R)C, Sb e 1(3)e
h72 HmF h ri/ru h D Sb InsCXF
h73 HmP3 sr
h74 in
h75 In(3L)mot-36e/B
h76 In(3LP), In(3R)P18, Me
h77 Ins(3)Ubx1-30T/(2;3)Xa
h78 Ins(3)AntrP/TM1, Me ri
h79 Ins(3)PDLB, st DLB/In(3R)
E+, st 1(3) W ca
h80 Ins(3R)Hm, Hm SbP/Payne
h81 Ins(3R)Ml, sr/Xa, ca
h82 Ins(3R)PFLA (homozygous)
hs3 jv
h84 jv HmF h
h85 jv1
h86 kar2
h87 Kl
h88 1(3)ac e5 M(3)w/LVM
h89 1(3)6d10/In(3LR)Cx, D
h90 1(3)tr Sb/InsLVM, Gm 1-, Ubx130, e5
h91 1(3)tr Ubx/TM1, Me ri Sb1
h92 1d
h93 Ly/D3
h94 Ly Sb/LVM
h95 M(3)/In(3R)C, l(3)e
h96 M(3)6e/In(3R)C, l(3)a
h97 M(3)4010/Payne, Dfd ca
h98 M(3)B/In(3R)C, e 1(3)e
h99 M(3)B/In(3R)C, Sb e 1(3)e
h100 M(3)S32/T(2;3)Me
h101 M(3)S34/T(2;3)Me
h102 M(3)S36/T(2;3)Me
h103 M(3)S37/Me
h104 M(3)w/In(3R)C, e 1(3)e
h105 M(3)y/Me
h106 ma
h107 ma fl
h108 mah
h109 Mv/Cx
h110 Me,InL bxD/ru h D
InsCXF Sb
h111 Me,InL InRC e 13e/ru h D
InCXF Sb e5
h112 Me,InL Sb/ru h D
InsCXF
h113 Me,Ins ri Sb3/ru h D
InsCXF ca
h114 Me,Ins ri Sb3/InRC e 13e
h115 Msq
h116 Msx/Xa
h117 obt
h118 p
h119 pP
h120 pP bx sr e5
h121 pP cu
h122 pP/In(3LR)Cx
h123 pbx/Xa
h124 Pr/TM1, Me ri
h125 Pr/In(3R)C, e
h126 Pr D/InTM, y ac+ ri
pP sep bxMe e5
h127 PrK/InFL InFR (Krivshenko)
28 Ph/Xa, ca
h129 pyd
h130 R Ly/In(3R)F, gm
h131 ra
h132 red (Malpigians)
23 red (Malpigians) e
h133 red (Malpigians) e
h134 ri
h135 ri bad e5/Me, In(3R)C,
Sb e 1(3)e
h136 ri e
h137 ri pp
h138 ri pp Ins /ru h D
InsCXF ca
h139 ri pp Ins /ru h D
InsCXF ca
h140 ri sbd e2
h141 ro
h142 ro Bd ca/In(3R),
2(3)a
h143 rs2
h144 rsd
h145 rsu
h146 ru h e5
h147 ru h ri
h148 ru h ri pP Inb
h149 ("threepi") ru h st
2(3)s e5
h150 ("ruca")/ru h th
st cu sr e5 ca
h151 ("ruca")/ru h th
st cu sr e5 ca
h152 ("rupes") ru h th st
pP cu sr e5
h153 ru h th st pP H e5 ro/
C(3)x, M(3)x e5
C(3)x = In(3LR)P
h154 ru st C3G e5 (iso 3)
(h se)
h155 ru st C3G e5
h156 ru tra p/ru h D
InsCXF e
h157 ru e6
h158 ry
h159 ry2
h160 Sb/In(3LR)UbxB101
h161 Sb bxD/Xa, T23
h162 Sb H/In(3R)C, cd
h163 Sb Ubx/Xa
h164 SbP/In(3LR)Cx
h165 sbP bx3
h166 se
h167 se h
h168 se ss
h169 se ss k e5 ro
h170 se51j
h171 se rt2 thMe,InL
h172 (*separated arms of 3"
Dubinin) T3L+41R+4R-
3R1 InLF Dfd InRP 1
h173 sep,InLR pP
h174 sep,InLR pP Pb/Sb/Me,
InL Dfd InRC e 13e
h175 sep,InLR pP Sb/Me,
InL InRC e 13e
h176 Ser/In(3R)C, e 1(3)e
h177 snb
h177 snb
h178 sr
h179 sr gl
h180 ss
h181 ss bx
h182 ss bx Sb2-ss
h183 ss bxk k e5/Xa
h184 ss ca {iso 1953}
h185 ss e5 {iso 1953}
h186 ss a
h187 ssF40a
h188 ssbB
h189 sSIn3/Sb bxD
h190 st
h191 st c3G ca/TM1, Me ri
Sb1 (sp2)
h192 st en in pP
h193 st Ki pP
h194 st Sb e5 rv ca
h195 st sr H2 ca/In(3R)P,1
st 1(3)W ca
h196 stp
h197 stS41ri pP
h198 su ve ru ve h th
h199 su ve ru ve bv
(h th?)
July 1961  Melanogaster - Stocks - Philadelphia

h202 th
h203 th cu sr e^s ro ca
h204 th st cp
h205 th st pb pP/ln(3L)Cx
h206 th st pb pP kar su^2-Hw
  Jy1 ss bx sr gl/TM1, Me ri Sb^1
h207 tra/Me,T23
h208 Tr1/rhu D InsCXF
h209 tt wo
h210 tx
h211 Ubx e^4/Payne, Dfd ca
  (Ubx=bx^D)

h212 ve
h213 ve bv (iso 1957)
h214 ve ca (iso 1953)
h215 ve h th
h216 ve R/ln(3L)F, gm
h217 ve R D^3 bx^D (es^?) Pr
c/a/lnP Dfd InRP ca
h218 ve R D^3 SoSp1 Bg^G/InsP
h219 ve st (iso 3)
h220 ve st sbd
h221 W
h222 W Sb/InsCXF
h223 wk/Payne, Dfd ca
h224 wo
h225 Xa,T23 ca/e^s cd ro
  cmp ca

Chromosome 4

j1 Bld,T12 InCyR/sc^2 pn; In^1
j2 ("scute,twelvepi") y sc%; al dp sc19i b pr cn vg c a px bw mr sp/al^2 Cy pr Bl
cn^2 L^4 sp^2
j3 sc^6 f In49 v; bw^VA/L^2 1 (iso Y,X,2)
j4 X-Y InEN v y; S dp Sp cn/dptx Cy cn (no free Y)
j5 y; S Sp cn/dptx Cy cn
j6 y ac; sc^19i/s2 Cy
j7 y f=; bw^VA/L^2 1
j8 f56e & y f=; cn bw
j9 y f=; Cy,Ins cn^2/da,InLR
j10 y f=; dp^tx Sp cn bw/s2^2 Cy cn bw
j11 y f=; net bw sp
j12 y Hw In49 m g/y sc^51 B Ins; net bw sp
j13 y sc^51 In49 v sp^5; dph b bw/dptx1 Cy pr Bl cn^2 y^4 sp^2
j14 y v fX:f^?in; bw^VA/L^2 1
j15 y^2 t^2; cn bw

X,2 (j1-j15)

j16 sc w B^33,Y^3 & y f= (B^33 Del.-Inser, into 3)
j17 sn^2; Mn/InLP InRP 1
j18 ("Tam tester 1") y f=; D^3 Sc/InLP Dfd InRP ca
j19 ("Tam tester 2") y sn oc e & y f^=; v y sc^51 B In49 1 snx^2 sc^6/j1 sc^1 oc pts; ru h
  D InsCXF/Me,Ins ri Sb^1
j20 ("Tam X3") TX3,red e & y f=; y
j21 ("Tam X3mn") sn TX3,red e & y f=; y
j22 w^a e & y v f=; y; tra/D InsCXF
j23 y Hw In49 m g/y sc^51 B Ins; ru bw
j24 y f=; su-ve ru ve bv (h? th?)

X,4 (j125-j127)

j25 y sc^51 InS sc^8 e & y f=; ci ey^R
j26 y f=; Cat/ci^D
j27 y f=; spa

Multiple Chromosomes
2,3 (128-1104)

j28 al h cn sp/al2 Cy Bl cn2 L4 sp2; ru
j29 "apl"/Cy sp; ru h InsCXF ca/Sb InRMO
j30 b; pP
j31 bw; e
j32 bw; ru h st d3 ri InRC e 13e/Me,Ins ri Sb1
j33 bw; ru h ri
j34 bw; ss
j35 bw sp; ru h D1 ri InRC e 13e/Me,Ins ri Sb1
j36 bw; st
j37 c; e
j38 cn bw; ri e39 cn bw; ru h th ri e3
j39 cn bw; ru h th ri e3
j40 cn crs/al2 Cy lt3 pr Bl cn2 L4 sp2; e3
j41 cn crs/Cy pr Bl cn2 L4 sp2; ve (iso)
j42 Cy/Pm; ru h D Ins CXF ca/InLP Dfd InRPF ca
j43 Cy/Pm; st (isc X,2,3)
j44 De bw; ro
j45 dp cu bw/Cy Fl cu2 L4 sp2; h ri e3
j46 dp03 cn bw; ru h D3 ri In3RC e 13e/Me,Ins ri Sb1
j47 dpT Sp cn bw/S2 Cy cn bw; ri e
j48 dpT Sp cn/s2 Cy cn; ri e
j49 dpT Sp cn/s2 Cy cn; ru h D3 ri InC e 13e/Me Ins ri Sb1
j50 dptx Sp cn/s2 Cy cn; ru h D InsCXF/Me Ins ri Sb1
j51 dptx Sp cn/s2 Cy cn; ru h D InsCXF Sb/Me,InL InC e 13e
j52 dptx Sp cn/s2 Cy cn; sep ri pP Sb Bl InL InC e 13e
j53 dptx Sp ms ta cn crs/s2 Cy pr Bl cn2 L4 sp2; e3
j54 dptx Sp pr cn/s2 Cy cn2; Me,InL InC e 13e/ru h CXF Sb
j55 dpV; vo3
j56 dpV2 cn bw; h ri e3 (iso 7/57)
75 fes ms cn sp/dptxI Cyc05 pr cn2; h ri e3/Me Ins ri Sb1 (iso 7/57)
76 InsNL InNSR mr/al2 Cy pr Bl lt3 cn2 L4 sp2; ri33
77 "(isier 1)" S Sp (crs)/Cy InL lt3; Me Ins ri Sb1/BdG
78 "(isier 2)" ms cn rm sp/al2 Cy lt3 pr Bl cn2 L4 sp2; ru h D InsCXF/ve th I
79 "(isier 3)" dp b cn c P/-al2 Cy lt3 pr Bl cn2 L4 sp2; ru h D Ins CXF/D; J e P1
80 M3a/al2 Cy pr Bl cn2 L4 sp2; ru
81 ms sp/Cy pr Bl cn2 L4 sp2; ri3 (iso)
82 net bw mr crs; Dl H e P1/ru h D Ins CXF (low iso 7.57)
83 net bw mr crs/dptxI Cy O pr cn2; ve bv/Me,Ins ri Sb1
84 net dp sp/dptxI Cy,0 pr cn2; Me,Ins ri Sb1/ve bv
85 "(Pale e") dp b cn c P/ Cy cn2; e p1/a P1
86 "(Pale H)" dp b cn c P/ Cy cn2; p56 DL H e P1/p56 In3R 1
87 "(Pale Indp)" indpt23 b P*-Dl H e P1/dp b Pm3; Sb In3R
87 S fes Sp T23B D3 ri Sb/Cy cn2 sp2; InsCXF
87 S fes Sp T2301, ms cn mr crs D3 st ri InC e 13e/al2,InMiss Cy pr Bl cn2 L4 sp2;
Me Ins ri Sb1
88 S fes Sp T2301 ms cn mr crs D3 st ri InC e 13e/dptxI Cy,Ins05 pr cn2; Me,Ins
ri Sb1
89 "(isier 0") S Sp P- T23,InsCXF/dptxI Cy,Ins05 pr cn2; DL H e P1
90 S Sp cn/dptxI Cy cn; h ri e3
91 S Sp cn/dptxI Cy cn; Me,InL InRC e 13e/ru h D Sb InsCXF
92 S Sp cn/dptxI Cy cn; ru h D3 ri InRC e 13e/Me,Ins ri Sb1
93 S Sp cn/dptxI Cy cn; ru h e11
94 S Sp cn/dptxI Cy,Ins05 pr cn2; h ri D3 InC e 13e/Me,Ins ri Sb1
95 S Sp cn/dptxI Cy,Ins05 pr cn2; ru h D InsCXF/Me,Ins ri Sb1
96 S Sp cn bw/dptxI Cy cn bw; h ri e3
97 S Sp cn bw/dptxI Cy cn bw; ru h D3 ri InRC e 13e/Me,Ins ri Sb1
98 S Sp ms ta cn crs/dptxI Cy pr Bl cn2 L4 sp2; e2
99 S Sp T23MI/dptxI InsCy,05 pr cn2; Me,Ins ri Sb1
100 S Sp T23B/dptxI Cy,Ins05 pr cn2; Me,Ins ri Sb1
101 S Sp T23B/dptxI Cy,Ins05 pr cn2; Me,Ins ri Sb1
j87 T23B cn bw InRC e 13e/Cy,Ins05; ru h D InsCXF (InAM?)
j88 T23B cn bw D3 ri/dp txI Cy,Ins05 pr cn2; Me ri Ins Sb1
j89 ta/Cy Bl cn2 L4 sp2; ru ri (iso)
j90 ta sp/Cy Bl cn2 L4 sp2; Jv (iso)
j91 ta sp/Cy cn2 L4 sp2; ru (iso)

j92 ("Tin") dp txI Cy,Ins05 pr cn2 T23 Me,Ins ri Sb1/S Sp cn; ru h D3 st InRC e 13e
j93 (2;4) "apl" 1/Cy cn2 sp2; IV-sim/ci ey
j94 (2;4) bw; c1D/IV-sim
j95 (3;4) bw; Cat/c1D
j96 (X,Y,2) Ytbw+/y v & sc ct n oc ptg car; y ct1,In In49 snx2; bw
j97 (X,Y,3) ("mult- ") X'Y ENEN y; st (no free Y)
j98 (X,Y,3) X'Y y; st (no free Y, no In)
j99 (X,Y,3) sc8'Y/X'Y ENEN y; ru h D InsCXF/ru tra p
j100 (X,Y,3) X'Y ENEN In49 y; st (no free Y)
j101 (X,Y,4) sc8'Y/x+ & y f=; ci gvl eyR syn
j102 (X,Y,4) Tp4'yX'Y ENEN In49 v y; ci gvl eyR syn
j103 (Y,2,3) scö'Y:bw+; dpv2 cn bw; h ri e8 (iso 7/57) (Cy Bl cn2 L4 sp2)
j104 (Y,2,3) Y:bw+; sc8'Y:bw+; fes ms cn sp/dp txI Cy,Ins05 pr cn2; h ri e8/Me,Ins ri Sb1

X.2.3 (j105-j112)

j105 ("MI") yS1 sc8 Ys3 In49 y3P; al2 Cy 1t3 cn2 sp2/dp b Fm1; ru h D InsCXF ca/Sb In3R
j106 ("Palex") w'; P/Cy; P1/P1
j107 yS1 sc8 B f In49 v1; T23B D3 st ri InC e 13e/dp txI Cy,Ins05 pr cn2; Me,Ins ri Sb1 (select AT )
j108 Y y=; cn bw; e
j109 Y In49 v; bw; e
j110 Y sc81 f In49 v sc8; bw; e
j111 yS1 sc8 B f In49 v; bw; e
j112 y sc81 In49 sc8; bw; st pp (to cross by sc8'Y/y B for losses, 1's & T's)
j113 (X,Y,2) ("scar") sc t2 v f car; Cy/bw; ey
j114 (Y,2,3) scö'Y:bw+; dpv2 cn bw; h ri e8 (iso 7/57) (Cy Bl cn2 L4 sp2)
j115 (Y,2,3) sc8'Y:bw+; fes ms cn sp/dp txI Cy,Ins05 pr cn2; h ri e8/Me,Ins ri Sb1
(jiso 7/57)
j116 (Y,2,3) sc8'Y:bw+; net bw mr crs/dp txI Cy,Ins05 pr cn2; Me,Ins ri Sb1 (iso 7/57)

X,Y,2.3 (j121-j131)

j121 ("Multipare") sc8'Y/y sc In49 BM1; twl bw; st 541
j122 ("Multipare ri") sc8'Yx2 y & y f=; twl bw; st 541
j123 ("Taxxy") sc8'Y/y sc In49 sn2 Bm1/y oc 1z'Ys; twl bw; st 541
j124 ("ny s cn bw e") YLcy/x'Ys; sc81 B f In49 v sc8; cn bw; e
j125 ("ny cn bw e") YLcy/x'Ys; cn bw; e
j126 sc6'Ys/y In49 v f'Y2; bw; e
j127 X'Y ENEN In49 y; cn bw; e (no free Y)
j128 X'Y ENEN In49 y; cn bw; ro (no free Y)
j129 (X,Y,2,3,4) Y f=; bw; e; ci eyR
j130 (X,Y,2,3,4) Y:InEN In49 Y:y2; cn bw; e; ci ey (no free Y)
j131 (X,Y,2,3,4) Y:InEN In49 Y:y2 & "snocty" y; cn bw; e; ci eyR (no free Y)

non-lethal "tumorous" stocks

m1 bw tu
m2 tu503
m3 tu51m
m4 tu'h
m5 vg tu
**SALT LAKE CITY, UTAH: UNIVERSITY OF UTAH**
Department of Genetics

Note: Stock list unchanged. See DIS 33, p. 53

**SYRACUSE, NEW YORK: SYRACUSE UNIVERSITY**
Department of Zoology

Several wild strains, each derived from a single inseminated female. Several polygenic crossveinless (cve) strains.

**TUSCON, ARIZONA: UNIVERSITY OF ARIZONA**
Department of Zoology

**ARGENTINA**

Buenos Aires: Atomic Energy Commission, Section of Genetics

Note: This list was copied from DIS 31, pg. 51 and DIS 32, pg. 40.

### Wild Stocks

- W1 Leningrad
- W2 Buenos Aires
- W3 Oregon-R

### Chromosome 1 (attached-X)

| Xb1 | 1z & y f:= |
| Xb2 | sc ct5 ca & y f:= |
| Xb3 | sc t2 v f & y f:= |
| Xb4 | y ac sc v & y f:= |
| Xb5 | y ac t2.Dp(yf ac+ scS1) & y f:= |
| Xb6 | y ct5 f & ac3 w2 ct f:= |
| Xb7 | y ct5 t2 v f c#: & y f:= |
| Xb8 | y.Dp(yf scV1) & y f:= |
| Xb9 | y In49 snx2 bl & y f:= |
| Xb10 | y sc lg v f & y f:= |
| Xb11 | y wm258-18 n2 v f & y f:= |
| Xb12 | sc8 B In49 & y f:= |
| Xb13 | sc8 B In49 m & y f:= |
| Xb14 | sc8 car f In49 v & y f:= |
| Xb15 | sc6 Tu wa & y f:= |
| Xb16 | yS1 sc6 B f In49 v wa & y f:= |
| Xb17 | yS1 sc6 f Ins wa & y f:= |
| Xb18 | scS1 f In49 v w & y f:= |
| Xb19 | y S1 B f In49 v & y f:= |
| Xb20 | sc8 sc8 & y f:= |
| Xb21 | scS1 B In49 sc8 & y f:= |
| Xb22 | yS1 scS1 B In49 sc8 & y f:= |
| Xb23 | scS1 At In49 v wa sc8 & y f:= |
| Xb24 | scS1 f In49 y wa sc8 & y f:= |
| Xb25 | scS1 f Ins Y3P & y f:= |
| Xb26 | YL f, yL & sc v f:= |
| Xb27 | y3L yL yS & y f:= |
| Xb28 | yS1 yS B YL & y f:= |
| Xb29 | YS, yS 2/y v f&YL & y f:= |
| Xb30 | scV1, yS In49 v B+YL & y f:= |
| Xb36 | sc8 v B+M, In & y w f:= |
July 1961 Melanogaster - Stocks - Argentina

Chromosome 3

III-1 e
III-2 ru h th st p^4 cu sr e^8

Chromosome 4

IV-1 Cat/gyl ey^R
IV-2 ci ey^R
IV-3 spa
IV-4 sy^R

Multichromosomal

M1 w^2 & y f:=; tra/D Ins CXF
M2 y sc^4 Ins w^6; S sc^191 Bl/Cy L^4 sp
M3 sc^19-{lJ1 sc^1}; fes sc^191 b pr/Cy
dp*x^1; pr cn^2
M4 y^Lc/x^8 & y v f:=; e
M5 y^Lc/x^y^8; bw; e
M6 y sc^2; al dp sc^191 b pr cn vg s a,
px; mr sp/ai^2 Cy pr Bl cn^2 L^4
sp^2
M7 y^2 t^2; cn bw
M8 bw; e
M9 y^81 sc^8 Ins y^3P; al^2 Cy lt^3 cn^2 sp^2/
dp b Pm^1; ru h D InsCXF ca/Sb
In^3R
M10 w^6; P/cy; P^1/p^1
M11 y f:=; cn bw; e
M12 sc t^2 v f car; Cy/bw; ey
M13 Y bb^-/v; bw^A/Bl L^2
M14 ("tester 1") y ac pn w rb wy^2 g^2 & y
f:=; sc^191/Cy
M15 ("tester 2") y^2 w^a cm wy^2 g^2 car & y
f:=; sc^191/Cy
M16 ("tester 3") y rb cm ras^2 g^2 & y f:=;
sc^191/Cy
M17 y ac sc pn w spl rb cx. & y f:=;
(sc^191(b pr)/)
M18 sc^8 B Ins w^6 sc^4 & y f:=; sc^191/Cy cn^2
M19 y sc^- (rein. sc^-4) w^a Ins bb & y f:=;
sc^191/Cy lt cn^2
M20 Y^c bw^+/X^c y f; bw (ring OK '57)
M21 sc^8:Y^c bw^+/ac^3; b bw
M22 Y^Lc/x^y^8; bw

Chromosome 2

II-1 ab^2 ms ta crs/Cy pr Bl cn^2 L^4 sp^2
II-2 al b cn sp (iso, 1954)
II-3 cn bw sp
II-4 net b cn crs/tp^xI Cy pr Bl lt^3
cn^4 L^4 sp^2 (iso, 1955)
## AUSTRALIA

**Brisbane, Queensland: University of Queensland**

Note: This list was copied from DIS 33, pg. 55

<table>
<thead>
<tr>
<th>Wild Stocks</th>
<th>Chromosome 1</th>
<th>Multichromosomal</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Oregon R-C</td>
<td>2 sc cv v</td>
<td>5 e11 dp</td>
</tr>
<tr>
<td></td>
<td>3 w</td>
<td>6 y; Cy/Fm, ds33k, H/Sb</td>
</tr>
<tr>
<td></td>
<td>4 z/B</td>
<td></td>
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</tbody>
</table>

## AUSTRIA

**Vienna: Institut für allgemeine Biologie**

Note: This list was copied from DIS 28, pg 51

<table>
<thead>
<tr>
<th>Wild Stocks</th>
<th>Chromosome 1</th>
<th>Chromosome 2</th>
<th>Chromosome 3</th>
<th>Chromosome 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Oregon-S</td>
<td>30 y</td>
<td>36 b pr vg a sp</td>
<td>48 ca</td>
<td>61 ey2</td>
</tr>
<tr>
<td></td>
<td>31 y w</td>
<td>37 bs (4 different alleles)</td>
<td>49 c3G</td>
<td>Multichromosomal</td>
</tr>
<tr>
<td></td>
<td>32 y ec ct6 v wy2 car</td>
<td>43 al dp</td>
<td>50 e</td>
<td>62 Bx; J</td>
</tr>
<tr>
<td></td>
<td>33 y v f</td>
<td>44 ex</td>
<td>51 e cu</td>
<td>63 v; cn bw</td>
</tr>
<tr>
<td>2 Oregon-R-c (Df(2)Ore)</td>
<td>34 y/f</td>
<td>45 j</td>
<td>52 gl</td>
<td>64 y v f/+; bw; e wo ro; ey2</td>
</tr>
<tr>
<td></td>
<td>35 bo</td>
<td>46 12/Cy</td>
<td>53 jv se</td>
<td>Aberrations</td>
</tr>
<tr>
<td>3 Oregon-R-c P</td>
<td></td>
<td></td>
<td>54 ru se h st bv</td>
<td></td>
</tr>
<tr>
<td>4 8 strains from different places in Austria</td>
<td></td>
<td></td>
<td></td>
<td>65 Df(1)N8/g y Hw dl-49 m2 g4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>66 Df(2)vg5, cn/Cy, al2 lt3</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>67 Dp(1;1f)135 y2; In(1)sc8, Df(0+ac) wa sc8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>68 In(2LR)Gla/Cy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>69 T(2,3)bw/Cy</td>
</tr>
</tbody>
</table>

## BRAZIL

**Curitiba, Paraná: Universidade do Paraná, Faculdade de Filosofia, Ciências e Letras, Laboratório de Genética**

Note: This list was copied from DIS 32, pg 43.

<table>
<thead>
<tr>
<th>Wild Stocks</th>
<th>Curitiba, Paraná</th>
<th>Florianópolis, Santa Catarina</th>
<th>Ponta Grossa, Paraná</th>
<th>Petrolina, Pernambuco</th>
</tr>
</thead>
<tbody>
<tr>
<td>Boa Esperança, Minas Gerais</td>
<td>Florianópolis, Santa Catarina</td>
<td>Sant'Ana, Santa Catarina</td>
<td>Salvador, Bahia</td>
<td>Santa Felicidade, Paraná</td>
</tr>
<tr>
<td>Buenos Aires, Argentina</td>
<td>Goiânia, Goiás</td>
<td>Gruta, Argentina</td>
<td>São Paulo, São Paulo</td>
<td>Teixeira Soares, Paraná</td>
</tr>
<tr>
<td>Campina Grande, Paraíba</td>
<td>Irati, Santa Catarina</td>
<td>Lins, São Paulo</td>
<td>Uberlândia, Minas Gerais</td>
<td>Xapecó, Santa Catarina</td>
</tr>
<tr>
<td>Cosmópolis, São Paulo</td>
<td></td>
<td>Paranaguá, Paraná</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
CANADA

Toronto: University of Toronto

Note: Inbred stocks of Marvin Barr Seiger (see DIS 32, pg. 156)

Oregon-R: 291 generations of inbreeding as of 60k28
Ives Oregon-R: 311 generations
M Oregon R: 284 generations
P1 Oregon R: 332 generations
2b Oregon R-C: Lost generation 256
f Oregon R: 221 generations
y Oregon R: 220 generations
Canton-S: 97 generations

Ives Oregon R: mass culture, extracted from inbred stock at generation 300
f Oregon-R: mass culture, extracted from inbred stock at generation 200
y Oregon-R: mass culture, extracted from inbred stock at generation 200

Multichromosomal
1 yf:=; bw; e; pol (1;2;3;4)
2 vv/FM6; DebD; linhu (1;2;3)
3 vV/v3; bwD; Hu (1;2;3)
4 vV/3; sp bwD; Sb (1;2;3)
5 bw; st (2;3)

FRANCE

Lyon: Faculté des Sciences, Laboratoire de Zoologie

Note: This stock list copied from DIS 29, pg. 51.

Wild Stocks Champetière (inbred) Lyon

GERMANY

Göttingen: Max-Planck-Institut für Tierzucht und Tierernährung

Note: This stock list copied from DIS 33, pg. 60

Wild Stocks

<table>
<thead>
<tr>
<th>Chromosome 1</th>
<th>Chromosome 2</th>
<th>Chromosome 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 normal (Berlin wild)</td>
<td>12 fes ltl L4/Cy al2 lt3</td>
<td>17 ciD/+</td>
</tr>
<tr>
<td>2 br ec rb</td>
<td>13 f3 px sp</td>
<td>18 eyD/+</td>
</tr>
<tr>
<td>3 C1B/+</td>
<td>14 lg1 cn bw/Cy on bw</td>
<td>Multichromosomal</td>
</tr>
<tr>
<td>4 sc8 y/fx sc8 y X c2 y v</td>
<td>15 pb/c Ms SB C</td>
<td>19 w; j; e11; ey2</td>
</tr>
<tr>
<td>5 svr</td>
<td>16 ss1</td>
<td>20 Cy/Pm ds33k; h/C Sb</td>
</tr>
<tr>
<td>6 w^</td>
<td></td>
<td>21 L Cy/+; C Ms Sb C/+</td>
</tr>
<tr>
<td>7 wch wy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 wco y f</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 wco y f</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10 w sn3 B</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11 y w bb</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Hamburg: Zoologisches Staatsinstitut und Zoologisches Museum

Note: This stock list copied from DIS 32:49.

**Wild Stocks**

<table>
<thead>
<tr>
<th>Chromosome 3</th>
<th>7 cu</th>
<th>8 e cu</th>
<th>9 Sb/H Payne</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 w&lt;sup&gt;1&lt;/sup&gt;</td>
<td>13 bw; cu; ey&lt;sup&gt;2&lt;/sup&gt;</td>
<td>14 cu; ey&lt;sup&gt;2&lt;/sup&gt;</td>
<td>15 X; ey&lt;sup&gt;2&lt;/sup&gt;</td>
</tr>
<tr>
<td>3 X/f</td>
<td>10 ey&lt;sup&gt;2&lt;/sup&gt;</td>
<td>Triploid</td>
<td></td>
</tr>
<tr>
<td>Chromosome 2</td>
<td>Multichromosomal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 bw</td>
<td>11 bw; cu</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 dp b</td>
<td>12 bw; ey&lt;sup&gt;2&lt;/sup&gt;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 Pm L Cy</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Hamburg-Eppendorf: Universitäts-Frauenklinik, Strahlenbiologische Abteilung**

Note: This stock list copied from DIS 28:56

**Wild Stocks**

| 5 w | 6 X<sup>0</sup>/ClB |
| Chromosome 1 (X) | Multichromosomal |
| 2 ClB/+ | 7 cn; ss |
| 3 scS<sup>1</sup> B InS w<sup>a</sup> sc<sup>8</sup> | Attached-X |
| 4 sc<sup>8</sup> Y/Y<sup>f</sup> x sc<sup>8</sup> X<sup>0</sup>2 Y v | 8 X |

### GREAT BRITAIN

**Bayfordbury, Hertford, Herts, England: John Innes Horticultural Institution**

Note: This stock list copied from DIS 33:62.

**Wild Stocks**

| Bayfordbury | 5 Bayfordbury (A) | 9 v |
| Hampton Hill | 6 Bayfordbury (B) | 10 w |
| Samarkand | 7 Oregon | 11 y w |
| Teddington | 8 Samarkand |

| 12 b pr vg |
| Multichromosomal |
| 13 Cy L<sup>4</sup>/Pm; H/Sb |

### Glasgow, Scotland: University of Glasgow, Department of Genetics

Note: This stock list copied from DIS 32:51.

**Wild Stocks**

| Florida-4 (inbred) | 3 B | 7 sn<sup>3</sup> | 13 w<sup>c0</sup> | 19 w<sup>t</sup> |
| Oregon-K (inbred) | 4 gt w<sup>e</sup> | 9 w | 14 w<sup>e</sup> | 20 y<sup>e</sup> ec |
| 5 sc w<sup>c</sup> ec ev | 10 wFF33 | 15 w<sup>h</sup> | 21 w<sup>e</sup> z (zeste) |
| 6 sc w<sup>bl</sup> ec cv | 11 w<sup>a</sup> | 17 w sn<sup>3</sup> | 22 w<sup>14</sup>G<sup>2</sup> z (zeste) |

| 12 wbf2 | 18 w sn<sup>3</sup> B |
July 1961

Melanogaster - Stocks - Great Britain

Attached-X
23 y v f
Chromosome 2
2b cn vg
25 bw

26 E
Chromosome 3
29 bw; v
30 sn p
Inversions
31 Muller-5

Harwell, Berks., England: Medical Research Council,
Radiobiological Research Unit

Note: This stock list copied from DIS 33:63.

Wild Stocks
1 Oregon-K

Inbred lines
2 Crianlarich (186 gens.)
3 Kaduna (90)
4 It (18)
5 Nettlebed (268)
6 Oregon-R (336)
7 Oregon-S (229)
8 stw (18)
9 Wild Edinburgh (260)

18 Cy/Bl L2
19 ds dp
20 el
21 hk pr
22 ho ed cl
23 lt
24 ltd
25 ltd cn
26 met
27 pd
28 pr
29 stw
30 stw lt

INdIA
Calcutta: Indian Statistical Institute

Note: This list copied from DIS 34:37-38.

Wild Stock
1 Canton-S

Chromosome 1
13 wbf
14 wbl
15 wc
16 wc
17 w h
18 w i
19 y
20 y

Chromosome 2
21 bw
22 cn
23 dp
24 ho
25 px
26 vg
27 vg bw

Chromosome 3
28 cu
29 es
30 Ly/b3
31 ry
32 se
33 as

Chromosome 4
34 ci w
35 ey2

Multichromosomal
36 Cy/Pm D/Sb
Melanogaster - Stocks - India  
July 1961

**Calcutta: University of Calcutta, Department of Zoology**

Note: This stock list was copied from DIS 32:53.

<table>
<thead>
<tr>
<th>Wild Stocks</th>
<th>Chromosome 2</th>
<th>Chromosome 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>a1 Canton-S</td>
<td>c2 L^4</td>
<td>d3 Ly/D^3</td>
</tr>
<tr>
<td>a2 Oregon</td>
<td>c3 vg</td>
<td>d4 es</td>
</tr>
<tr>
<td></td>
<td>c4 ho</td>
<td></td>
</tr>
</tbody>
</table>

**Wild Stocks Chromosome 1**

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>b1 y Hw 49</td>
<td>v^2 m^2, f/C1B36d</td>
<td></td>
</tr>
<tr>
<td>b3 x^a-2</td>
<td>y/y w f</td>
<td></td>
</tr>
<tr>
<td>b6 y</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Chromosome 1**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>y</td>
<td></td>
</tr>
<tr>
<td>Hw</td>
<td></td>
</tr>
<tr>
<td>49</td>
<td></td>
</tr>
</tbody>
</table>

**Chromosome 2**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>d1 se h</td>
<td></td>
</tr>
<tr>
<td>d2 ssa</td>
<td></td>
</tr>
</tbody>
</table>

**Multichromosomal**

| f1 Cy/Pm; D/Sb |

**Hiroshima: Hiroshima University, Zoological Laboratory**

Note: This stock list was copied from DIS 33:68.

<table>
<thead>
<tr>
<th>Wild Stocks</th>
<th>Chromosome 1</th>
<th>Chromosome 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Kandy, Ceylon</td>
<td>10 BB</td>
<td>14 e</td>
</tr>
<tr>
<td>2 Hiroshima</td>
<td>11 w</td>
<td></td>
</tr>
<tr>
<td>3 Matsuyama</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 Miyakonojio</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 Naze</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6 Oregon-R</td>
<td>12 S/Cy E-S</td>
<td>15 Muller-5; Cy/Pm; Sb,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>e/Ubx^130 e</td>
</tr>
<tr>
<td>7 Samarkand</td>
<td>13 vg</td>
<td>16 y; bw; e; ci, eyR</td>
</tr>
<tr>
<td>8 Sapporo</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9 Taichu, Formosa</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Mitaka, Tokyo: International Christian University**

Note: This stock list was copied from DIS 33:70.

<table>
<thead>
<tr>
<th>Wild Stocks</th>
<th>Mutants</th>
<th>Mutants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oregon-S</td>
<td>Muller-5</td>
<td>w^8</td>
</tr>
<tr>
<td>Tokyo</td>
<td>y</td>
<td>w m f</td>
</tr>
<tr>
<td></td>
<td>w</td>
<td>cu</td>
</tr>
<tr>
<td></td>
<td>wa</td>
<td>e</td>
</tr>
</tbody>
</table>

**NETHERLANDS**

**Leiden: Rijksuniversiteit, Laboratorium voor Stralengenetica**

Note: This stock list was copied from DIS 33:74-75.

<table>
<thead>
<tr>
<th>Wild Stocks</th>
<th>Mutants</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Oregon-K</td>
<td>6 f B odsy car</td>
</tr>
<tr>
<td></td>
<td>7 fu^v f/C1B</td>
</tr>
<tr>
<td></td>
<td>8 g^2</td>
</tr>
<tr>
<td></td>
<td>9 pn</td>
</tr>
<tr>
<td></td>
<td>10 pr^27-9 cv v f^3N</td>
</tr>
<tr>
<td></td>
<td>11 ras dy</td>
</tr>
<tr>
<td></td>
<td>12 rb</td>
</tr>
<tr>
<td></td>
<td>13 rb^27-4 v f^3N &amp; y f:</td>
</tr>
<tr>
<td></td>
<td>14 sc^8 S^1 B In-S wa sc^8 (Muller-5)</td>
</tr>
<tr>
<td></td>
<td>15 sw</td>
</tr>
</tbody>
</table>
35 : July 1961
Melanogaster - Stocks - Netherlands

16 "tester 1-y" y ac pn w rb wy2 g2 & y f:=
17 "tester 2-y" y2 wa cm wy2 g2 car & y f:=
18 "tester 3-y" y rb cm ras2 g2 & y f:=
19 un4 Bx2 & y f:=
20 w sn B
21 y ac sc pn w rb cm ct6 ras2 v g2 f car & y f:= sc19i/Cy, In-L 1t ("Maple")
22 y sc51 B In-49 sp2 sc8/oc ptg
23 y sc51 n49 v sc8
24 y w In-49 f
25 y w ac cv v f
26 XO2 y B & y f:=

Altered Y's

27 X Y In-EN v ptg oc sn5 & y sc t2 v f car:= (no free Y)
28 X Y In-EN y; st (no free Y) ("Multi 9")
29 1 J1+y/y J1 sc-J1(y) In-49 ptg oc B1/ y51 sc51 in car od sy f g2 dy v ras2 sn3 ct6 cm rb ec w 1 pn sc8 ("Maxy-
nee")
30 1 J1+y/y sc4 B v41b & y sc4 B v41b/y w In-49 12s
31 1 J1+y/y In-49 r51 B M1
32 sc8 Y/y sc51 B f In-49 v & y f:=/ sc8 y
33 sc8 Y/y In-49 v ptg oc r51 pM1 y s51, sc51, sc51 in car od sy f g2 dy v ras2 sn3 ct6 cm rb ec w 1 pn sc8
34 sc8 Y/y In-49 B; bw D & y f:=; bw D ("Multi 9")
35 YLC/X.Y5 ("Sterilizer +")
36 YLC/X.Y5; cn bw; e ("Sterilizer cn bw e")

Chromosome 2

37 b pr vg
38 dp
39 dp b cn bw
40 dp stw3 bw
41 dp Tp Cy, In-L pr cn2 In-Cy R-O/Ins-NSL Ins-NSR px sp ("Cy Oster")
42 dp Th Cy cn In-49 Sc sp1 n49 sc cp bw
43 fes mg cn sp/net dp txI Cy b pr Bl 1t3 cn2 14 sp2
44 1s dp Sp ms ta cn crs/3 Cy 1t3 pr+ Bl cn2 14 sp2
45 net bw mr crs/al2, In-Mis dp txI Cy Bl cn2 1t3 sp2
46 S fes Sp ms cn mr crs/al2 In, Mis dp txI Cy pr Bl cn2 1t4 sp2

Chromosome 4

47 Cl D/sp Cat

Multichromosomal

48 y sc51 In-49 sc8; dp b cn bw
49 cn bw; e
50 Cy/Pm; Cx, D/In(3R)Sb
51 y sc51 In-49 sc8; bw; st p

Leiden: Rijksuniversiteit, Genetisch Laboratorium

Note: This stock list was copied from DIS 32:62
Only some unusual stocks are listed.

Chromosome 2

on bw Dr/Pm
on px bw Kr/Cy
crc cn/Pm

Chromosome 3

Triploid

g 3N/W/FM4 & Y/FM4 &

Utrecht: Rijksuniversiteit, Genetisch Instituut

Note: This stock list was copied from DIS 33:75-76.

Wild Stocks

5 f2/CI B
6 g2
7 pn
8 ras dy
9 rb
10 rb27-4 cv v f3N & y f:=
11 sc cv v f
12 sc51 B In-S w2 sc8
13 ("tester 1") y ac pn w rb wy2 g2 & y f:=; sc191/Cy
**Melanogaster - Stocks - Netherlands** July 1961

| 14 | "tester 2") y^2 w^a cm wy^2 g^2 car & y f:==; sc191/Cy |
| 15 | "tester 3") y rb cm ras^2 g^2 & y f:==; sc191/Cy |
| 16 | w sn B |
| 17 | y scSI B In-49 snx^2 scg^2/oc ptg |
| 18 | y sc^2 B f In-49 v |
| 19 | y w m B |
| 20 | y w^a cv f |

**Altered Y's**

| 21 | X·Y·In-EN y; st (no free Y) ("Multi ii") |
| 22 | 1J1+Y/1J1 sc^1(+) In-49 ptg oc BM1/ySl |
| 23 | sc^1, In car odsy f g^2 dy v ras^2 sn^3 |
| 24 | ct^6 cm rb ec w l pn sc^8 ("Maxy-new") |
| 25 | sc^2-Y/y In(1)49 B; bw^D & y f:=; bw^D |

**Chromosome 2**

| 29 | b pr vg |
| 30 | bw |
| 31 | Bl L/Cy |
| 32 | dp |
| 33 | dp b cn bw |

| 34 | dp^Th Cy, In-L pr cn^2 In(2)Cy R-)/ |
| 35 | Ins-NSL Ins-NSR px sp |

**Chromosome 3**

| 37 | e |
| 38 | h ri |
| 39 | 1 tr/e In(3R) In(3L) |
| 40 | Mio/In(3R)Sb |
| 41 | St |

**Chromosome 4**

| 43 | ci |
| 44 | ciD/spaCat |

**Multichromosomal**

| 45 | y scSI In-49 sc^8; dp b cn bw |
| 46 | 6w^a & y v f:=; tra/D Ins-CXF |

**Deficiencies**

| 53 | Df(1)N^3/dl-49, y Hw m^2 g^4 |
| 54 | Df(1)N^2/dl-49 y Hw m^2 g^4 |
| 55 | Df(1)N^3/dl-49 y^3d sc^8 dm B |

**SOUTH AFRICA**

**Johannesburg: University of the Witwatersrand, Department of Zoology**

Note: This stock list copied from DIS 33:78

<table>
<thead>
<tr>
<th>Wild Stocks</th>
<th>Chromosome 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Bethulie</td>
<td>17 bl ct^6 g^2</td>
</tr>
<tr>
<td>2 Bloemfontein</td>
<td>18 bo</td>
</tr>
<tr>
<td>3 Canton-S</td>
<td>19 B</td>
</tr>
<tr>
<td>4 Cape Town</td>
<td>20 car</td>
</tr>
<tr>
<td>5 Cedar</td>
<td>21 car^2</td>
</tr>
<tr>
<td>6 Florida</td>
<td>22 cm</td>
</tr>
<tr>
<td>7 Graaff-Reinet</td>
<td>23 cm car</td>
</tr>
<tr>
<td>8 Inhaca Island</td>
<td>24 cm g^3 car</td>
</tr>
<tr>
<td>9 Johannesburg</td>
<td>25 ct v</td>
</tr>
<tr>
<td>10 Limpopo</td>
<td>26 ct^2 v dy g</td>
</tr>
<tr>
<td>11 Nelspruit</td>
<td>27 ct^6</td>
</tr>
<tr>
<td>12 Oregon-R</td>
<td>28 ec</td>
</tr>
<tr>
<td>13 Stanford Lake</td>
<td>29 ec ct^6 v g^3</td>
</tr>
<tr>
<td>14 Stellenbosch</td>
<td>30 f^3 B</td>
</tr>
<tr>
<td>15 West Rand</td>
<td>31 f^2 m</td>
</tr>
<tr>
<td>16 Zoutpansberg</td>
<td>32 f^5 v</td>
</tr>
</tbody>
</table>

| 25 | f^5 v |
| 33 | g |
| 34 | g^2 |
| 35 | g^3 |
| 36 | m |
| 37 | pn^2 |
| 38 | ras dy |
| 39 | ras^2 |
| 40 | ras^3 m |
| 41 | rb |
| 42 | rb car |
| 43 | rb cm g^3 |
| 44 | rb cm car |
| 45 | rb cx |
| 46 | rb g^4 |
| 47 | rb g^3 car |
| 48 | sc ec cv ct^6 v g^2 f |
| 49 | sc ec cv ct^6 v g^2 f/FM3 |
| 50 | svr wa  |
| 51 | v |
| 52 | v^36f |
| 53 | v g^3 |
July 1961

Melanogaster - Stocks - South Africa

54 w
55 w m
56 w m f
57 w a
58 w a3
59 w a4
60 w a rb
61 w h
62 w c h
63 w c o sn2
64 w c o l
65 w e
66 w e2
67 w e3
68 w e car
69 w e cm
70 w e g3
71 w e rb
72 w e rb car
73 w s at
74 w t f w
75 w w f5
76 w w rb 77 y
77 y
78 y g4
79 y m
80 y pr
81 y rb
82 y w
83 y w m
84 y2 su-wa wa bb
85 y2 w a w

Chromosome 2

86 albasp/Cy L sp2
87 al dp b pr Bl c px sp/
SM1 al2 Cy sp2
88 a sp2
89 b
90 b pr
91 b pr cn
92 b pr cn a
93 bw
94 bw2b
95 bw4
96 bw D
97 c px

98 cn
99 cn35k
100 cn vg
101 cl
102 cl 50a
103 dke c
104 dp
105 lt std/cy sp2
106 lt stdw3
107 lt d
108 pd
109 pr
110 pr 42d
111 sf2
112 Su-H/Cy, pr
113 tk stf2 abb
114 vg
115 vgdn

Chromosome 3

116 ca
117 cd
118 cu Kar
119 D/Gl
120 e
121 es8
122 es8 cd ro cmp ca/Xa, ca
123 ma fl
124 ma
125 p
126 p P cu
127 p P cu sr es8
128 res
129 ru
130 ry
131 se
132 sr
133 stsp
134 suB-pr/In(3R)C, e; pr
135 th st
136 th st pP

Chromosome 4

137 ci ey

Multichromosomal

138 bw; e
139 bw; e; ci ey
140 bw; ci ey
141 bw; st
142 Cy/Pm; ds33k; H/
In(3R)Mo; sr
143 g3; bw
144 g3; st
145 g3; st pP
146 ras6; st
147 rb; bw
148 rb; ry
149 rb; se
150 rb; st
151 car; ry
152 car; se
153 vg; se
154 we rb; se
155 we; cd
156 y; bw; e; ci ey

Attached-X

157 f B/suS2-v-pr v
158 y +
159 y2 su-wa wa bb/y

Inversions

160 In(1)A99b
161 In(1)dl, y fan
162 In(1)rst3, y rst3
car bb
163 In(1)rst3, y rst3
g3; car
164 In(1)w m4
165 In(1)w m4; bw
166 In(1)w m4; st
167 In(1)sc81, S, sc81
we B sc83

Translocations

168 T(1;3)04, D/ClB
169 T(1;4)w m5

Spain

Madrid: Centro de Investigaciones Biológicas, Laboratorio de Genética

Note: This stock list was copied from DIS 33:79

Wild Stocks

Madrid
Ribadeo
Ronda 10
Mallorca
Rocafort
Ronda 30
SWEDEN

Stockholm: University of Stockholm, Institute of Genetics

Wild Stocks

1. Algeria
2. Canton-S
3. Djursholm 55
4. Florida
5. Karlsnäs
6. Oregon
7. Stäket
8. Tunnelgatan
9. Örebro
10. Skäftö

Chromosome 1

11. B
12. bb
13. B; sc8\(Y\)\(\phi\) c y f:=; sc8\(Y\)\(\phi\)
14. B car; sc8\(Y\)\(\phi\) y f:=; sc8\(Y\)\(\phi\)
15. BB car; sc8\(Y\)\(\phi\) y f:=; sc8\(Y\)\(\phi\)
16. Bx2
17. car
18. cm ct6 sn3\(\phi\) y f:=
19. cv
20. cv sn
21. cv y B\(\phi\) y f:=
22. ct6
23. ec ct v f
24. f
25. f B
26. f BB; sc8\(Y\)\(\phi\) y f:=; sc8\(Y\)\(\phi\)
27. fa
28. fu; sc8\(Y\)\(\phi\) y f:=; sc8\(Y\)\(\phi\)
29. g2
30. g f car\(\phi\) y f:=
31. lw29a H0\(\phi\) y Hw g In-49 m
32. lw7b H0\(\phi\) y sc8\(Y\) f In-49 V wa
33. lz\(\phi\) y f:=
34. m
35. m f
36. Df(1)N8/wa
37. od car
38. pn
39. rb
40. sc
41. sc8
42. sc cv
43. sc cv v f
44. sc cv v car
45. sc eq cv ct6 v g f/FM3; y31d sc8 dm B 1
46. sc t2 v f Tu car\(\phi\) y f:=
47. sc81 B In-S wa sc8\(Y\)
48. sc81 B In-S wa sc8; y sc8\(Y\)
49. sn3
50. v
51. w
52. w cv
53. w cv sn3
54. w sn3

Chromosome 2

94. a px sp
95. a px or
96. al b c sp
97. al sp b pr B1 c px sp/SM1, al2 Cy sp2
98. al dp b pr c px sp/al2 Cy lt3 L4 sp2
99. al dp b pr cn vg c a px bw mr sp/ S2 Cy
100. al2 Cy lt3 L4 sp2/fm
101. al S ast ho/Cy, E-S
102. al sp
103. b
104. b cn vg
105. b pr vg
July 1961

Melanogaster - Stocks - Sweden

106 Bl/In(2LR)dp
107 bw
108 cn bw
109 cn vg bw
110 Cy/Pm
111 dp b
112 dp b pr c px sp
113 ed Su2-dx
114 ft
115 L²/Cy
116 L⁴
117 pr
118 px bw mr sp/ds²³K-pm
119 S² Cy pr Bl cn² L⁴ bw sp/In-NSL
   In *NSR px sp
120 sca
121 sp
122 stw³
123 vg

Chromosome 3

125 ca
126 D³/In-P
127 es⁳
128 gl
129 Hn²
130 ri²
131 ro
132 ru Hn²
133 ru h st pP ss es³

134 ru se h st pP ss es³
135 ru h th st cu sr es³ Pr ca/M⁶,
   T(2;3)
136 se ss k es³ ro
137 ss
138 st
139 st ss e¹
140 ve h th
141 ss e¹

Chromosomes 4

142 ey
143 ci ey

Multichromosomal

144 cn bw; e¹
145 bw; st
146 rb cm ras² c;²; sc¹⁹²/Cy ³ f=:
   sc¹⁹²/Cy ³
147 sc cv v; ri
148 sp; th
149 T¹(1;2)B¹⁴/Cy ³ M²e/Cy ³
150 T¹(1;2)B¹³/Cy ³
151 TX(16A¹)² B³ ³ w f=:
152 T¹(2;3)M³/Cy
153 It¹/T¹(1;2)A
154 y; v; bw¹A²/L² ¹
155 y; pr; ss
156 = +; sv³, ³ +; sv³ ³

Uppsala: Botanik-Genetiska Institutionen, Lantbrukshogskolan

Note: This stock list was copied from DIS 23:65-66.

Wild Stocks

1 Algeria
2 America
3 Bayforbury
4 Boa Esperanca, Minas Gerais, Brazil
5 Canton-S
6 Crimea
7 Curitiba, Paraná, Brazil
8 Florida
9 Gruta, Argentina
10 Håkone-R (resistant to BHC, DDT, parathione, nicotine)
11 Karos
12 Kochi-R (resistant to parathione)
13 Oregon
14 Salvador, Bahia, Brazil
15 Samarand
16 San Miguel, Buenos Aires, Argentina
17 Stäket
18 Tunnelgatan
19 Örebro
20 Örebro-R (resistant to parathione)
35:44

Melanogaster - Stocks - Sweden
July 1961

45 w
46 w ct
47 w cv
48 w cv sn^3
49 w sn^3
50 w^a
51 w^ab
52 w^a su-f^d y f: = q
53 wbf^2
54 wbf f^5
55 wbl
56 wch^w y
57 wco
58 wco sn^2
59 we
60 we^2
61 we^2 en-w^e o^' y f: = q
62 wh
63 w^a ct
64 w^a yb
65 w^sat
66 y
67 y ac sc pn w rb cm ct^6 sn^3 ras^4 v m g
  f car/scs^1 B InS w^a sc^8
68 y ec ct v.f
69 y f Rb/scs^1 B InS w^a sc^8
70 y^c sc w^a wch fa o^' y f: = q
71 y^c sc w^a
72 y^c sc w^a wch o^' y f: = q
73 y^c su-w^a w^a w^e o^' y f: = q
74 y^c w^a
75 y^c w^a v
76 y z
77 z ec
78 z we^2 ec^4
79 z w^11 E^4

Chromosome 2

80 bw

81 cn vg bw
82 Cy/Pm 83
83 Cy/S
84 fes lt^3/cy al^2 lt^3 L^4 sp^2
85 net
86 pr
87 S/NS, px sp
88 vg

Chromosome 3

89 D^3/InP
90 ri^2
91 ro
92 ru h st pP ss e^8
93 se
94 ss
95 st ss e^11

Chromosome 4

96 ey
97 sv^n

Multichromosomal

98 cr-u/Cy; (w^e)
99 Cy/S; D/InP
100 In(1)w^b y^51; E-Var 4/Cy
101 In(1)w^b'; E-Var 5/Cy
102 In(1)w^b'; E-Var 8/Cy
103 L^2/+; sp; th
104 sp; th
105 T(2;3)bw^De^4/Cy
106 T(1;4)w^m^5
107 wch; Se-wch/Cy
108 y^S^1 sc^8 InS y^3P; al^2 Cy lt^3 sp^2/dp b Pm^4;
   ru h D^3 InCXF ca/Sb In(3R)
Report of A. B. Burdick

**dy<sup>58k</sup>: dusky-58k** M.E.Krawinkel, 1958k. Spontaneous in an isogenic pol stock, W-160 pol. A bona fide new mutant since dy, the only other dy mutant known at the time dy<sup>58k</sup> was discovered, was not present in stocks at this institution at that time, and the pol marker of the stock in which it occurred was present in the single mutant male observed. Recombination and complementation tests show that dy<sup>58k</sup> is the right-most known element of the m-dy complex. It recombines with Df(1)259-4, with all m's except m<sup>D</sup>, and not with dy. It appears to be the right-most element because it consistently shows higher recombination and more complementation with m-type mutants than does either m<sup>D</sup> or dy. Wing length is shorter than dy, about the same length as the longer m-type mutants. Fertile in both sexes. RK1.

**m<sup>59a</sup>: miniature-59a** M.E.Krawinkel, 1959a. Probably spontaneous in an isogenic wild-type background (W-126) which several generations before had been treated with about 50r of X-ray. Shows low complementation with m-mutants and high with dy-mutants. Recombines with Df(1)259-4 on its left and m on its right. No m-mutant (except Df(1)259-4) has been shown to be left of m<sup>59a</sup>. Female fertility very low; male fertile. RK2.

**dy<sup>60k</sup>: dusky-60k** A.B.Burdick, 1960k. Spontaneous in a SM5, a<sup>12</sup> l<sup>v</sup> Cy sp<sup>2</sup>/da stock; isolated in uniform stock background so that we know that it could not have arisen as a contamination. Allele tested with m<sup>60l</sup> and dy<sup>61a</sup> (see DIS-35, new mutant report of P. T. Ives); shows high complementation with m<sup>60l</sup> and low with dy<sup>61a</sup>. Similar to other dy's; fully fertile in both sexes. RK2.

**rk<sup>4</sup>: rickets-4** R.C.Jackson, 1954. Originally called cq (creeper) in DIS-28, 1954. H. U. Meyer in DIS-32 reports cq to be an allele of Edmondson's rk. Therefore, cq is now rk<sup>4</sup>.

**r<sup>58a</sup>: rudimentary-58a** M.Burdick, 1958. Induced in mature wild-type sperm by 4000r X-ray. Resembles r (Morgan, 10f) and r<sup>9</sup> (Bridges, 20b3), wings truncated, blistered, wing-veins, particularly l<sup>4</sup>, frequently interrupted, marginal bristles sparse, somewhat longer, and disarrayed. Expression variable but does not overlap wild-type. Female entirely sterile. Recombination tested in 2894 flies to car and 974 to f giving confidence intervals that include 1-54.5, Morgan's locus for r. Functionally allelic with r<sup>9</sup>. RK2.

Report of W. W. Doane

(This report supersedes that in DIS-34 inadvertently attributed to S. Counce.)

**fs<sub>2</sub>adp; female sterile(2)dipose** Counce, 1956. 2-83+ Pub. Doane, 1959. Genetics 44:506; developmental and physiological studies, Doane, 1960, Ph.D. Thesis, Yale Univ. Spontaneous in Kaduna wild stock maintained in Edinburgh. Adult fat body hypertrophies as cells become distored by enormous oil globules. Abnormal fat bodies visible through body wall of 6-day old and older adults when submerged in 95% alcohol, then water. Adult corpus allatum of mated females hypertrophies. Females completely sterile, sterility autonomous. Eggs laid by homozygotes show meiotic and/or mitotic abnormalities and never develop beyond early cleavage stages. Males 78% fertile. Heterozygotes fertile, but females become sterile with age. Viability generally good, but longevity reduced; homozygotes with selective advantage under starvation; heterozygotes superior under desiccation. Average water content of well fed adults reduced, while percentage of lipids, as a function of dry body weight, is almost double that of wild type. Iodine numbers show greater degree of saturation of mutant lipid extracts than of wild type. RK2.

The mutant flag (<sup>fg</sup>), reported as a new mutant in DIS-34 and located approximately at 2-20+, has been more accurately located by linking it with a dumpy marker. The new cross-over data indicate it to be at 2-22+, and so it has been checked for allelism with the mutant sprad (spd), located at 2-22.3+. The latter was obtained from the spd gt<sup>-</sup>/SM5, a<sup>12</sup> l<sup>v</sup> Cy sp<sup>2</sup> stock at Pasadena, California. Spd is given
the rank of RK5 in Bridges and Brehme because of its poor penetrance; \( \text{fg} \) is fully penetrant with an RK1. None of the \( \text{spd/sdp} \) flies examined from stock bottles has shown wing effects comparable to those described for \( \text{fg} \), and most of them resemble wild type. However, when \( \text{spd} \) \text{gt}-4/Cy females were crossed to \( \text{fg} \) males (or the reciprocal cross made), all of the non-Cy offspring showed a wing effect which ranged from a slight shortening to a shape midway between the phenotypes of the two different mutants. It appears, therefore, that \( \text{fg} \) and \( \text{spd} \) are allelic and that the former should henceforth be referred to as \( \text{spdfg} \) (spade ag).

Report of J. L. Hubby

**Recovery of another rosy allele.** \( \text{Ins}(3\text{RC};3\text{LP}) \) \text{Sb} e\(^8\)/\( \text{ry}^2 \) was shown to have a rosy phenotype and greatly reduced xanthine dehydrogenase activity (Hubby and Forrest, 1960). A double crossover between \( \text{st} \) and \( \text{Sb} \) was recovered from this inversion which proved to have a rosy phenotype when homozygous or when in combination with rosy or \( \text{rosy}^2 \). No crossovers have been recovered among approximately 5000 progeny from females carrying \( \text{rosy}^2 \) and this mutant. This mutant has therefore been designated \( \text{rosy}^3 \).

Homozygous \( \text{ry}^3 \) males show traces of isoxanthopterin and uric acid in their testes, hence \( \text{ry}^3 \) is a "leaky" mutant with respect to the products of xanthine dehydrogenase. Thus far no satisfactory procedure has revealed convincing evidence of xanthine dehydrogenase activity in extracts of this mutant.


d\(^{61}\text{a}; \text{dusky}^{61}\text{a}. \) Ives, 61a24. Like dy. Induced by 1 kr \( \gamma \) radiation in an Oregon-R/ruccua sperm which was deposited on day 5 of an exhaustive mating schedule. Functional allelism to dy established by A. B. Burdick who found normal recombination (with g) and good fertility and fecundity in both sexes. RK1

\( \text{m}^{601}; \text{miniature}^{601}. \) Ives, 60126. Like m. Induced by 1 kr \( \gamma \) radiation in an Oregon-R/ruccua sperm which was deposited on day 6 of an exhaustive mating schedule. Functional allelism to m established by A. B. Burdick who found recombination with g somewhat reduced and fertility and fecundity good in both sexes. RK1

\( \text{sd}^{58d}; \text{scalloped}^{58d}. \) Ives, 58d14. Strongest sd allele, with vg-like wings and weak vg-like effects on halteres and bristles. Induced by 1 kr \( \gamma \) radiation in an Oregon-R sperm which was deposited on day 7 of an exhaustive mating schedule. Functional allelism to sd indicated by strap shaped wing in \( \text{sd/sd}^{58d}. \) Recombination normal in \( \text{y ct}^6 \text{ ras}^2 \) regions but reduced by 80% between \( \text{ras}^2 \) and \( f \), suggesting a small In associated with \( \text{sd}^{58d} \). Not studied cytologically. Genetic tests show no T. Relative frequency of \( \text{sd}^{58d} \) is sometimes low when competing with non-sd flies but a pure line breeds well. Combines readily with vg alleles and vg deficiencies. RK-2.

Report of James F. Kidwell

\( \text{Dfdr}^{60J} \) - Spontaneous recurrence in sixth generation of full sib mating derived from Princeton wild stock. Expression varies from both eyes absent to wild type. Expression may be asymmetrical. Penetrance varies from 75 to 100 per cent. Penetrance increased by selection for reduced eye. About 5 per cent of flies \( \text{Dfdr}^{60J}/+; \text{ey}/+ \) exhibit deformed phenotype.

Report of H. W. Lewis

\( \text{alpha}^{-1}. \) The report of this mutant was erroneously included in DIS-34 under the report of E. B. Lewis.

Report of A. Schalet

\( \text{ma-l}^{12}; \text{maroon-like}^{2}. \) Schalet, 1961. From X-rayed \( \text{y ct}^6\text{f}. \) \( \text{Dp(y}^+\text{sc}-^1\text{y}^1 \) mated to ma-l\(^1\) females. Hemizygous male and homozygous female are viable and fertile. Brownish-red eye color is like \( \text{ma-l}^1 \) and \( \text{ma-l}^{12}. \) ma-l\(^2\) does not complement ma-l\(^1\)
ma-l\(^1\), ma-l\(^1\)bz or ma-l\(^3\) (see below).

ma-l\(^3\); maroon-like\(^3\) Schalet, 1961. From X-rayed y ct\(^6\)f. Dp(y\(^+\)sc\(^{vi}\)) dd mated to ma-l\(^1\) females. Hemizygous males inviable. In a small-scale test the absence of crossovers between v and ma-l\(^3\) indicate possible association with a gross rearrangement. Heterozygotes of ma-l\(^3\) with ma-l\(^1\) or ma-l\(^2\) are mutant in appearance and similar in color to the homozygotes of the latter alleles. A chromosome carrying v and ma-l\(^3\) heterozygous with y v f ma-l\(^1\)bz chromosome also shows a mutant appearance with respect to maroon-like.

Report of A. H. Sturtevant

Correction:

**spaPol**: sparkling-poliert. The mutant poliert of Hadorn (Rickenbacher, DIS 27: 59) is an allele of spa, giving when crossed to spa, heterozygotes with eyes slightly more extreme than spa. spaPol is probably the most useful recessive in the fourth chromosome.

Report of V. Tinderholt

**Cyg**: The Curly gene Tinderholt, 58f 2-6.1±9. The Curly gene is generally inseparable from the inversion, In (2L) Cy, but has been crossed out by increasing the frequency of double crossovers. This was accomplished by using females carrying the complex inversions FM6; SM5 (which includes In (2L) Cy); TM3 Ser, as the three major chromosomes. The dominant character associated with this gene when free of the inversion is identical to the one resulting from its presence within the inversion. The symbol Cyg was chosen because Cy generally is used to designate the gene-inversion combination.

The Curly gene was first localized between heldout (ho, 2-4.0) and echinoid (ed-11.0). Out of 2,489 flies, there were 1,127 Cyg ed and 1,241 wild type non-crossovers. The crossovers consisted of 64 Cyg and 57 ed giving a value of 4.9±.9 crossover units to the left of ed. RK1.

**TM3 Sb Ser**: Third Multiple 3 with Stubble and Serrate See DIS 34:51, Report of E. B. Lewis, and DIS 34:53-54, Report of V. Tinderholt. The third chromosomal multiple rearrangement, TM3 Sb Ser carries the genes bx\(^{34c}\) Sb ri pS sep and eS. The Stubble gene apparently arose from a new mutation and not from a rare double crossover as was suggested in the previous DIS note. The gene bx\(^{34c}\), 0.6 crossover units from Sb still remains in the chromosome. A double crossover would have removed bx\(^{34c}\) as Stubble was inserted.

LINKAGE DATA


The dominant sex-linked miniature wing mutant reported in DIS-34 appears to be identical to m\(^3\). The mutant is allelic to both m and dy, and in its interactions with m and dy cannot be distinguished phenotypically from the interactions involving m\(^3\). No crossovers have been found among over 5,000 progeny which might have shown a crossover between this allele and m\(^3\).
STOCK LISTS

PITTSBURGH, PENNSYLVANIA: UNIVERSITY OF PITTSBURGH

D. persimilis

Wild Stocks

Yosemite National Park, el. 8000' (White Wolf)
  Whitney (4 strains)
  Klamath (4)
  Mendocino (4)

Yosemite, el. 10,000' (Timberline)
  Whitney (4)
  Klamath (4)
  Mendocino (4)

Mather, California: 15 strains incl. Wh, Kl, and St

ROCHESTER, NEW YORK: THE UNIVERSITY OF ROCHESTER

D. busckii

There are more than 300 stocks of D. busckii in the collection of J. Krivshenko. They include X-chromosomal and autosomal mutants (with visible effects), in various combinations, as well as a number of special stocks in which lethals are associated with chromosomal aberrations. Dominant and recessive markers, associated with inversions or other types of chromosomal aberrations, are available for each chromosome. There are also 17 strains from geographically remote populations.

D. persimilis

Wild Stocks: 12 strains from various localities in Western North America.

D. pseudoobscura

Wild strains homozygous for gene arrangements on the third chromosomes, as follows:

<table>
<thead>
<tr>
<th>Standard</th>
<th>Ferron (13)</th>
<th>Chiricahua</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mather (6 strains)</td>
<td>Gunnison (16)</td>
<td>Mather (6)</td>
</tr>
<tr>
<td>Pinion (6)</td>
<td>Leman Cave (13)</td>
<td>Pinion (6)</td>
</tr>
<tr>
<td>Arrowhead</td>
<td>Mono Lake (10)</td>
<td></td>
</tr>
<tr>
<td>Bryce (8)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
114 inbred lines of the above Arrowhead stocks in F3-F28 of sib mating
33 strains from various geographical localities in Western North America
12 strains each from: Helena, Calif.  Lone Pine, Calif.
    Sebastopol, Calif.  Wild Rose, Calif.
    Hopeland, Calif.  Placerville, Calif.

Other Species

D. fuliginea: Rochester, N.Y. (1 strain)
D. funebris: Rochester, N.Y. (1)
D. hydei: 1 strain heterozygous for an inversion on the second chromosome including
sections 2-D1 through 2-G2 (collected at Raleigh, N.C., August 1954). 1 wild
strain from Rochester, N.Y.
D. immigrans: Rochester, N.Y. (1)
D. miranda: Big Basin (1)
D. repleta: Rochester, N.Y. (2)
D. robusta: Rochester, N.Y. (1)
Megacelia scalaris: Princeton, N.J. (1)

FRANCE

Gif sur Yvette (S et O): Centre National de la Recherche Scientifique,
Laboratoire de Génétique Evolutive  (From DIS 27:70)

D. funebris
Wild type from Challuz
Wild type from Chatenay-Malabry
Wild type from St Mandé

D. simulans
Wild type from Dr. Haldane
Mutant types: Net Pm
Wild type from Dr. Sturtevant
se (?)
Wild type from South Africa

ITALY

Pavia: University, Institute of Genetics
(Type Culture Collection of Drosophila Species)  (From DIS 33:113)

D. acanthoptera (1 strain)  D. duncanii (1)  w sn np
D. affinis (1)  D. funebris
D. algonquin (1)  Wild Stocks (3)
D. ambiguus (2)  Mutants: BbY
D. athabasca (1)  b w s; st
D. azeteca (1)  cn
D. bifasciata  co np; st
Wild Stocks (3)
Mutants: co no; st, cu
D. busckii (1)  co np/StubY
D. buzzatii (1)  cu; st
D. cameraria (1)  ev
D. cardini (1)  miniature-vermilion
D. descabibi Burla (1)  np
D. dscabibi Burla (1)  np
D. giberosa (1)  Pch
D. guttifera (1)  sn^2; st
D. helvetica (1)  w
D. hymenoptera (1)  sn^2 w y np
D. immigrans (1)  st 45h
D. kurtzei (1)  Va
D. latifasciaformis (1)  y
D. lativittata (1)  D. lebanonensis (1)
D. littoralis (1)  D. miranda (1)
D. montium (1)  D. narragansett (1)
D. nitens  Wild Stocks (3)
D. obscura (2)  Mutants: or
D. persimilis (1)
Drosophila Species - Stocks

July 1961

<table>
<thead>
<tr>
<th>Species</th>
<th>Stocks</th>
<th>Other Species</th>
</tr>
</thead>
<tbody>
<tr>
<td>D. Phalerata (1)</td>
<td>D. spinofemora (1)</td>
<td>D. alboralis (1 strain)</td>
</tr>
<tr>
<td>D. prosaltans (1)</td>
<td>D. subbadia (1)</td>
<td>D. angularis (1)</td>
</tr>
<tr>
<td>D. pseudoobscura (2)</td>
<td>D. subobscura</td>
<td>D. arizonensis (1)</td>
</tr>
<tr>
<td>D. putrida (1)</td>
<td>Wild Stocks (1)</td>
<td>D. auraria (5)</td>
</tr>
<tr>
<td>D. repleta (1)</td>
<td>Homozygous standard:</td>
<td>D. bifasciata (2)</td>
</tr>
<tr>
<td>D. robusta (1)</td>
<td>Esperöd</td>
<td>D. bizonata (3)</td>
</tr>
<tr>
<td>D. setifemur (1)</td>
<td>Kussnächt</td>
<td>D. busckii (3)</td>
</tr>
<tr>
<td>D. simulans (2)</td>
<td>D. transversa (1)</td>
<td>D. buzzatii (1)</td>
</tr>
</tbody>
</table>

KOREA

Kwangju: National Chunnam University, Laboratory of Genetics

D. virilis

Wild Stocks

Japan (2 strains)
Korea (5 strains)

Inversion

In(X)Spd

Other Species

D. alboralis (1 strain) D. cheda (3) D. lutea (3)
D. angularis (1) D. coracina (3) D. mirim (1)
D. arizonensis (1) D. duncani (1) D. milleri (1)
D. auraria (5) D. hamatofila (1) D. nigromaculata (3)
D. bifasciata (2) D. hayashi (2) D. repleta (1)
D. bizonata (3) D. histrio (2) D. suzuki (2)
D. busckii (3) D. immigrans (3) D. testacea (1)
D. buzzatii (1) D. lacertosa (2) D. unispina

SPAIN

Barcelona: Centro de Genética Animal y Humana del Consejo Superior de Investigaciones Científicas

D. ambigua: several Spanish stocks
D. bifasciata: Pavia (Italy)
D. busckii: Barcelona
D. camararia: Cantonigrós (Spain)
D. funebris: several Spanish stocks
D. immigrans: Barcelona
D. kuntzei: Cantonigrós (Spain)
D. mercatorum mercatorum: Barcelona
D. mercatorum pararepleta: Jijuca (Brazil)
D. phalerata: several Spanish stocks
D. phalerata: Pavia (Italy)
D. repleta: New Haven (Conn.); Berlin
D. simulans: Barcelona
D. subobscura: several Spanish stocks; mutant stocks
D. transversa: Montnegre (Spain)
Parascaptomyza disticha: Barcelona
Irwin H. Herskowitz, Editor

D. = Drosophila; D.m. = Drosophila melanogaster

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The following are the results and ecological notes of flies of the immigrans sp. group caught during February 1961.

Results:

<table>
<thead>
<tr>
<th>Location</th>
<th>rubida (%)</th>
<th>pararubida (%)</th>
<th>setifemur (%)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Port Moresby</td>
<td>479 (60)</td>
<td>169 (21)</td>
<td>155 (19)</td>
<td>801</td>
</tr>
<tr>
<td>Bulolo</td>
<td>245 (45)</td>
<td>199 (36)</td>
<td>105 (19)</td>
<td>549</td>
</tr>
<tr>
<td>Lae</td>
<td>14 (5)</td>
<td>247 (58)</td>
<td>169 (59)</td>
<td>430</td>
</tr>
<tr>
<td>Kaviang</td>
<td>17 (20)</td>
<td>40 (50)</td>
<td>24 (50)</td>
<td>81</td>
</tr>
<tr>
<td>Rabaul</td>
<td>428 (18)</td>
<td>632 (28)</td>
<td>1348 (56)</td>
<td>2408</td>
</tr>
<tr>
<td>Total</td>
<td>1183 (28)</td>
<td>1287 (30)</td>
<td>1799 (42)</td>
<td>4269</td>
</tr>
</tbody>
</table>

It was noted that D. pararubida was the dominant species over fermenting cocoa pods and citrus and that D. setifemur was particularly associated with rotting five corners (Averrhoa carambola).

Barbour, Evelyn and S. Zimmering
Preliminary analysis of a Y chromosome from nature carrying a mutant allele of bobbed.

In routine experiments to introduce Y chromosomes derived from males caught in nature into a y f double-X female, it was found that in the presence of one of these Y chromosomes, symbolized Y+17, the double-X females hatched very late and showed an extreme bobbed effect, but proved to be fertile. Similar results were obtained from sc^4-sc^6/Y+17 males. To test the dosage effect of one vs. two Y+17 chromosomes, y In49 f car/y sc^4 w sc^6/Y+17 females were crossed by wec^5,Y+17 males. Of 194 y sc^4 w sc^6 males recovered, 121 (62%) appeared non-bobbed and 73 (38%) appeared bobbed. It is inferred that the non-bobbed males carried two Y+17 chromosomes and the bobbed males only one. On this interpretation, the bobbed allele in the Y+17 chromosome acts as a typical hypomorph, as described for bb by Stern (1929). Experiments were carried out to determine the effect of this modified Y chromosome on secondary non-disjunction. Females of the constitutions y In49 f car/y^2 sc w^a ec/Y+17 and y In49 f car/y^2 sc w^a ec/Y Oregon R, and having approximately the same autosomal background, were crossed by Oregon R males. The frequencies of XX-Y segregations, calculated from F1 female offspring only, were found to be as follows: 40.2% (2577 F1♀♀) from the former, and 64.9% (1794 F1♀♀) from the latter. The results suggest a possible impaired pairing site in the Y+17 chromosome. No information is as yet available on disjunction of X and Y in males carrying the Y+17 chromosome.

Bateman, Angus J. X-ray induced "crossing-over".

Analysis is continuing of "cross-overs" recovered from matings of b pr vg/+ + + ♀♂ to b pr vg ♀♀♀, over the period 5 to 11 days from irradiation of the ♀♂. It had earlier been supposed that r1 recombinants (b, pr or vg) could be point mutations or deletions as well as true cross-overs, but that r2 recombinants (b pr or pr vg) must represent true cross-overs. The latter assumption is now found to be untrue for two reasons: some r2 recombinants are lethal when homozygous; and in some samples the r2 class is larger than the r1 class. It is concluded that there are at least 4 modes of origin of "cross-overs" during the period under study (which we presume to consist largely of irradiated spermatocytes)

1. Point mutations (r1)
2. Deletions (r1)
3. Illegitimate crossing over (r1 = deletion; r2 = duplication)
4. True crossing over (r1 = r2)

Each illegitimate crossing-over will yield one deletion and one duplication. But the duplication would be expected to be more viable in the zygote than the deletion, so that the observed yield of r2 from this source would be more than that of r1. We have found that on days 5, 6 and 7 r1 exceeds r2 on days 8 and 9 r2 exceeds r1 on days 10 onwards r1 = r2

This is interpreted to mean that the commonest modes of formation of "cross-overs" on days 5, 6 and 7 are point mutations and simple deletions, on days 8 and 9 illegitimate crossing over and on later days true crossing over.

The testes of the Crianlarich strain of Drosophila melanogaster show two differences from other strains: (1) the ends are bulbous to a greater or lesser extent, (2) the colour varies from normal yellow to nearly white. The former condition is here named bulbous testes and apparently has not been reported before in this species.

Stern (1941) demonstrated the importance of the vas in determining the shape of the testes. He showed that the vasa deferentia from species with non-coiling testes fail to induce coiling in testes which normally have coils, and vice-versa, that the vasa deferentia of "coiling-species" will cause normally uncoiled testes to coil. In coiling species, the testes are uncoiled and ball shaped without any attachment up to 30 hours after puparium formation (Stern 1941). The coiling of testes is a differential growth function due to a growth promoting substance. The interaction between testes and vas gives rise finally to the imaginal form of testes. This sort of development of testes in males is common in the "coiling species" of Drosophila. However, the high frequency of bulbous testes ends in Crianlarich strain, and a low frequency of cases of uncoiled testes (mostly cases of failure of one testis to coil, and remaining ball-like) show that the differential growth function, of the growth promoting substance, fails to complete the growth in the first case and leaves the testes uncoiled in the second case.

It has been noted that there are cases of failure of completion of growth in testes in D. subobscura also. In one male belonging to a natural large strain from Aberdeen, the testis of one side was found to be ball-shaped, instead of having the elongated and tubular shape, characteristically found in the species.

The uncoiled testis lobe sends no spermatozoa into the vesicula seminalis of its side, and the latter is smaller in size than the normal one of the other side, and empty. This vesicula seminalis is seen to be translucent, (being empty), in a permanent stained preparation.

It is believed that the fertility of the males must be affected, in case of occurrence of ball-shaped testes which yield no mature spermatozoa and their seminal vesicles remaining empty. In the case of bulbous testes, probably the fertility remains unaffected. It is still a matter of speculation that the Crianlarich strain of Drosophila melanogaster does not have a low fertility because of the character of bulbous testes ends and some though very low frequency of uncoiled testes. The overall fertility of the strain probably remains unaffected in spite of the occurrence of some half-sterile males (i.e. males with one testis uncoiled).

The colour of testes in Crianlarich strain varies from yellow to creamy white, the colour of the eyes of the flies always being red. The previous literature on the colour of testes shows that the colour of eyes and pigmentation of the tunica externa of testes go together, i.e. if the eyes are dark coloured as in wild type, the testes are yellow in colour, and flies with light eye colour have light colour of testes also, ranging from yellow to nearly white. In the Crianlarich strain, it has been found that the colour of testes varies from yellow to creamy white, in spite of the colour of eyes always remaining dark, i.e. red.

The bulbous testes ends character is inherited as a recessive. The crossing of Renfrew stock and Crianlarich gives all normals in F₁, and nearly ½ bulbous testes ends, in F₂.

We are grateful to Dr. F. W. Robertson for very kindly providing us the stocks of flies used for these studies.

References


Table to show the higher incidence of bulbous testes ends in Crianlarich strain compared with Oregon.k.

<table>
<thead>
<tr>
<th>Strain</th>
<th>No. of flies investigated</th>
<th>No. of flies with bulbous testes ends</th>
<th>No. of flies with bulbous testis end on one side only</th>
<th>No. of flies with Uncoiled testis on one side</th>
<th>No. of flies with normal testis in the population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crianlarich</td>
<td>46</td>
<td>25</td>
<td>10</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>F_1 generation of Renfrew X</td>
<td>51</td>
<td>7</td>
<td>2</td>
<td>-</td>
<td>50</td>
</tr>
<tr>
<td>(C x R) flies</td>
<td>25</td>
<td>10</td>
<td>1</td>
<td>-</td>
<td>16</td>
</tr>
<tr>
<td>Or.K. flies</td>
<td>60</td>
<td>6</td>
<td>4</td>
<td>-</td>
<td>50</td>
</tr>
</tbody>
</table>

Brosseau, George E., Jr.
The effect of M(2)S10 on the fertility of some compound XY chromosomes.

On two separate occasions an attempt to establish a stock of the constitution:

\[ y^2 su-w^a w^a b^a / y'^Y; Cy/M(2)S10 X \]

\[ y^S x y^L, In(1)EN In(1)d1-49, y v f car/y^Y; \]

Cy/M(2)S10 failed owing to the infertility of the M(2)S10 males, while their non-Minute brothers were normally fertile. A survey of the effect of M(2)S10 on the fertility of several compound XY's of diverse origin and structure was then undertaken. In these tests, the fertility of males of the constitution X-Y/y+y; M(2)S10/+ was compared to their X-Y/y+y; Cy/+ brothers. These males were crossed individually with 2 yv/yv;bw/bw females from a stock that consistently yields over 95% fertile matings. Twenty males of each genotype were tested. Over 90% of the non-Minute males were fertile in every case. In contrast, all but one of the compounds showed a lowered fertility when M(2)S10 was present. The exception was Y^SXY^L-Y^S which showed 100% fertility. Only 60-65% of the males that were X-Y^L, X-Y^S or Y^SXY^L were fertile. For Y^SXY^L-Y^L and Y^SXY^L, In(1)EN In(1)d1-49 the value was 35-40% and for Y^SXY^L, In(1)EN it was only 15%. In all cases of a lowered number of fertile males, the fertility of the fertile males was drastically reduced, single males often yielding only a very few progeny. No generalization concerning the structure of a compound and the effect of M(2)S10 on its fertility is possible. Nor does the amount of Y chromosome material, as expressed by the number of fertility factors sets present, seem to be important. This latter point also argues against a position effect explanation (M(2)S10 is a strong enhancer of variegation). It is likely that M(2)S10 interacts with some, as yet undefined, property of the sensitive compounds. These results suggest that this factor may not be the same in each of the compounds.

Burdette, W. J. Effect of penicillin on mutation rate following irradiation in different concentrations of oxygen.

Previous work indicated diminution in lethal mutation rate in Drosophila melanogaster following irradiation when antibiotics were administered. These studies have been extended to an inquiry into the effect of penicillin on the alteration in pattern of induced mutations in an atmosphere of oxygen. Males of the sc sr es ro ca; tu 36a strain were irradiated (3000 r) and lethals detected on the X chromosome by appropriate crosses with the sc^S1 B InS w^a sc^B stock. The first group was not treated, the second was irradiated, the third was raised on medium containing 20,000 units of penicillin per ml. of medium and irradiated, the fourth group was maintained one minute before and ten minutes during irradiation in an atmosphere of 100 per cent oxygen, and the fifth group was similar to the fourth except it was raised on medium containing penicillin in the same concentration as group three. After irradiation at 20 hours of age, males were mated successively to different virgin females at intervals of two days. Lethals in the progeny of these respective matings representing successive stages of spermatogenesis during irradiation are indicated by the letters A - G and their number and distribution are recorded in table 1 and figure 1.

Striking reduction in mutation rate was found when penicillin was added to the medium both in the groups irradiated in air and in oxygen. In the latter, the effect is apparent in stages B and C, but in the former it is evident throughout spermato-
<table>
<thead>
<tr>
<th>Stage of Spermato-genesis</th>
<th>Control</th>
<th>3000 r Penicillin</th>
<th>3000 r O₂ Penicillin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Total</td>
<td>Lethals</td>
<td>Per Cent</td>
</tr>
<tr>
<td>A</td>
<td>795</td>
<td>1</td>
<td>0.12</td>
</tr>
<tr>
<td>B</td>
<td>654</td>
<td>1</td>
<td>0.15</td>
</tr>
<tr>
<td>C</td>
<td>649</td>
<td>2</td>
<td>0.30</td>
</tr>
<tr>
<td>D</td>
<td>647</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>E</td>
<td>551</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>F</td>
<td>512</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>G</td>
<td>440</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total</td>
<td>4248</td>
<td>4</td>
<td>0.09</td>
</tr>
</tbody>
</table>
Figure 1: Frequency of Lethal Mutations at Different Stages of Spermatogenesis

The increase in frequency of lethal mutations when the content of oxygen prior and during irradiation is raised from that in the ambient atmosphere to 100 per cent is greatest in stage C, but this is at the expense of such a change in other stages. When the total frequency of lethal mutations at all stages for the groups irradiated in air is compared to the frequency for those irradiated in 100 per cent oxygen, no increase is found in the latter group. Apparently an increase in percentage of oxygen has resulted in a redistribution of the mutations without increasing total number, whereas penicillin has reduced the number when appropriate groups are compared. The reduction with antibiotic treatment is rather uniform throughout spermatogenesis when irradiation is carried out in air.

Burdick, A. B. 1(2)55i at Erie, Pennsylvania.

1(2)55i was found in our so-called "Erie" wild stock (W-I) about a year after the stock had been brought into the laboratory. Various reports by T. Mukai, S. M. Schnick, and myself have dealt with heterozygote viability of this lethal. We have found that this heterozygote is super-viable and have not been able to implicate anything but the single locus itself as a cause of the apparent heterosis. All through these studies we have wondered whether the lethal came from the wild population, or was a mutation that had occurred in the laboratory after the stock had been brought in. Certain considerations have led us to think that even if the lethal had come from the wild population it would not necessarily still be in the population -- six years later. I went back to the Erie area this fall and collected flies again. Our tests show that 1(2)55i is still in the Erie wild population.

Carlson, E. A. and R. Sederoff. A selective scheme for recovering pseudoallelic recombinants, The principle first suggested by Whittinghill (Science 111:377) for the recovery of selected recombinant types has been
"conversion" phenomena, and reverse mutations. Successfully applied in the construction of a lethal selector system for complex loci by Schalet and Chovnick (DIS 34:104).

However, this has the inherent disadvantage that four lethals must be used, with each pair of lethals on either side of the allelic region very close to one another. This makes such a system difficult to construct without considerable effort. Furthermore, two sets of such "tester" stocks must be employed so that the wild type crossovers between the alleles can survive in the "sifter" stock used to kill the undesired chromosomes. Finally, this system is valid almost exclusively for single wild type crossovers and it cannot be used to detect double crossovers, reverse mutations, or "conversion" phenomena in the complex locus.

It is possible, however, to use a modification of this principle with certain loci and obtain any wild type event picked up by the "sifter." This proposed system is also simpler to construct and involves only one initial stock for the allelic tests.

In the system illustrated here, the dumpy region is used for the allelic series in question. The stocks are S dpX Sp / InCyL,Cy and Cy dpY / InCyL. The "tester" heterozygotes are thus: S dpX Sp / Cy dpY q q. In this example the Cy is a dominant visible with recessive lethal manifestation which has been obtained free of its former location near the left end breakpoint of the InCyL,Cy chromosome (Tinderholt, unpublished). S and Sp are also dominant visibles with recessive lethal effects. The alleles dpX and dpY represent any two members of the dp series used for analysis. The "sifter" stock has the composition Df(ed+ dp+)2MB / S2 InCyL,Cy. Hence all non-recombinant chromosomes from the "tester" heterozygote are killed by the S2 InCyL,Cy chromosomes. The distance between S and Cy is about six map units. Hence half of these, carrying neither S nor Cy may be passed on to the "sifter" stock, with a total of 1.5% surviving as a S dpX Sp / S2 InCyL,Cy combination. The other chromosome in the "sifter" stock, bearing the dumpy deficiency renders the allelic combinations semi-lethal, with a survival to the adult stage of about 5% at 27°C. The total surviving progeny would thus not exceed 7%. Any change occurring in the dumpy region resulting in a non-dumpy phenotype, would complement the deficient area and hence its chances of survival would be nearly unity. Phenotypically there are only two classes of wing mutation in this system -- those with oblique, curly wings and those with very reduced truncated wings. The exceptions would appear as non-dumpy flies. The rate of recombination can be determined by the same procedures outlined by Schalet and Chovnick.

If males are used in this tester stock, and mature sperm are irradiated, then reverse mutations can be selected appearing with an apparent frequency 20 times greater than would be obtained without selective techniques. The tester stock in these reversion studies could use the same scheme as outlined before, but the allele tested would be homozygous. By further increasing the temperature to 29°C, the viability of the heterozygotes is diminished to less than 1% and the selective technique can thus determine reversion frequencies of 1 X 10^-7 with the same amount of labor as is presently used for frequencies of 1 X 10^-5.

This system should work for any allelic series which expresses a decrease in viability in compound with a deficiency for its entire region. Other types of selective techniques can be devised using other viability characteristics (such as prune-killer as a "sifter" for pseudoallelism or reverse mutation among prune alleles). This study is supported by Grant G 14222 from the National Science Foundation.

Chandley, A.G. Mutations induced in presumed spermatocytes successfully applied in the construction of a lethal selector system for complex loci by Schalet and Chovnick (DIS 34:104).

Mating of F1 males of Drosophila melanogaster following 1000r X-rays has shown maximum sensitivity to the induction of sex-linked lethals and translocations on the 5th day from irradiation followed by a period of low fertility on the 8th. By analogy with the mouse, the 8th day could be expected to represent irradiated spermatogonia and the 5th day irradiated spermatids.

The intervening days 6 and 7 could therefore be expected to represent spermatocytes. These have been investigated for the incidence of dominant lethals, sex-linked recessive lethals, translocations and deleted X's, with the following results:
Sex-linked lethals and translocations show a rough parallelism with the peak on days 5 and 6. In the case of translocations the level drops sharply through day 7 to the lowest level on day 8.

For sex-linked lethals the high level on days 5 and 6 is maintained through day 7 and then drops sharply to day 8.

Deleted X's (as in previous studies) show a continuous and steep rise from day 5 to a peak on day 8.

Dominant lethals maintain a high level over days 6, 7 and 8. Previously it had been thought that the large percentage of unhatched eggs on day 8 might include some which were unfertilized. However, recent studies on eggs collected within 1/2 - 1 hr. of laying from matings with day 8 irradiated males have shown almost 100% fertilization — indicated by the presence of polar bodies and early mitotic cleavages.

Amongst the eggs examined were some showing micronuclei, chromosome fragments and stickiness, effects which would lead to breakdown of the mitotic cleavages at an early age and so cause death of the egg.

In order to study directly the effect of X-rays upon spermatocytes, cytological examination was made on testes of irradiated late pre-pupae when only spermatocytes and spermatagonia were present. Twenty-four hours after doses of 1000r and 2000r many of the dividing spermatocytes showed chromosome aberrations including sticky anaphases, chromosome breaks and fragments.

Chandley, Ann C. Timing spermatogenesis in Drosophila melanogaster with tritiated thymidine.

In view of the spate of research on mutation in immature male germ cells it was felt that there was a great need for direct timing of spermatogenesis using tritiated thymidine to label the germ cells. We have injected the abdomen of newly emerged ♀♂ with 0.08 c.mm. of tritiated thymidine (activity 25μC/ml).

On each successive day from injection, the testes of mated and unmated ♀♂ were fixed in 3 : 1 alcohol-acetic, cut at 8 μ, and stained with Feulgen. The exposure time for autoradiographs was 3 weeks. Labelling of young spermatocytes was detectable on the second day but in the later auxocyte stage the degree of Feulgen staining is so slight and the dispersion of the label so great that it was difficult to recognize label in these cells. We have since found that a much clearer picture of this stage can be obtained using eosin as a counterstain. By day 5, however, groups of very young labelled spermatids were visible, all the labelled nuclei being apparently in a single cyst. With succeeding condensation of the nuclei and agglomeration into sperm bundles, the label became increasingly obvious. To date we have not observed testes more than 8 days from injection. By this time, heavily labelled sperm bundles are present in the testis close to the exit into the seminal vesicle. Assuming that the last stage to incorporate tritiated thymidine is the early spermatocyte the complete life span of a spermatocyte would appear to be 4 days. No differences were apparent between mated and unmated ♀♂ in the rate of spermatogenesis. This study is now being repeated using a heavier dose of tritium, eosin as a counterstain, making autoradiographs over a longer period than 8 days, and also looking for labelled sperm in ejaculates.

Divelbiss, J. E. A sterility factor affecting both males and females in Drosophila melanogaster.

An attempt to make a stock of the constitution In(2L)t, Roi+In(2R)Cy, by45a sp2 or45a/Ins(2L+2R)Cy, Cy by45a sp2 or45a (abbreviated Roi and Cy respectively) failed due to sterility. Outcrosses of Roi/Cy males and females to Oregon-R showed the sterility to be present in both sexes. Since mutant females produced no eggs...
and only very few eggs were produced by Oregon-R females mated to mutant males, the observed results are most likely due to sterility rather than zygote lethality. Roi probably arose as the consequence of crossing-over between In(2L)t and In(2L)Cy in In(2L)t, Roi/Ins(2L+2R)Cy, bw^45a sp^2 or^45a, hence it would carry the right hand portion of In(2L)Cy. Ives, DIS-25:70, reported the presence of a lethal near each end of In(2L)Cy in Ins(2L+2R)Cy, Cy bw^45a sp^2 or^45a. Roi/Cy would be homozygous for the right hand portion of In(2L)Cy and, hence, also for the right hand lethal. However, Roi would carry a duplication for salivary bands 22D2-3; the duplicated piece originating from In(2L)t and carrying 1+. This would suggest that the right hand lethal is associated with bands 22D2-3. Roi must also be 1+ for the left hand lethal. Since the right arms of the two homologues are derived from the same source, they are probably genetically identical. The sterility could be explained by the presence of an undetected mutant in In(2R)Cy which arose previous to the time that Roi was derived, and which would become homozygous in the Roi/Cy heterozygote.

Doane, W. W. Persistence of fs(2)adp in the Kaduna population after four years.

in the Kaduna wild stock, maintained at the Institute of Animal Genetics in Edinburgh, from which it was originally screened in 1956 (Doane, 1960, J. Exp. Zool., 145: 23-42). In the summer of 1960, 18 sample vials of this stock were received from Dr. A. Robertson of that laboratory and all emerging males were mated individually to Cy/fs(2)adp females. Five non-Cy female progeny from each of these matings were tested for fertility and, where sterility occurred, the lines were perpetuated by crossing their Cy brothers to H-40 females (stock #114 in DIS-34, with dominant markers and cross-over suppressors on chromosomes I, II and III). There followed a breeding program for these lines by means of which the individual Kaduna chromosomes were isolated in the H-40 background so that their effects on the adult fat body and on fertility might be tested. Through this procedure, the factor fs(2)adp has been screened from the descendents of at least 13 of the original 18 samples, suggesting that it persists in the Kaduna wild stock at a fairly high frequency. In addition, other factors affecting the ovaries and fat body have been screened out. Certain second chromosome lethals picked up this way are able, in the heterozygous condition, to mask the effect of fs(2)adp on the fat body. A second chromosome factor, apparently allelic with fs(2)adp but which causes hypertrophy of the fat body without accompanying sterility, is very prevalent in the Kaduna wild stock. This latter mutant is especially well-suited for histochemical studies on fatty tissues.

(Dorn, G. L. and A. B. Burdick. Recombination between Df(1)259-4 and various mutants of the miniature-dusky complex in D. melanogaster.)

Df(1)259-4 produces a hemizygous effect with the mutants in the miniature but not the dusky cistron. This would seem to indicate that the deficiency extends through the miniature cistron but stops short of the dusky cistron.

Transheterozygotes of Df(1)259-4 with each of three miniature mutants (m, m^59, and m^0) and two dusky mutants (dy and dy^58) were formed. These transheterozygotes have been examined for recombination. All five combinations have been found to yield recombinants.

Below is constructed a genetic map which indicates the relative distances between Df(1)259-4 and each of the five mutants.
If it is assumed that Df(1)259-4 lies to the extreme left, then the gene order agrees with that which we have previously determined (see DIS-33, 1959). Vermillion and garnet markers were also employed in these recombinational experiments. At present, it seems that recombination between Df(1)259-4 and any one of the miniature mutants is also associated with a high negative interference for the outside markers. This does not seem to be the case with the dusky mutants.

Young males from the Bucaramanga, Columbia strain of D. paulistorum were exposed to approximately 4000 r-units of X-rays, and then crossed to untreated virgin females of the same strain. The F1, F2, and F3 progenies were examined for autosomal and sex-linked, dominant and recessive mutations. Although this work is still in progress, and more males will be irradiated, the initial results permit the reporting, for the first time, of mutant genes in this important subspecies:

**Delta-Autosomal dominant, lethal when homozygous.** This mutation occurred frequently in X-rayed cultures. Wing veins thickened at the margins and the cross-veins. Eyes very rough. This mutation already exists in the Amazonian and the Andean-South Brazilian subspecies of D. paulistorum.

**Minute-Autosomal dominant, lethal when homozygous.** Bristles reduced in size, especially the scutellar bristles. The developmental period is lengthened, and the viability of both sexes is poor. A Minute exists in the Centro-American subspecies.

**Star-Autosomal dominant, lethal when homozygous.** This mutation occurred several times in X-rayed cultures. Eyes roughened because of irregularly arranged facets. Star has also been induced in the Amazonian, Andean-South Brazilian, and Centro-American subspecies; thus, it has been acquired in every D. paulistorum subspecies irradiated.

**Veinless-Sex-linked recessive.** This mutation occurred frequently in X-rayed cultures. Many of the wing veins are absent or shortened or interrupted. The wings themselves are warped distally where there are virtually no veins. This mutation was previously induced in the Centro-American subspecies.

The mutations listed above, and others which may be induced and firmly established in stocks, will be employed as genetic markers in the study of reproductive isolating mechanisms (hybrid male sterility and sexual isolation). Because Drosophila paulistorum is now known to represent, at our time level, a number of forms in a state of transition between race and full species, the response of its "bridging" subspecies to a metagenic agent is a necessary prerequisite for further genetic analysis.

(This investigation was supported by a postdoctoral fellowship, GF-9033, from the Division of General Medical Sciences, U.S. Public Health Service.)

A fairly high proportion of the chromosome breaks produced by alpha particles in Tradescantia do not rejoin, and by an indirect method, it can be shown that the same is true of the chromosomes of maize endosperm, Fabergé, 1959. For this reason, a preliminary trial was made on treating Drosophila with alpha particles. The penetration of unaccelerated alpha particles is much too low to permit the use of an external source, and the treatments were made by exposing flies to an atmosphere containing Radon, in our case 6.1 microcurie per ml of air. The Radon is believed to equilibrate with the tissues of the fly very rapidly, and since, moreover, the time to gain Radon activity is about the same as the time to lose activity, quite short exposures are practical. The exact distribution, on a microscopic scale, of the ionization does in the tissues of the fly is difficult to assess, since Radon is, in round figures, about 50 times more soluble in lipids than in water. A discussion of these problems will be found in Gray and Read (1942). About 5% of the ionization is also attributable to 8-particles, and a negligible amount to gamma radiation.

A solution of Radium Bromide in a closed vessel was allowed to equilibrate with its Radon for several weeks. The Radon was then swept over into a one liter spherical flask which has previously been evacuated by admitting air into it which was bubbled through the Ra Br2 solution. The spherical flask now contained air at atmospheric pressure, mixed with 0.1 microcurie of Radon per ml. This spherical flask had a small finger-shaped reentrant consisting of an outer sleeve and an inner cylinder,
ground-fitted to each other. The inner cylinder could be put into communication with the atmosphere in the flask by rotating it in the outer sleeve, and aligning several large holes bored through the walls of both cylinder and sleeve. Flies were placed in a lusteroid plastic centrifuge tube, 12.5 mm in diameter, 40 mm long, in which numerous holes had been punched; the stopper of the tube had a cavity filled with mashed banana. To expose the flies this plastic cage was placed inside the re-entrant cylinder, which was then closed to the outside atmosphere, and rotated in its sleeve to align the holes. The Radon atmosphere then diffused among the flies. Since the volume of the re-entrant cylinder was about 10 ml, each exposure resulted in a dilution of the remaining Radon by about 1%. Five exposures of 3.3, 10, 30, 90 and 270 minutes were made, at 25°C, treating about 200 wild type Oregon R males at each exposure. After exposure, the flies were left in fresh food vials in a chemical hood for 30 hours. After this time very little radioactivity remained, and they could be safely handled without special precautions. The males were mated between 48 and 50 hours after exposure to y v f X X q q, using 10 df and 10 q q per bottle. Four transfers were made to fresh bottles every 2 days.

The first dose of 3.3 minutes was discarded as having had too little exposure. From the other four doses, a total of 40 duplications were found, among 22922 q q examined. The relative frequencies of different classes of duplications are about the same as have been observed from X-ray treatment, in so far as can be judged from so small a sample.

<table>
<thead>
<tr>
<th>X Duplications covering the markers</th>
<th>Radon treatment (all doses)</th>
<th>X-ray Treatment (22922 q q)</th>
<th>(1000 r, + 2000 r, + 4000 r) (58081 q q) (Bishop 1941)</th>
</tr>
</thead>
<tbody>
<tr>
<td>y</td>
<td>31</td>
<td>298</td>
<td></td>
</tr>
<tr>
<td>v</td>
<td>2</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>f</td>
<td>2</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>y v</td>
<td>3</td>
<td>28</td>
<td></td>
</tr>
<tr>
<td>y f</td>
<td>2</td>
<td>48</td>
<td></td>
</tr>
<tr>
<td>v f</td>
<td>0</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>y v f</td>
<td>0</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>

Thus there is no suggestion from this limited information that alpha particles produce duplications of a different nature in Drosophila.

A very small estimate of dominant lethals was also made on a sample of the treated males. The dosage effect curve for dominant lethals from X-rays is of a complex character, and our very small counts do not permit the establishment of the equivalent function for alpha particles. It is thus difficult to make quantitative comparisons of alpha particle and X-ray doses. However, from the dominant lethal counts, as well as the deficiencies, we would hazard the estimate that 1 microcurie per ml of atmosphere for 1 minute is roughly equivalent to 2 r of X-rays, in the region of 1000 r. This estimate may easily be wrong by a factor of 2. Despite the fact that F1 males were not carefully examined for mutations, several X-linked visibles and autosomal dominants were found. A large fraction were Minutes or Rough-eye mutants. Only 11 of more than 45 mutants were saved for mating. Among the X-linked visibles were y, spl, ec, p n-like, I z-like, sn. Among the autosomal dominants were a furrowed-thorax, a Delta, and several good Minutes.

The method for exposing flies and the apparatus was devised by Dr. D. G. Ott, who, with Dr. W. H. Langham, made preliminary estimates of a reasonable dosage range. We are very much indebted to them for their assistance in carrying out the treatment at the Biomedical laboratory, Los Alamos Scientific Laboratory.

Bishop, Maydelle, 1941 The recovery of a simple and multiple breaks of the X-chromosome of Drosophila melanogaster.
Thesis, M. A., University of Texas.
Acid hydrolysis of the sex peptide yields ten ninhydrin-positive products. These have been identified by paper chromatography of the free products themselves and of their dinitrophenol derivatives, prepared by reaction with 1-fluoro-2,4-dinitrobenzene according to the method of Levy (1954). Eight are conventional amino acids: aspartic acid, glutamic acid, serine, glycine, α-alanine, leucine, valine, and methionine. The ninth is ethanolamine. The tenth remains unidentified.

Since ethanolamine lacks a carboxyl group, it must occupy what would otherwise be the C-terminal position of the peptide chain, or constitute a side branch through peptide linkage with one of the dicarboxylic amino acids or phosphate linkage with the serine. Initial attempts to identify the N-terminal residue by preparation of the dinitrophenol derivative of the intact peptide according to the method of Sanger and Thompson (1953), suggest that this position in the peptide is occupied by the unidentified residue. Molar ratios have not yet been determined.

The sex peptide is not present in detectable amounts in male third instar larvae, nor in pupae, nor in males during the first two hours after emergence. In male third instar larvae and pupae, but not in females, there is present instead a substance tentatively identified as phosphoethanolamine. This disappears in newly emerged males just prior to the appearance of the sex peptide.

(Supported by grant C-2440, National Institutes of Health, U. S. Public Health Service, and by a grant from the Rackham Foundation. *National Science Foundation Undergraduate Research Participant in the Department of Agricultural Chemistry and the Honors College of Michigan State University.)

Frost, J. N. Double fertilization mosaics. In experiments involving triploid females and in which approximately 109,000 offspring were examined eight double fertilization mosaics occurred. In all these experiments the parental origins of the sex chromosomes could be determined. Four of the mosaics were diploid female-intersexes, one was a diploid male-intersex, one was a diploid male-triploid female, one was an intersex-triploid female, and the last was intersexual in both portions of the mosaic. In four of the mosaics the two original egg nuclei had carried complementary chromosome sets, in another the two egg nuclei had carried identical chromosome sets. In three of the mosaics the chromosome sets of the two egg nuclei had been neither complementary nor identical, a fact suggesting that at least some, and perhaps most of the double nuclei in the eggs had arisen from independent meiotic divisions of the two nuclei in a binucleate oogonium.

The distribution and proportions of the mosaic parts were quite variable, only two mosaics being bilateral. Each part of the sex mosaics showed complete autonomy in development.

Frost, J. N. Two mosaics of unusual origin. A diploid female-intersex mosaic occurred in the following cross: y w 3N (free X) females by y:CY/Gla;D/Sb males. The diploid female portion was yellow, Curly, Glazed, Dichaete, and Stubble and thus (with the possible exception of one X chromosome) obtained all its chromosomes from the male. The intersex portion was yellow, Glazed, and Stubble and arose from a normal zygotic nucleus while the diploid female portion apparently originated from the independent development of a diploid sperm nucleus. The latter could have been produced by a tetraploid spermatogonium.

Another unusual mosaic occurred in a cross of y w (attached-X) 3N females by y,sc8-Y;L,sp/L,sp;Sb,e/eS males. The entire fly was a diploid female and both the left and right sides were yellow, Lobe, Stubble, and ebony, indicating that both of the third chromosomes on each side had come from the male. In addition the left side of the fly was speck and the left eye was completely absent indicating that the left side had received both of its second chromosomes as well as both of its third chromosomes from the male. A satisfactory explanation for this mosaic has not yet been devised.
Out of 547 transmissible X-ray-induced "yellow" mutants in scute-8 chromosomes the frequencies of different combinations of affected loci in order of decreasing frequency were as follows: (using the same symbolism as before, Frye, 1960, DIS 34) - - + + (398), - - - + (76), + - + + (45), + - + + (25), + - - + (5). No "yellows" were recovered of the other 3 possible classes (+ - - +, - + + +, or - - + -). This obviously means that ac is closer to y than lJl and that ac is closer to lJl than to bb. None of the foregoing tabulations yet allows for the decision as to whether ac is to the left or to the right of y. However, other evidence suggests that ac may not be to the right of y since genetic analysis of 10 cases of dark yellows, included in the above tabulation, showed that 4 were not deficient or affected at the loci of lJl, ac or bb ( + ± ± + where ± represents the dark yellow and the order is assumed to be lJl y ac bb), 2 were deficient or affected at the locus of ac, but not lJl or bb ( + ± - + ), and 4 were deficient or affected at the loci of lJl, ac, but not bb ( - ± - + ). This can be seen by comparing the two possible orders (ac to the left and ac to the right of y) of the 4 loci concerned with the 3 recoverable classes of dark yellows.

<table>
<thead>
<tr>
<th>lJl</th>
<th>y</th>
<th>ac</th>
<th>bb</th>
</tr>
</thead>
<tbody>
<tr>
<td>+</td>
<td>±</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>+</td>
<td>±</td>
<td>-</td>
<td>±</td>
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<tr>
<td>±</td>
<td>±</td>
<td>±</td>
<td>±</td>
</tr>
</tbody>
</table>

The latter order (ac to the left of y) is the only one that allows for one of the breaks (in this case, the right one) to fall in a common region. One must also allow for the difference in the size of the regions — on the basis of breakage the third region is about 6X the first region (Frye, unpublished) regardless of the order of the loci. Another advantage of the suggested order (that of the right hand column) is that it allows for a plus allele of bobbed to border the y locus since those cases of dark yellows that have a normal allele bordering on either the left or the right side of the yellow locus can be explained by position effect, sub-gene deficiency of "point" mutation, whereas those cases where the dark yellows occur between 2 affected loci (as in the last row on the former order) are less easily explained (except as skipping effects on intermediate loci in cases of minute inversions).

Traditional evidence that places ac to the right of y is based chiefly on the mutant ac2, found by Dubinin to affect both achaete and scute strongly, whereas here are 4 cases in which lJl and ac were strongly affected but y (which on Dubinin's view would be between them) was only slightly affected.

(Work supported by a grant to H. J. Muller and associates from the U. S. Atomic Energy Commission (Contract AT (11-1)-195).)

Frye, Sara H. Frequency of "transmissible mutation" at the w and f locus in the scute-8 chromosome in relation to X-ray dose in Drosophila.

Frye, Sara H. Evidence that achaete may not be to the right of yellow.

Males of composition sc8 B (0-24 hrs. old) were given 500r, 1000r, 3000r or 4000r and these and simultaneous controls were mated to y w In49 f virgin-females (48-72 hrs. old). The offspring recovered from sperm ejaculated from 1 to 4 days after irradiation. The number of female and male parents introduced per bottle, representing different X-ray doses, were respectively: 500r - 6 prs., 1000r - 8 prs., 3000r - 14 prs., 4000r - 20 prs. Controls were run with 2, 3, 4, 5, or 6 prs. in order to test for crowding. Usually 4 prs. were used. The frequencies of exceptional phenotypes that were transmissible after progeny testing among the F1 Bar females were for the control, 500r, 1000r, 3000r, and 4000r treatments, respectively: "white" 0.0002% (1/412,459), 0.0050% (14/277,667), 0.0060% (11/180,942), 0.0173% (8/46,067), 0.0217% (9/41,310); "forked" 0.0% (0/263,694), 0.0028% (8/277,667), 0.0033% (6/180,942), 0.0195% (9/46,067), 0.0121% (5/41,310).

Since the number of transmissible mutants per each X-ray dose is small and a genetic analysis was not conducted to determine their qualitative composition no conclusions are drawn (even though most of the w and f mutants were male-viable it does not follow that these are gene mutations since presumptive point mutations,
Many "yellow" mutants were induced by exposing young sc0's carrying the scute-8 chromosomes to various X-ray doses, mating them to y w In49 f virgin-females, and recovering the yellow mutants among the F1 Bar ff's. After an extensive genetic analysis (Frye, 1958) to determine their qualitative composition each yellow mutant was kept for several years in an unbalanced stock (y- sc0 B/y w In49 f ff x y w In49 f d') which meant that frequent selection was necessary in order to maintain the y- sc0 B chromosome and to prevent the stock from becoming homozygous for y s In49 f ffi's. However, the last year before these yellow mutant stocks (each stock was kept in a group of 4 vials) were discarded no selection was performed. At the time of discarding (total no. of yellow mutant stocks remaining was roughly 150) I randomly sampled 52 of these stocks to see what qualitative types had persisted and which had not in spite of the absence of selection. All one had to do was to etherize and check for y B ffi's (where they were present no counts were taken to compare the no. of y B ffi's with the no. of y w In49 f ffi's). Out of 30 stocks, selected randomly, the following qualitative types had persisted (using the same symbolism as before, Frye, 1960, DIS 34) - - - + (9), + - - + (5), - - - (4), - - + + (1) (3), - - - 1 (2). Out of 22 stocks the following had not persisted - - - + (3), + - - + (3), - - - - (6), - - - + 1 (2), - - - 1 (5), + - - + 1 (2), IV + - - - (1).

It can be seen that several of the same qualitative types are common to those that did and those that did not persist, and that there is no simple correlation between sheer number of loci absent or affected and the ability to persist, therefore persistence must be a very complex phenomenon. (This is not to be taken as meaning that persistence is independent of qualitative composition of these mutants). It would be of interest to see if one could correlate the no. of generations that is required for different genetically analyzed mutants to become eliminated in a population with their qualitative structure.

Frye, Sara H. Persistence of qualitatively diverse "yellow" mutants in scute-8 chromosomes in the absence of selection for one year.

Frye, Sara H. Spontaneous "yellows" as gross rearrangements in Drosophila. Frye (1958) reported 4 yellows, recovered in the female, that arose spontaneously and singly in separate control series in crosses of scute-8 B males to y w In49 f virgin-females, to be attached-X's with one break having occurred in the paternal-X and the other in the maternal-X. Their resemblance to Sidky-like rearrangements (X-ray induced break in one chromosome, spontaneous break in another non-homologous chromosome) is only superficial since in my cases both breaks are spontaneous. Genetic analysis showed that all 4 were deficient or affected at the loci of 11J1, y, and ac in the paternal scute-8 chromosome.

In order to see if these 4 yellows acted as if they were structurally the same (as implied by the genetic analysis) 5 virgin-females of each of the 4 yellow stocks were crossed with 5 y w In49 f males in half-pint bottles and a count of the sex ratio was made to see if the proportion of F1 males to females varied among the 4 yellow stocks; the idea being that if the break in the paternal-X had resulted in more of the X-chromosome being lost or affected in some cases than in others this differential viability would shift the sex ratio in favor of the males. The 4 yellow stocks were designated y0001, y0002, y0003 and y0004, and the results (giving the count of the males first) were for each of the above, respectively: 1652 - 730 aggx. 2:1; 838 - 684 appx. 1:1; 993 - 727 appx. 1:1; 1146 - 399 appx. 3:1. Thus y0001 and y0004 are of lower viability than y0002 and y0003 and perhaps involve a greater loss of distal X-chromosome material (especially the b+ locus which could not be tested for directly since the attached-X "yellows" carried a Y-chromosome).

The most unexpected fact is that no yellows with attached-X's occurred in the treated series even though all 607 X-ray induced yellows (in scute-8 chromosomes) were progeny tested and analysed in exactly the same way as the yellows of spontaneous origin. The total number of F1 females in the treated series here was 583,248 and that in the above-mentioned controls was 412,439.

Inversions, or extremely small deletions, could survive in the male) concerning their relation to X-ray dose but only their frequencies are reported.

(Work supported by a grant to H. J. Muller and associates from the U. S. Atomic Energy Commission (Contract AT (11-1)-195).)
Also, there is no reason to conclude that "yellows" occurring spontaneously in the scute-8 chromosome are exclusively or even highly likely to always be exchanges with \( \gamma^S \), but may be due to breaks the results of which may be classifiable as minute chromosome changes, crossovers, or gross rearrangements. (None of the above 4 involved exchanges with \( \gamma^S \), and out of a total of 10 "yellows" arising spontaneously in the scute-8 chromosome of males two were found to involve an exchange with the \( \gamma^S \). Tests for the exchange with \( \gamma^L \) were not made.)

Other spontaneous gross rearrangements involving the tip of the X-chromosome are known (Burtart, 1930 - Blond, Müller, 1945 - "double-X") and the tip of the autosomes (Bridges, 1919 - Pale). Chromosome tips may be more likely to enter into gross rearrangements spontaneously (and possibly when X-rayed) than is ordinarily thought.

Fuscaldo, Kathryn E., and Allen S. Fox. Immunogenetic studies of white-variegated position effects.

derivative, \( \text{In}(1)w^{34w} \), in which the rearrangement is unaltered but a change has occurred in the white pseudoallelic segment (Schultz, 1943, D.I.S. 17:64); the translocation, \( T(1:4)wm^5 \); the mutants \( w, w^e, \) and \( w^a \); the double mutant, \( bw \) \( cn \); and the isogenic wild stock, Oregon-R-I.

In all cases an alteration of the relationship of heterochromatin to the white pseudoallelic segment resulted in a change in the immunochemical properties of an antigen, designated \( H(w)-1 \). The protein \( H(w)-1 \) exhibits a higher antibody combining power in the inversion and translocation stocks than in the wild stock. The difference most probably is associated with a difference in the number of combining sites on the antigen molecule, along with a small difference in the configuration of the antigenic site. The effect is reminiscent of the effect of the Y chromosome on the antigen \( Y-L \) (Fox, 1959, J. Nat. Cancer Inst. 23:1297).

The properties of \( H(w)-1 \) extracted from the mutants \( w, w^e, \) and \( bw \) \( cn \) are the same as that extracted from the wild stock. \( H(w)-1 \) extracted from \( w^a \), on the other hand, behaves like that extracted from the inversion and translocation stocks. The mutants \( w \) and \( w^e \) occupy a locus to the right of that occupied by \( w^a \) (Lewis, Green). It thus appears that some, but not all, alterations of the white pseudoallelic segment affect the structure of \( H(w)-1 \). Furthermore, the effect is not directly associated with eye pigmentation (vide \( bw \) \( cn \)).

The results may be rationalized by the hypothesis that the euchromatic white pseudoallelic segment determines the primary structure (amino acid sequence) of the protein \( H(w)-1 \), but that the tertiary structure of the protein depends on the relationship of this euchromatic segment to heterochromatin. The participation of heterochromatin in the determination of tertiary structure has been postulated previously; in connection with the effects of the Y chromosome on the protein \( Y-L \), and the respective roles of euchromatin and heterochromatin in protein synthesis have also been discussed (Fox, 1959, Science 150:1417).

(Supported by grant C-2440 from the National Institutes of Health, U. S. Public Health Service.)

Ghini, Clara. Effect of nebularine and EOC (8-etoxy caffeine) on selection response for sternopleural hairs in \( D. melanogaster \).

character selected for was high number of sternopleural hairs. A family selection method was applied every two generations. The mutagens were given by intra-abdominal injections to adult flies of both sexes, aged 18-24 hours. Three concentrations (.050%; .025%; .010%) were tested for nebularine and two for EOC (.50%; .10%)

With nebularine a positive response to selection was obtained from the first generation on, but the response continued only in the .025% lines. From average number of hairs of 20.31 ± .099, after 8 selection cycles corresponding to 16 generations, one obtained values of 26.01 ± .379 and 29.52 ± .302 in two different replications. In the same period the two-untreated lines from an initial average value of 20.74 ± .098 reached a level of 21.29 ± .128 and 20.96 ± .106. The response to selection was found associated with an increase in variability, expressed both in terms of standard deviations and of coefficients of variation. With EOC the average

Agar-diffusion techniques were employed to investigate the antigenic specificities of proteins extracted from the following stocks of \( D. melanogaster \): \( \text{In}(1)w^{34w} \); a

The effects of two mutagens: EOC(8-etoxy caffeine) and nebularine on the induction of genetic variability as shown by progress under selection, has been studied. An isogenic stock has been used; the

The effects of two mutagens: EOC(8-etoxy caffeine) and nebularine on the induction of genetic variability as shown by progress under selection, has been studied. An isogenic stock has been used; the
values of sternopleural hairs reached, after 8 selection cycles, were 21.22 ± 1.15 and 21.19 ± 1.09, not significantly different from the values reached by untreated control lines. From experiments with plants one knows the mechanism of action of the two mutagens as being very different, EOC causing mainly chromosome breakage while nebuline (Ehrenberg and Gustafson, 1954) is supposed to produce mainly point mutations. Experiments designed to compare the mutagenic action of these substances in D. melanogaster are in progress.

Grell, R. F. The penetrance of sparkling-Cataract. A research note in DIS 33: 150 (1959) by H. J. Muller states that a single fourth chromosome carrying sparkling-Cataract (spa) in combination with two normal fours produces a wild-type eye. Flies have been synthesized in this laboratory that carry three free fourth chromosomes, each marked with a single dominant mutant (spa/CI/eyD), in an otherwise diploid background. The penetrance of spa in these tripl-o-four flies is complete, although its expression is less extreme than is one dose of spa in the diplo-four condition.

The mutant, spa, has also been used to mark the free fourth chromosome of tripl-o-four females that are homozygous for T(3;4)86D in order to follow the assortment of the free four and an extra Y chromosome. In this situation one dose of spa was found to be fully penetrant and classifiable both in the mother and in her tripl-o progeny. Mothers of this genotype (T(3;4)86D/T(3;4)86D/spa) mated to diplo-four males carrying two normal fours produced 332 spa and 329 non-spa offspring [Genetics 44: 421 (1959)]. Mothers of the same genotype mated to M-4/eyD males produced non-eyeless-Dominant flies that were either M-4 or spa, clearly demonstrating that the mutant phenotype is always classifiable when present in one dose in tripl-o-four flies.

It is of interest that one dose of spa in the F1 diplo-four hybrid between D. melanogaster and D. simulans (synthesized by E. H. Grell) shows an extreme mutant phenotype, whereas spa/simulans-four in an otherwise completely melanogaster background, as observed by Muller, is wild type. (This work was done at the Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee, operated by Union Carbide Corporation for the United States Atomic Energy Commission.)

Hildreth, P. E. and J. C. Lucchesi. Extended to eight additional species, including D. virilis; this author found an average of 5 to 6 spermatozoa in D. melanogaster and 50 to 100 spermatozoa in D. virilis eggs. Fertilization in D. melanogaster and D. virilis.

Using the Feulgen whole-mount procedure of von Borstel and Lindley (Stain Technol. 34:23, 1959), preliminary work on D. melanogaster eggs failed to show polyspermy (Hildreth, unpublished); therefore further cytological examination of D. melanogaster and also D. virilis eggs was conducted. In D. melanogaster 96 eggs were found in meiotic stages; among these 91 had a single sperm, 2 had two sperms, and 3 had no visible sperm. This is consistent with the observations of Hinton and Lucchesi (Genetics 45:87, 1960). Among 127 meiotic eggs of D. virilis, 87 eggs had a single sperm, no sperm was visible in 40 eggs, and no case of polyspermy was observed.

The reason for the differences between our results and those of Huettner and Counce are not known. Autoradiographic studies of fertilization in D. virilis are now being conducted in an attempt to obtain further information on the question of polyspermy. (This work was carried out under the auspices of the U.S. Atomic Energy Commission.)

Hochman, B. On the viability of the brown-Variegated/brown-Variegated heterozygote. More than 20 brown-Variegated (bv) alleles are listed in Bridges and Brehme (1944). Their viabilities in the homozygous state are described variously as, generally lethal, nearly always lethal, lethal in 95% of the cases, etc. The first one found,
bwV1 (usually referred to as Plum (Pm)), is reported to be, "generally lethal when homozygous, and also lethal with all other brown-Variegateds."

During the course of an experiment involving the Notch locus, data were obtained on the viability of the Pm/bwV57e genotype which demonstrate that the statement in the preceding sentence does not apply to this combination of bwV alleles. (Dr. E. H. Grell produced bwR57e by irradiating the SML chromosome. The presence of Cy in SML (see DIS 27: 57-58) precludes an examination of the bwV57e homozygote. When heterozygous with wild type, bwV57e causes the same eye mottling as Pm+. Under certain genetic and environmental conditions, it was observed that 0.2-0.4 of expected Pm/bwV57e individuals reached the adult stage. The findings also indicate that a slight temperature rise markedly increases the number of surviving Pm/bwV57e flies.

From the cross, w^a fa^no spl rb/fa^no spl; SML, Cy/Pm x y w^a N^40/Y; SML, Cy, Dp(1;2)w51b7 bwV57e/+, three classes of male offspring are expected if one disregards the X chromosomal genes. These three phenotypic categories may be expressed simply as: Cy/+; Pm/+; and Pm/bwV57e. If one assumes equal viabilities, each class should comprise one-third of the total male progeny. The expected numbers and observed results are presented in the following table:

<table>
<thead>
<tr>
<th>Temperature</th>
<th>Combined Cy/+ and Pm/+</th>
<th>Pm/bwV57e</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>obs.</td>
<td>exp.</td>
</tr>
<tr>
<td>26 ± 1°C</td>
<td>5,219</td>
<td>4,028</td>
</tr>
<tr>
<td>23.5 ± 0.5°C</td>
<td>10,888</td>
<td>7,788</td>
</tr>
</tbody>
</table>

The Pm/bwV57e genotype, while considerably below the other two classes in viability, survives too frequently to warrant the lethal designation. A more appropriate description would be semi-lethal. It is possible that the higher than expected rate of survival of this particular heterozygote is due in part to a reduction in the number of its competitors by factors associated with the cross. All female zygotes (except rare cases of nondisjunction) are N^40/fa^no, a lethal combination permitting less than 0.01 imagos. The Dp (1;2) w51b7, which had been placed in the SML, Cy, bwV57e chromosome by Dr. W. J. Welshons, also carries N^+. Since the presence of N^+ cancels the N^40/fa^no lethal interaction, there emerges a single group of female offspring namely, Pm/bwV57e. Male progeny of this same genotype must compete only with other males and genetically similar females. The effect that additional classes and larger numbers of females will have on the relative viability of Pm/bwV57e males is currently under investigation.

The following unpublished observations by Dr. W. J. Welshons on the phenotype of Pm/bwV57e have been confirmed:

1. Bodily dimensions range from clearly smaller than normal to approximately normal in size. The larger individuals are often characterized by a chubby (or bloated) appearance.
2. Wings either fail to expand completely or, if unfolded, they tend to diverge and curl to an extent greater than that of Curly alone.
3. Occasional patches of unpigmented microchaetae are another feature of the syndrome.

A limited number of tests show that these flies can be fertile.

The emergence of twice as many Pm/bwV57e adults at the higher of the two temperatures employed provides one more example of the strong influence of environmental factors on the viability of a given genotype. It is interesting to note that Gowen and Gay (1933) found that the extent of variegation is diminished by increased temperature. From the results reported here it appears that the degree of semi-lethality of this particular brown-Variegated heterozygote manifests a like tendency.

(This work was done at the Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee, operated by Union Carbide Corporation for the United States Atomic Energy Commission.)

Hunter, Alice S. and Sara Newball. Drosophila of old Providence Island.
varies from 18-30°C, depending on the sun and wind, but averages 24°C. The terrain varies from the swamps and bays to wooded hills and mountains up to 1,152 feet above sea level. The wind is generally northeast from December to February, and the weather is calm from then until September. During October and November there may be hurricanes with strong winds mostly from the North.

The island is about 15 square miles, and there are about 3,000 human inhabitants. There are many fruits which might be natural breeding sites of Drosophila, such as the semi-cultivated mangos, papayas, coconuts, bananas, and the wild grapes, pine- apples and guavas. However, despite the abundance of fruits, a total of only six different species of Drosophila were encountered in our collections made during the months of January and February.

Collections were made by sweeping over a bait composed of cut oranges, bananas and squash pulp. Whenever possible, bait was placed in shaded protected areas, but in some places there were no trees, and it was impossible to avoid the wind. In fifteen sites the various species encountered in at least one of the sweepings were sorted and counted. The totals of the six different species found in the counted collections are as follows: D. melanogaster-simulans-1,140, D. ananassae-1,017, D. hydei (?)-922, D. latifasciaeformis-493, and D. willistoni-236. No attempt was made to determine the relative proportions of melanogaster and simulans. The identity of hydei is being checked in test-crosses with a University of Texas stock. Because of the lack of electricity on the island, all of the willistoni group flies collected were taken to Bogotá for identification. A sample of 50 males was checked by studying the genitalia. In addition 50 females were isolated in separate culture jars and the offspring were checked. In all the 100 cases the male genitalia were those of D. willistoni.

Comparing one collection site with another, considerable variation in the relative frequencies of the different species was encountered. For example:

<table>
<thead>
<tr>
<th>Species</th>
<th>Free Town</th>
<th>Bush-pen</th>
<th>Camp</th>
</tr>
</thead>
<tbody>
<tr>
<td>D. melanogaster-simulans</td>
<td>38</td>
<td>58</td>
<td>131</td>
</tr>
<tr>
<td>D. ananassae</td>
<td>349</td>
<td>20</td>
<td>68</td>
</tr>
<tr>
<td>D. hydei</td>
<td>54</td>
<td>1</td>
<td>250</td>
</tr>
<tr>
<td>D. latifasciaeformis</td>
<td>1</td>
<td>1</td>
<td>163</td>
</tr>
<tr>
<td>D. willistoni</td>
<td>0</td>
<td>32</td>
<td>0</td>
</tr>
</tbody>
</table>

The lack of variety of species is notable, but may be related to the isolation of this island and the winds.

(Imaizumi, T. XXY strain derived from the wild Miyazu stock of D. melanogaster and its lethality. Of the lethal strains in the preceding report (DIS 33:140), it is ascertained genetically that a strain derived from an X-rayed male is XXY. Perhaps primary non-disjunction occurred in a sperm of the X-rayed Miyazu male. The crossing tests are as follows:

<table>
<thead>
<tr>
<th>Crosses</th>
<th>Free</th>
<th>Bush-pen</th>
<th>Camp</th>
</tr>
</thead>
<tbody>
<tr>
<td>Y/+/Basc ? x Basc ?</td>
<td>B/+</td>
<td>B</td>
<td>+</td>
</tr>
<tr>
<td>1) Y/+/Basc ? x Basc ?</td>
<td>235</td>
<td>172</td>
<td>318</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>725</td>
<td></td>
</tr>
<tr>
<td>(2) Y/+/Basc ? x w m f</td>
<td>red</td>
<td>eye orange</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>212</td>
<td>156</td>
<td>157</td>
</tr>
<tr>
<td></td>
<td>134</td>
<td>191</td>
<td>0</td>
</tr>
<tr>
<td>(3) Y/+/w m f x w m f</td>
<td>+</td>
<td>w m w m w m</td>
<td>y w m f m f</td>
</tr>
<tr>
<td></td>
<td>572</td>
<td>244</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>523</td>
<td>0</td>
<td>8</td>
</tr>
<tr>
<td>(4) Y/+/y w m f ? x y w m f ?</td>
<td>y w m f</td>
<td>y w m f</td>
<td>y w m f</td>
</tr>
<tr>
<td></td>
<td>449</td>
<td>231</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>413</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>1150</td>
<td>0</td>
<td></td>
</tr>
</tbody>
</table>

*A new character found in cross (4); it represents reduced bristles, not forked, phenotypically and its chromosomal constitution is XXY.
It is noticed firstly from the crosses (3) and (4) that the crossing-over between two X-chromosomes is very suppressed in this XXY strain. Next, at meiosis, three kinds of segregation are expected in the XX females: (a), (Miyazu-X, introduced-X) and (Y); (b), (Miyazu-X, Y) and (introduced-X); and (c), (introduced-X, Y) and (Miyazu-X). But we can prove that the last segregation (c) never occur in this strain by the tests in F1 flies of the cross (2). In F1 of this cross the chromosomal constitution of the females with hetero Bar (orange eye) phenotype is all XX and that of the females with wild phenotype all XXY. Further, it is clear that the wild male never appears in all cases. The XY zygotes (wild genotype) are not formed from the segregation (c) and the XXY males (wild genotype) derived from the segregation (b) would be lethal in this strain, though one knows that the XYY males are viable in general.

The percent mortality in F1 of several crosses is shown in the following table. The lethality of the XXY strain is given in the top cross (1) and that of the attached-X strain in the next cross (2); and the other three are controls. Percentages per the first total eggs tested are indicated in each stage.

<table>
<thead>
<tr>
<th>Crosses</th>
<th>Total eggs tested</th>
<th>Mortality in egg</th>
<th>Mortality in larva</th>
<th>Mortality in pupa</th>
<th>Total mortality rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Y+/+/Basç X (XXY)</td>
<td>757</td>
<td>66.4</td>
<td>1.3</td>
<td>19.5</td>
<td>29.2</td>
</tr>
<tr>
<td>Oregon-RS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(2) g2 ty/y (Attached-X) X</td>
<td>745</td>
<td>37.9</td>
<td>2.8</td>
<td>27.1</td>
<td>4.6</td>
</tr>
<tr>
<td>Oregon-RS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(3) Basç/Basç X (XX)</td>
<td>727</td>
<td>10.3</td>
<td>4.7</td>
<td>0.4</td>
<td>1.0</td>
</tr>
<tr>
<td>Oregon-RS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(4) Oregon-RS X</td>
<td>542</td>
<td>1.3</td>
<td>0.9</td>
<td>0</td>
<td>0.4</td>
</tr>
<tr>
<td>Oregon-RS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(5) Miyazu X</td>
<td>673</td>
<td>3.3</td>
<td>2.5</td>
<td>0</td>
<td>0.2</td>
</tr>
</tbody>
</table>

*U: unfertilized eggs; C-B, Bk and T indicate primary, middle and late stage in the embryonic development respectively.

Regarding triple-X females, it is revealed that they mostly die in the pupal stage, but some in the late larvae as indicated in the progeny of attached-X strain. But in this XXY strain the mortality rate in the pupal stage is evidently low as compared to the case of the attached-X. On the contrary the mortality in middle or late embryonic stages is higher. This fact suggests that the time of death of the triple-X females in this XXY strain is not in pupal stage, but it would be in embryonic stage, perhaps in the middle of the stage. Finally, the YY individuals die in the primary stage of embryonic development both in XXY and attached-X strains.

Ives, P. T. More data from ras²/ras⁴ and y/y² recombination tests.

In 1951 I reported a count of 66,907 flies from ras²/ras⁴ which showed no ras*. In 1961 I have added 69,585 flies to that total with the same result. No evidence of pseudo-allelism or conversion has appeared in these 136,292 flies. Both series of tests were done with outside markers, with free-X chromosomes and without autosomal Inversions. During the summer of 1961 Dr. Hexter and I plan to test ras⁴/ras⁴ with outside markers in free X's and with Cy and Ubx¹³⁰ present. The 69,585 flies recorded here also tested y/y². No y* flies appeared.
Increase significantly the rate of pupal oxygen consumption and adult egg production.

In studies of flies homozygous and heterozygous for the ebony gene found in a wild population, with randomization of other gene loci, showed heterozygosity to oxygen consumption and adult egg production.

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Kato, M. Influences of essential fatty acids on the growth and egg productivity of D. melanogaster.

Jacobs, M. E. Influence of ebony and + alleles on oxygen consumption and egg production in D. melanogaster.

Studies of flies homozygous and heterozygous for the ebony gene found in a wild population, with randomization of other gene loci, showed heterozygosity to oxygen consumption and adult egg production.

Studies of flies homozygous and heterozygous for the ebony gene found in a wild population, with randomization of other gene loci, showed heterozygosity to oxygen consumption and adult egg production.

Influences were tested by addition of the so-called essential fatty acids or vitamin F to the culture medium which was prepared by extracting lipids from the homogenized yeast with absolute alcohol and ether. In this medium the emergence rate of the adult fly was 8.0±4.0% on an average while in the standard medium it was 86.3±1.57%. When whole EFA were added to the medium it increased to 44.1±0.93%. When only EFA with double bonds at 6,7- and 9,10- position were added, the rate approached the level obtained with whole EFA, namely, 36±0.55%. In the EFA-deficient medium, most of the larvae died before pupation and most of those that pupated did not survive to adulthood. Neither abnormality in morphology nor that in sex-ratio were found. Daily ovulation of females emerged in the EFA-deficient medium is very low, being 9.3±1.13% as compared to that of 28.5±2.09 produced by the female reared in the EFA added medium.

Kishin, Aziz F. Induction of mutations in D. melanogaster by "immersion" in solutions.

The experiments designed to test the possibility of inducing lethal mutations in Drosophila melanogaster by "immersing" larvae or pupae in formaldehyde solutions gave definite positive results (Kishin, published and unpublished). However, it should be questioned whether in these cases the formalin itself or even a derived product is the sole mutagenic agent, or whether the mere immersing has an effect and thus should be held responsible for all or some of the observed changes.

To test the second possibility a set of experiments were started in which tap water, saline solution prepared with distilled water, and formaldehyde solutions were used as media for immersion. The same procedure was followed for the three agents used, and larvae were treated for 30 minutes and 60 minutes. In all cases dominant lethality (D.L.) was calculated over certain period. Preliminary results indicate the following:

1. Tap water induces more D.L. than either saline (0.05% Na Cl) or 10% formaldehyde solutions.
2. Saline solution induces about the same percentage of D.L. as 10% formaldehyde solution when either is used for 30 minutes.
3. Saline induces more D.L. than formaldehyde when either is tested for 60 minutes.
4. 10% Formaldehyde used for 30 minutes gives about 2-3 times as much D.L. as when applied for 60 minutes.
5. Saline solution used for 30 or 60 minutes gives about the same result.

Mead, Charles G.*, and Allen S. Fox. The characterization of the deoxyribonucleic acids of Drosophila melanogaster.

DNA was isolated from lyophilized Oregon R flies by a modification of the Kay, Simmons and Dounce procedure (1952). Approximately 2 mg. of DNA were recovered per gram dry weight of flies. The isolated product had an E(P) of 6930, exhibited a 20% increase in O. D. at 260 m u upon alkaline denaturation, and was unusually low in viscosity. Upon precipitation of the isolated product with cold ethanol, two types of DNA were observed. One of these was fibrous, typical of most DNA's, and the other was of a flocculent nature. After exhaustive deproteinization the two types of DNA retained their differences.

Perchloric acid and formic acid hydrolysates of the isolated DNA, when subjected to paper chromatography, exhibited an exceptional UV-absorbing spot. This unusual compound was identified as 5-methylcytosine by chromatographic and spectrophotometric means. The two types of ethanol precipitable DNA's, one fibrous and the other flocculent, were analyzed for their 5-methylcytosine contents. A preparation of DNA was precipitated with cold ethanol and the fibrous DNA collected by winding the fibers on a glass rod. The fraction which could not be collected in this manner was considered flocculent DNA. Each fraction was hydrolyzed with perchloric acid, the hydrolysates chromatographed on paper, the UV-absorbing spots eluted, and the molar concentration...
of 5-methylcytosine and thymine measured spectrophotometrically. The molar ratio (5MC/T) of the fibrous fraction was 0.165 ± 0.008 (n=4), and that of the flocculent fraction was 0.234 ± 0.007 (n=6). Thus, the pyrimidine composition of these two fractions is definitely different.

Ion exchange chromatography of a phosphodiesterase digest of whole DNA yielded five UV-absorbing peaks. The molar concentrations of these peaks, as calculated from their respective molecular extinction coefficients at 260 μM, are as follows:

<table>
<thead>
<tr>
<th>Peak No.</th>
<th>Nucleotide</th>
<th>M</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Deoxy-5-methylcytidilic</td>
<td>0.79</td>
</tr>
<tr>
<td>2</td>
<td>Deoxycytidilic</td>
<td>1.63</td>
</tr>
<tr>
<td>3</td>
<td>Deoxythymidilic</td>
<td>2.78</td>
</tr>
<tr>
<td>4</td>
<td>Deoxyadenylic</td>
<td>2.76</td>
</tr>
<tr>
<td>5</td>
<td>Deoxyguanylic</td>
<td>2.47</td>
</tr>
</tbody>
</table>

The existence of 5-methylcytosine in the DNA of Drosophila makes possible a variety of experiments which might lead toward the elucidation of the relationship between the genetic units and the chemistry of DNA. We now have a non-randomly distributed label in a DNA which can be defined genetically with great accuracy and manipulated with ease. An attempt to identify this label with specific genetic units could yield valuable information.

(Supported by grant C-2440 from the National Institutes of Health, U.S. Public Health Service. *Predoctoral Fellow, National Institutes of Health, U.S. Public Health Service).

Meyer, Helen U. and Michael L. Criswell. Crossover analysis of sex-linked mutations induced in oogonial cells by repeated treatments with 4000r of X-rays.

When heavy doses of X-rays are divided into installments and given at 4-day intervals, only the potential chromosomal breaks from the same irradiation can collaborate with one another. The proportion of chromosomal rearrangements is therefore determined independently by each of these particular installments. This method of fractionated treatment lowers the mortality of the treated cells considerably in comparison with that with that occurring when the same total dose is given as a continuous, "acute" treatment. Such a method has been used in many of our experiments in which mutations induced in gonial cells were studied, where a rather heavy X-ray dose is needed to counteract the relatively low X-ray sensitivity of these cell stages. Not many gross changes in chromosomal structure were expected when irradiating in this manner; however it was desirable to get more information on this question. From an experiment in which a total dose of 24,000r, given in 6 installments, resulted in 15.2% sex-linked lethals, we selected nine different cases of sex-linked mutations at random, derived from eggs laid 8–12 days after the last irradiation, for analysis of the crossover pattern. The mutation-carrying X-chromosomes were originally of isogenic origin, had the normal gene sequence and were marked by an oc. One was a visible mutation, one a detrimental (about 15% of expected survivors), four near-lethals (less than 10% of expected survivors), and three fully lethal. In only one out of these nine cases had some rearrangement of the gene sequence occurred; this had resulted in a small inversion which was connected with a lethal. In the remaining 8 cases the crossover values were found to be in reasonable agreement with the expected map frequencies. Similar, more recent studies on mutations recovered from gonial cells by only one treatment with 4000r seem to agree with these findings that intrachromosomal rearrangements may be recovered from gonial cells, but in a much lower proportion than when a similar dose is given to mature spermatozoa.

(This work was supported by a grant to H. J. Muller and associates from the Public Health Service, Contract RG-5286 (3), and a grant from the National Science Foundation Summer Programs for Secondary School Students.)


To learn how sperm from successive copulations is utilized, young virgin females homozygous for the second chromosome markers cn bw sp were mated first with
homozygous cn sp males and then remated to homozygous bw sp males. Only one copulation with each of these two types of males was allowed, and the females isolated immediately afterwards.

An interval of 4-5 days was unfortunately necessary between the two inseminations, since the females refused to mate for a second time before a sizable number of fertilized eggs had been deposited. It was found that the length of this interval varied with temperature and with the type of culture medium used. That various strains behave differently in this respect has been pointed out by Ehrlich in a similar study (D.I.S. 33:129-130, 1959).

39 females, which had been observed to have copulated once with each type of male and had been immediately separated from them, were bred individually and transferred to fresh culture vials every 24 hours for 12 successive days. After this time only one of them still laid fertilized eggs. All offspring from these daily broods were classified for phenotype, cn sp progeny indicating fertilization by the first male, and bw sp by sperm from the second one.

Two parent-females gave only one type of offspring, cn sp in one instance and bw sp in the other one, even though they had been observed to have copulated with both types of males. This could be explained by either non-functional sperm (as in XO males) or by copulation without insemination.

The results from the remaining 37 doubly inseminated females are summarized in the following table:

<table>
<thead>
<tr>
<th>PERIOD</th>
<th>EGGLAYING</th>
<th>AV. NO.</th>
<th>PHENOTYPE OF F₁</th>
<th>% F₁, FIRST INSEMIN.</th>
</tr>
</thead>
<tbody>
<tr>
<td>BEFORE 2nd Insemination (4-5 days)</td>
<td>31*</td>
<td>19.5</td>
<td>2936</td>
<td>-</td>
</tr>
<tr>
<td>AFTER 2nd Insemination days 1-2</td>
<td>36.5</td>
<td>35.4</td>
<td>64</td>
<td>2521</td>
</tr>
<tr>
<td>3-4</td>
<td>33.0</td>
<td>24.3</td>
<td>27</td>
<td>1578</td>
</tr>
<tr>
<td>5-6</td>
<td>27.0</td>
<td>13.6</td>
<td>13</td>
<td>719</td>
</tr>
<tr>
<td>7-8</td>
<td>19.5</td>
<td>5.1</td>
<td>4</td>
<td>196</td>
</tr>
<tr>
<td>9-10</td>
<td>11</td>
<td>3.5</td>
<td>1</td>
<td>76</td>
</tr>
<tr>
<td>11-12</td>
<td>6</td>
<td>3.8</td>
<td>1</td>
<td>45</td>
</tr>
<tr>
<td>Totals, days 1-12</td>
<td>110</td>
<td>5135</td>
<td>2.1</td>
<td></td>
</tr>
</tbody>
</table>

*No record kept on the no. of F₁ from 6 other P-♀♀ during this period, which are included in the second part of this table.

Thus an average of only 2.1% of the total offspring obtained after the second mating was derived from sperm retained from the first insemination. However, this frequency did not vary significantly from brood to brood except for a slight drop after the first two days. These findings therefore do not support the view that spermatozoa are stored in the order in which they are received, but imply that they are mixed in the storage organs of the females and used at random to fertilize the eggs, in agreement with similar results obtained by Ehrlich as described in the report quoted above.

At the end of the observation period only 6 of the initial 37 females were still reproducing. Since the females were not yet very old, this was undoubtedly due to sperm exhaustion.

Fewer offspring resulted from the first insemination than from the second one. This might have been due to a variety of factors: the lower egg production of very young females, possibly a not yet fully developed storage capacity for sperm, or discharge of stored sperm before or during the second copulation. It also might have been due to the different genotype of the first male.

On the basis of our data it is possible to make a tentative estimate of the number of eggs which can be successfully fertilized by the amount of sperm deposited during one copulation. By adjusting the number of progeny from the first male by addition of an estimated 568 (for those 6 females for which no record was kept during the first period), a total of 3614 offspring were obtained from the first male, and 5135 from the second male. Dividing their average by 37, we obtain an estimate of
118 offspring from one combination; or, if one considers the result of the second mating to be more representative for the middle period of reproductive life, the number would be 140 successfully fertilized eggs.

(This work has been supported by a grant to Dr. H. J. Muller and associates from the U.S. Public Health Service, Contract RG-5286(3)).

Novitski, E. Post-treatment of irradiated sperm by low temperature. In a talk given before the Conference on Problems of General and Cellular Physiology in 1949, I made the statement that post-treatment of irradiated sperm by low temperature caused an increased recovery of sex-linked lethals. The transcript of this talk was subsequently published (American Naturalist, LXXXIII, 185-193) without the inclusion of any supporting data. Since that time I have been asked several times about these experiments.

In these runs, Basc 99, previously inseminated by Canton-Sa', were irradiated in order to remove the ambiguity of possible differential sensitivity of the stages of spermatogenesis. A control series, irradiated with 3600r at 25°C, gave 128/1246 (9.7%) lethals; a parallel series, held at 0.5°C during the irradiation, gave 34/138 or 24.6% lethals.

In the more extensive sets described below the dose was decreased to 1800r. The controls at 25°C gave 54/1166 (4.6%) lethals. An unirradiated control exposed to -1°C for 14 hrs. produced no lethals in a total of 877 tests. When the cold treatment (6 hrs.) preceded the irradiation (but with an hour and a half separating the two) there was no appreciable change in the lethal frequency (50/1172 = 4.3%). In two runs the females were kept at 0°C during the treatment and the lethal rate jumped appreciably (78/1113 = 7.0%; 99/1121 = 8.8%). Finally, in a run in which the females were exposed to the low temperature immediately after X-raying, the lethal rate was 11.7% (11/94).

Although statistically significant, the low numbers in this last case, which was the only one involving bona fide post-treatment only, suggested repetition. Unfortunately, at this stage, desemination (which was undoubtedly responsible for the low numbers in this last experiment) became a serious problem and attempts at repetition failed dismaly. This line of experimentation was then abandoned, although the effect of cold temperature in deseminating females was duly investigated and published.

Parker, D. An apparent incompatibility among seemingly normal members of the species D. simulans. Peculiar results were observed in experiments with D. simulans whenever a cross involved the mutant vermilion. Difficulties with D. simulans crosses cropped up continuously when vermilion was mated with the mutants yellow-white, black, sepia, scarlet peach, peach-hairless, plum, and with various wild type stocks. These difficulties were due to rather unpredictable abnormalities in the development of the offspring.

The crosses using vermilion males were moderately successful with the only deviation from normal being in the reduced amount of progeny from each cross. But the results when using vermilion females were more noteworthy. In roughly 70% of all the vermilion female crosses, development went no further than the egg stage. In approximately 15% of all crosses involving vermilion females, the development progressed until death occurred in the larva stage. The time of death established no predictable time pattern. Death occurred any time between the first instar and pupation. In the remaining 15% of the crosses, adult offspring emerged only to die sometime within the first five days. Even when such adult progeny did appear, they were much reduced in number from what is normally found.


That is, in flies in which the site of a posterior dorsocentral bristle is ac tissue, a bristle will usually not differentiate even when most of the surrounding tissue is ac+. Conversely, when the site of the bristle is ac+, differentiation is always initiated regardless of the amount and distribution of surrounding ac tissue.
Out of 1600 duplication carrying male offspring of the cross of females, Dp(w<sup>VC</sup>)6094b/y w f:=/Y, by males, y ac w<sup>a</sup> ct<sup>f</sup> y<sup>S</sup> /Y<sup>L</sup>, 110 had some loss of the duplication (carrying non-yellow, y<sup>*</sup>, and non achaete, ac<sup>+</sup>) and were mosaics involving the dorsocentrales. In this genotype, 99% of the male progeny not carrying the duplication (patroclinous males) had both anterior and posterior dorsocentrales missing, so both of these sites were scored. Of these 110 mosaics, 91 exhibited autonomy as described above, and 19 exhibited non-autonomy in which dorsocentral bristles differentiated in sites in ac tissue close to ac<sup>+</sup> tissue. Stern interpreted this as due to spread of ac<sup>+</sup> material into the ac tissue patch. However, 12 of the 91 autonomous cases had similar proximity of ac<sup>+</sup> tissue to the bristle site with maintenance of ac autonomy. Seven cases were observed in which a dorsocentral bristle differentiated at an abnormal site near a bristle site occupied by achaete tissue, but apparently within a potential area of differentiation.

Results are consistent with Stern's observations and his interpretation that ac and ac<sup>+</sup> are not establishing a regional singularity but responding to a "prepatterning" present in both genotypes.

Sandler, L. and C. W. Cotterman.
A possible interpretation of the conversion of X chromosome by SD.

In the presence of SD (which is located on chromosome II and conditions abnormal segregation of this chromosome pair in males), some X chromosomes may be specifically converted into suppressors of SD action (Sandler and Hiraizumi, 1961).

It is not known by what mechanisms either (1) the conversion of the X chromosome takes place, or (2) the converted X chromosome suppresses SD action. One possible supposition that can account for both of these effects is as follows. It may be imagined that the conversion results from the X chromosome physically acquiring a part of the SD locus. This modified X chromosome, in subsequent generations, can pair with the SD region of chromosome II in SD heterozygotes and thus prevent SD from pairing properly with SD<sup>+</sup>. This should indeed suppress the phenomenon of segregation-distortion because synapsis of SD and SD<sup>+</sup> is known to be necessary for distortion (Sandler, Hiraizumi and Sandler, 1959).

The only test of this notion that immediately suggests itself is to see whether there are any differences in the segregation of the X and chromosome II in SD heterozygotes, according to whether or not the X chromosome has been converted.

Accordingly, males heterozygous for SD and In(2LR)Cy (Cy, itself, suppresses SD action by failing to pair properly with SD and is used here so as to maximize the probability of X-II synapsis) with either a modified X or an unmodified X, were crossed to cn bw females. The results were as follows:

<table>
<thead>
<tr>
<th>Type of X</th>
<th>Cy&lt;sup&gt;++&lt;/sup&gt;</th>
<th>Cy&lt;sup&gt;00&lt;/sup&gt;</th>
<th>Cy&lt;sup&gt;++&lt;/sup&gt;</th>
<th>Cy&lt;sup&gt;00&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Modified</td>
<td>229</td>
<td>259</td>
<td>265</td>
<td>246</td>
</tr>
<tr>
<td></td>
<td>(.23)</td>
<td>(.26)</td>
<td>(.27)</td>
<td>(.25)</td>
</tr>
<tr>
<td>Normal</td>
<td>315</td>
<td>288</td>
<td>315</td>
<td>277</td>
</tr>
<tr>
<td></td>
<td>(.26)</td>
<td>(.24)</td>
<td>(.26)</td>
<td>(.23)</td>
</tr>
</tbody>
</table>

It is clear that X-II segregation is the same irrespective of whether or not the X has been modified. Thus we must suppose that either the suggested hypothesis is incorrect or that pairing between the X and SD, while sufficient to suppress SD action, is not of such a kind as to affect the pattern of segregation.

The relation between allelic phenotype and allelic localization within the dumpy region.

A specific sublocus has been established in the dumpy gene for the ov mutants. These ov mutants exhibit two phenotypic effects of the dumpy gene, the oblique wing, written as o, and the bristle disturbances and protuberances of the thorax called vortex, written as v.

A series of six independently arising ov mutants (ov<sub>1</sub>, ov<sub>2</sub>, ov<sub>3</sub>, ov<sub>51</sub>, ov<sub>52</sub>, ov<sub>h</sub>) were localized within the genetic map of the dumpy region. A modified "four-point" test using the outside markers -- echinoid (ed) at 11.0 and clot (cl) at 16.5 was used in the mapping procedure. The position of these alleles was determined with respect to two other alleles of the dumpy region, the thoraxate (tv) allele on...
on the left of ov^1 and the vortex (v) allele on its right. In all instances the other ov alleles were mapped between lv and v. These localizations establish a sublocus within the dumpy gene which appears to be specific for the ov expression. No other alleles have been localized at this site. Therefore, the region may be referred to as the ov sublocus. (See Table 1 for the summarized results.)

One of the ov mutants, called ov^h (dumpy-humpy-like) shows a more extreme effect than the other ov alleles, and it is a facultative lethal in the homozygote. It is not lethal, however, in the heterozygous compound with any of the alleles of the dumpy region containing the lethal factor (e.g. l, ol, lv, olv). The localization of this allele between thoraxate, lv, and vortex, v, suggests that the ov^h mutant might be a minute deletion of the ov sublocus, or that it might occupy a separable site within the region.

These localizations indicate that mutation at the dumpy locus will usually be specific in expression for that portion of the map which is affected. A possible exception to this may be found for the olv alleles. These mutants are characterized by a loss of the total function of the dumpy gene. In phenotype they resemble deficiencies for the dumpy region. These extreme mutants could possibly be located anywhere on the pseudoallelic map. Localization of a series of olv alleles is now in progress (Southin and Carlson, unpublished).

The localization of the ov alleles to a specific sublocus makes possible an investigation of the fine structure of this portion of the dumpy region. A selective technique has been designed (see p., this issue) which will be used to investigate this possibility of fine structure. Southin (1961, unpublished) has obtained recombination between two similar oblique alleles, o^2 and o^2m. Resolving power of this test was approximately 1 X 10^{-5} and we anticipate possible resolution with the selective technique to reach 1 X 10^{-7}.

<table>
<thead>
<tr>
<th>Trans Alleles</th>
<th>Verified &quot;Conversions&quot;</th>
<th>Verified Single C.O's</th>
<th>Total Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>lv^1/ov^1</td>
<td>1</td>
<td>6</td>
<td>66,009</td>
</tr>
<tr>
<td>lv^1/ov^h</td>
<td>0</td>
<td>1</td>
<td>3,548</td>
</tr>
<tr>
<td>lv^1/ov^x</td>
<td>0</td>
<td>2</td>
<td>22,668</td>
</tr>
<tr>
<td>lv^1/ov^n</td>
<td>1</td>
<td>2</td>
<td>22,766</td>
</tr>
<tr>
<td>lv^1/ov^51f</td>
<td>0</td>
<td>1</td>
<td>34,544</td>
</tr>
<tr>
<td>lv^1/ov^52b</td>
<td>0</td>
<td>1</td>
<td>21,477</td>
</tr>
<tr>
<td>v^2/ov^1</td>
<td>0</td>
<td>7</td>
<td>35,200</td>
</tr>
<tr>
<td>v^2/ov^h</td>
<td>0</td>
<td>1</td>
<td>2,955</td>
</tr>
<tr>
<td>v^2/ov^x</td>
<td>0</td>
<td>2</td>
<td>7,894</td>
</tr>
<tr>
<td>v^2/ov^52b</td>
<td>0</td>
<td>1</td>
<td>20,820</td>
</tr>
<tr>
<td>v^2/ov^51f</td>
<td>0</td>
<td>1</td>
<td>7,298</td>
</tr>
<tr>
<td>v^2/ov^n</td>
<td>0</td>
<td>2</td>
<td>4,539</td>
</tr>
<tr>
<td>TOTALS</td>
<td>2</td>
<td>27</td>
<td>249,718</td>
</tr>
</tbody>
</table>

This work is supported by Grant G 14222 from the National Science Foundation.

In a recent paper Hadorn (1959 Arch. Jul. Klaus-Stiftg., 34:234-239) reported that the phase specificity of 19 recessive lethals had remained unchanged over the past 7-8 years. Similar observations made in this laboratory tend to confirm Hadorn's general conclusions. Several strains of second chromosome recessive lethals, which manifest developmental effects in the larval-pupal and pupal stages, have been maintained in a balanced condition (Cy/le) for several years. During this interval the period of action of various lethals had been determined on several occasions in the course of various experiments, either by counts or by direct observations of their visible effects. In most of the lethals the phase specific action and characteristic phenotypes remained unchanged but a few had lost their larval-pupal or pupal effects and were later discarded. A summary of the observations on the various lethals are given below:
A series of lethals on chromosome II were extracted from a population cage of D. melanogaster descended from the control #3 population of Bruce Wallace, which was initiated with Oregon-R in 1949 and had been maintained for at least 170 generations at 25°C in a plastic population box before this laboratory obtained subsamples from it. Lethals were extracted in conjunction with an attempt to obtain "good viability" chromosomes (done by Mr. Archie Allen using the Cy L/Pm technique), and they were maintained in balanced condition with Cy L. Salivary analysis revealed no aberrations. The frequency of lethal chromosomes in the cage was estimated at 0.175, but linkage tests showed that the lethals were all in the left arm of chromosome II. Mapping of lethals was done in two sets of crosses: the first set utilizing markers at pr c sp and the second set using markers in either right or left arms depending on where the
first set of crosses placed the lethal: al dp b pr and pr c px sp. Females heterozygous for lethal/recessive markers were backcrossed to males with these markers homozygous plus a dominant such as Cy or B1. All crossover classes containing the dominant were backcrossed to the lethal/Cy L. By counting 100 flies in the progeny and finding no wild type, lethality was classified.

The following linkage map was obtained from classifying ten lethal chromosomes; a single lethal obtained as a spontaneous mutant in another experiment (Allen) (II-1-5g) was placed in the right arm:

![Linkage Map]

By testing lethals for allelism, 17 occurred four times, twice with another lethal locus (for example 12 was lethal at the 17 locus plus 12e at 53.4); 11 occurred twice; and in addition two other multilocus lethals were obtained which have not yet been located exactly. With such high frequency of certain lethals and high frequency of multilocus lethals, it might be inferred that high fitness must be conferred upon lethal heterozygotes in this population. Relative viability tests are now being made by the senior author. Certainly the non-random distribution of these lethals would imply possibly blocks of genes in the left arm of chromosome II which might be heterotic.

Lethals obtained by similar tests on chromosome III are currently being carried out also. Allelism is very high but linkage analysis has not been completed.


In order to find out whether females of Drosophila melanogaster or Drosophila simulans caught in nature would produce offspring of unusual sex-ratios, traps were set out at various localities, far enough away from experimental laboratories to exclude the possibility of trapping laboratory-bred flies. Each female was put singly into a culture bottle and allowed to lay eggs for seven days.

Of a total 626 females 606 produced offspring in a 1~ :1~ ratio, 14 females gave an F1 in the ratio of 3~ :1~ , and 6 females had only female progeny. Each one of the all-female progeny was first mated to D. melanogaster, then to D. simulans males, and none were fertile. One of the mothers was mated to D. melanogaster males and gave offspring in the normal sex-ratio.

The results fit well with Sturtevant's findings of 1929, where he reports 10-40% all female hybrid offspring from D. melanogaster \( \varphi \times D. \textit{simulans} \delta \delta \) in crosses done in the laboratory. In the reciprocal crosses, i.e. D. melanogaster \( \delta \delta \times D. \textit{simulans} \varphi \varphi \), he obtained 2% all-male hybrids. None was found in our experiments.

Whether the hybridizations occurred in nature or within the trapping bottles is unknown. In any case, no maternally determined "sex-ratio" condition was found in the sample of 606 tested females.

Strangio, V. A. Radiosensitivity to certain breakage aberrations during spermatogenesis in D. melanogaster. A re-investigation of radiosensitivity during the spermatogenesis of D. melanogaster has been made utilising a doubly-marked Y-chromosome, \( y^0 \text{YBS} \) (see Brosseau et al, 1961. Genetics 46:339-346 for full description). Recently emerged Canton-S males carrying this Y-chromosome were irradiated with 1000r and then mated daily for twelve days afterwards either to three \( y \ \text{apr ec} \) females (Series I) or to four attached-X \( y \ \text{v f} \) car females (Series II). In Series I, the regular offspring were wild type females and apr ec - Bar males; in Series II, \( y \ \text{v f} \) car - Bar females and wild type males. The improved technique described here allows the following irradiation-induced aberrations to be detected simultaneously:

(a) sex-chromosome losses as:
XO males in Series I, phenotypically "y apr ec".
XXO females in Series II, phenotypically "y v f car".

(b) individual Y-chromosome marker deletions as:
XY" males in Series I, where loss of \( y^0 \) yields \( y \ \text{apr ec} - \text{Bar} \) ("yellow") males or loss of B\(^0\) results in apr ec ("non-Bar") males.
XXY" females in Series II, loss of \( y^0 \) produces \( y \ \text{v f} \) car - Bar ("yellow")
<table>
<thead>
<tr>
<th>Day</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SERIES I</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Offspring</td>
<td>2390</td>
<td>2207</td>
<td>2637</td>
<td>2125</td>
<td>1625</td>
<td>845</td>
<td>420</td>
<td>409</td>
<td>1862</td>
<td>1858</td>
<td>1481</td>
<td>1747</td>
</tr>
<tr>
<td>% &quot;y apr ec&quot; ♀♂</td>
<td>0.04</td>
<td>0.09</td>
<td>0.15</td>
<td>0.56</td>
<td>0.98</td>
<td>1.07</td>
<td>1.43</td>
<td>-</td>
<td>0.21</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>% &quot;yellow&quot; ♀♂</td>
<td>-</td>
<td>0.05</td>
<td>-</td>
<td>0.09</td>
<td>0.06</td>
<td>0.83</td>
<td>0.95</td>
<td>0.24</td>
<td>0.05</td>
<td>0.16</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>% &quot;non Bar&quot; ♀♂</td>
<td>0.04</td>
<td>0.09</td>
<td>0.04</td>
<td>0.19</td>
<td>0.25</td>
<td>0.71</td>
<td>0.48</td>
<td>1.71</td>
<td>0.05</td>
<td>0.11</td>
<td>0.07</td>
<td>0.06</td>
</tr>
<tr>
<td>% &quot;Bar&quot; ♀♂</td>
<td>0.04</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>0.24</td>
<td>0.95</td>
<td>0.24</td>
<td>-</td>
<td>-</td>
<td>0.14</td>
<td>-</td>
</tr>
</tbody>
</table>

| **SERIES II** |     |     |     |     |     |     |     |     |     |     |     |     |
| Total Offspring | 1278 | 2235 | 2330 | 2287 | 1245 | 952 | 380 | 194 | 997 | 1709 | 1582 | 1685 |
| % "y v f car" ♀♂ | 0.31 | 0.22 | 0.26 | 0.48 | 1.37 | 1.05 | 2.63 | 1.58 | 0.10 | 0.12 | -   | -   |
| % "yellow" ♀♂ | -   | -   | -   | -   | 0.16 | 0.32 | 2.63 | 1.03 | 0.20 | 0.23 | 0.13 | -   |
| % "non Bar" ♀♂ | -   | 0.04 | 0.26 | 0.57 | 0.64 | 1.47 | 3.42 | 4.12 | 0.20 | 0.12 | -   | -   |
| % "non yellow" ♀♂ | -   | -   | 0.04 | 0.17 | 0.08 | 0.11 | 0.79 | 0.52 | -   | -   | -   | -   |
| % "Bar" ♀♂ | -   | -   | -   | -   | -   | 0.11 | 0.26 | -   | -   | -   | -   | -   |
females or loss of bS yields v f car ("non-Bar") females.

(c) non-disjunction of X and Y as:
  XX females in Series I, phenotypically "Bar".
  XY males in Series II, also "Bar".

(d) large interstitial X-deletions as:
  XXX hyperploid females in Series II, phenotypically "non-yellow" and
  usually also showing one or two of the v f car markers.

The results are summarized in the accompanying table and are similar to those published as separate experiments by Ives (1960), SHvhagen (1960) and Chandley & Bateman (1960). The egg-laying period of the females is kept constant for each brood so that the relatively low adult numbers on the seventh and eight days are probably a reflection of maximal dominant lethal induction. This has been confirmed by direct egg mortality counts which incidentally also reveal a remarkable consistency in the radiosensitivity patterns for individual males. The aberration frequency versus brood curves for sex-chromosome loss, Y-deletions and X-deletions are all essentially similar and reach peak level on the seventh or eight day after irradiation. When these are compared with the onset of induced non-disjunction of X and Y chromosomes which must occur before their separation during the reductive meiotic division, it appears that the chromosomes exhibit their greatest radiosensitivity to the induction of at least these types of aberration during the early meiotic stages i.e. in the spermatocytes. Further investigations are in progress with ring and inverted X-chromosomes.

In earlier experiments it was observed that treatment with cyanide following high-intensity X-irradiation (2200 r/min.) resulted in a significant increase of the frequency of sex linked lethals and translocations in stages with greatest sensitivity to X-rays (Sobels 1960). These stages, presumably corresponding to spermatids, were sampled by means of the "brooding technique" after treatment of adult males. The extent to which cyanide enhanced the mutation rate showed, however, a considerable variation from experiment to experiment. This was felt as a serious handicap at a further analysis of the post-treatment effect. Assuming that susceptibility to the action of cyanide is restricted to one particular stage only, imperfections of the brooding technique in specific sampling could be responsible for the variation mentioned above. Since according to observations of Khishin (1955) and Oster (1955) radiated spermatids can be obtained in a more selective manner by treating 48-hour pupae, we investigated whether post-treatment of 48-hour pupae would yield more uniform results. Also, this method would be less time consuming than that of sampling spermatids by the brood technique from treated adults.

Male pupae of the genetic composition In(1)dl-49, y B/sc8.Y; bwP were irradiated with either 1200 or 2000 r at a dose rate of 3000 r/min., 100 KVP, 3.9 mA, without additional filtration. Ninety seconds after completing the radiation, part of the pupae were exposed to hydrocyanic acid, equivalent to 37.5 mg KCN, at a rate of flow of 100 ml/min. during 5 minutes. Preliminary tests had shown that with this procedure sterility was approximately 25% and mortality less than 10%. After hatching the males were mated individually to three females of the YSIn(1)EN-YL, y; st stock. Their progeny was tested for the incidence of sex-linked lethals and translocations of the II-III, Y-II, Y-III and Y-II-III types, according to Muller's (1954) multipurpose method. The results are presented in tables 1 and 2.

Table 1

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Dose (r)</th>
<th>n. chrom.</th>
<th>leth.</th>
<th>% leth.</th>
</tr>
</thead>
<tbody>
<tr>
<td>R1</td>
<td>1200</td>
<td>968</td>
<td>129</td>
<td>13.3</td>
</tr>
<tr>
<td>R1-CN</td>
<td></td>
<td>914</td>
<td>139</td>
<td>15.2</td>
</tr>
<tr>
<td>R2</td>
<td>2000</td>
<td>436</td>
<td>95</td>
<td>12.8</td>
</tr>
<tr>
<td>R2-CN</td>
<td></td>
<td>293</td>
<td>69</td>
<td>23.5</td>
</tr>
</tbody>
</table>
Table 2

<table>
<thead>
<tr>
<th>Experiment</th>
<th>Dose (r)</th>
<th>Translocations (total number)</th>
<th>Translocations II-III</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>number gametes</td>
<td>transl.</td>
</tr>
<tr>
<td>R1</td>
<td>1200</td>
<td>424</td>
<td>71</td>
</tr>
<tr>
<td>R1-CN</td>
<td></td>
<td>483</td>
<td>96</td>
</tr>
<tr>
<td>R2-</td>
<td>2000</td>
<td>260</td>
<td>57</td>
</tr>
<tr>
<td>R2-CN</td>
<td></td>
<td>199</td>
<td>59</td>
</tr>
</tbody>
</table>

The data show that post-treatment enhanced the frequency of both lethals and translocations, though not in a significant manner. Compared to the observations on treated flies, the modification is less pronounced. The method of treating pupae does not offer, therefore, technical advantages for a quantitative study of the cyanide effect.

Terzaghi, Eric and E. Novitski. An attempt to produce fertile "transformed" males. Because of its obvious technical utility and its intrinsic developmental genetic interest, a number of attempts have been made in the past, by several investigators, to obtain a fertile "transformed" male (X/X; tra/tra). The authors have performed a large-scale radiation experiment in an attempt to produce the desired "transformed" male.

In a large-scale radiation experiment, it is of great benefit to have an experimental set-up in which a large number of individuals may be tested for the desired characteristic with a minimum of time expended. Towards this end, the following mating scheme was designed, in which the progeny of the last cross select themselves in respect to the desired characteristic, fertility.

\[
yf:=(RA)/Y^S, Y^R; +/-/tra \quad x \quad x^{D+}B^S.Y^S/Y^L,Y^R; +/- \quad \downarrow \quad \text{irradiated with 4000r}
\]

\[
w^a/V; tra/tra \quad \text{or} \quad +/-/tra \quad x \quad yf:=(RA)/B^S.Y^S; +/-/tra \quad \text{or} \quad +/- \quad \downarrow \quad w^a/V/B^S.Y^S; +/- \quad \text{or} \quad +/-/tra \quad \text{or} \quad tra/tra, yf:=(RA)/Y; +/- \quad \text{or} \quad +/-/tra \quad \text{or} \quad tra/tra
\]

Among the progeny of the first mating, no fertile males are expected, hence, rigorous and frequent virgin collection is eliminated. Among the progeny of the second mating, no fertile males would normally be expected except in that case when the appropriate dominant mutation had been induced, or the appropriate recessive allele of "transformer" had been induced.

Contingencies which would produce undesired fertile males, such as breakdown of the double X (reversed acrocentric) or non-disjunction of the X and Y in the irradiated male, have been met, respectively, by making certain that both arms of the double X contain lethals and including the Bar Stone fragment in the chromosomal complement of the mates of the irradiated males. Thus, in the latter case, any zygote getting both the X and the Y from the male parent, would get either the Bar Stone fragment or the double X from the mother. The former combination produces a sterile male, and the latter is a super-female.

In order to attain the maximum yield of offspring per female and to conserve labor, a culture technique suggested by Spencer was employed. In this system, 100 to 200 females, plus the appropriate number of males, were placed in a quarter pint milk bottle with the standard cornmeal-molasses medium. They were allowed to remain for two days, and then were transferred to a fresh bottle. Two days later, a small wad of Kleenex (two sheets), saturated with a thick solution of fresh bakers yeast, was placed on the food in the bottom of the bottle. Females were kept and used until they died, only making certain that the initial number of flies per bottle was maintained, by combining bottles when necessary.

To date, approximately 150,000 potential transformed males have been tested, with no case of the desired type of fertility yet appearing. There were, however, occasional cases of spurious fertility due either to contamination or to lack of virginity somewhere in the sequence of matings, where it was essential to have virginity.
Tokunaga, Chiyoko. Notes on the sex chromosome constitution of oogonial cells in gynanders.

During the course of development a female zygote of D. melanogaster heterozygous for a ring X-chromosome may lose the ring chromosome from a cleavage nucleus, thus developing into a gynander. If the differentiation of germ cells should be the result not of their own genotype but of that of the somatic tissues of the gonads it is conceivable that XO oogonia could occur in the ovaries of gynanders.

175 gynanders were obtained among the progeny of a cross between y ac sn³ females and the ring X-chromosome carrying Xc² f car males. In order to increase the frequency of gynanders among the progeny, y ac sn³ virgin females were aged for eleven days at 17°C before mating. Of these 175 gynanders, one had underdeveloped gonads, eleven had one testis and one ovary, 43 had a pair of testes, and 120 had a pair of ovaries. The oogonia of 83 gynanders with 2 ovaries each were examined for their X-chromosomal constitution by means of the smear method, after fixation with aceto-lacto-orcein. 33 of these individuals clearly showed two X chromosomes, seven more seemed to have two X chromosomes, and the remaining 43 specimens did not show good mitotic metaphase figures. Oogonial mitotic figures of eight gynanders with one ovary and one testis were also investigated. One showed two X chromosomes clearly, while seven smears were unsuitable for cytological analysis.

Thus no mitotic figures were found which showed oogonial cells of XO constitution among the ovaries studied.


The crosses recorded here were designed to detect possible cases of abnormal chromosome segregation in interspecific hybrids of D. pseudoobscura and D. persimilis.

The rationale behind this test is to check on the possibility that during the divergence of these two species, there occurred in one or the other of the two a case of meiotic drive which became fixed and now would be undetectable by ordinary tests.

GENERAL PROCEDURE: Successive backcrosses with progeny counts each generation were carried out more or less extensively for a large number of cross types. For each type of cross, tests for possible abnormal chromosome segregation in the female and male were carried out in separate lines of backcrosses. For convenience, the following terminology was used: female Backcross -- female progeny with stock male male Backcross -- male progeny with stock female.

Mass matings were used in the original hybrid crosses. Pair matings were set up for each successive female Backcross. From the progeny of each generation of female Backcrosses, mass male Backcrosses were set up until successful crosses were obtained. Then a separate line of pair male Backcrosses were set up.

Where recessive markers were followed, + progeny were backcrossed to the original stock carrying the mutant. Where dominant markers were utilized, progeny showed the character were mated to the original wild type stock.

Although crossing-over was partially suppressed by normal or, for chromosome IV, special stock inversion, unequal recovery in the progeny of females might indicate unequal recovery of only the chromosomal region adjacent to the loci involved.

CHROMOSOME II:

Original Matings:

Iv. g₁²/g₁² D. pseudoobscura females x wild type #12 D. persimilis males.

Iw. g₁²/g₁² D. pseudoobscura females x wild type #21 D. persimilis males.

Iy. up Ba g₁²/g₁² D. pseudoobscura females x wild type #21 D. persimilis males.

(Dominant marker followed)

Results:

Iv. 1st female Backcross -- Equal recovery -- g₁²/g₁²+ 283/281

Iw. Equal recovery. Combined data through 3rd female Backcross -- g₁²/g₁²+ 456/463

Iy. From 18 successful 1st female Backcross matings, 612 Ba and 926 Ba+ progeny were obtained. However, practically all of the excess Ba progeny came from only ten matings. Unfortunately, the female Backcross matings from these ten were singularly unsuccessful; the 2nd female Backcross yield were small and no successful 3rd female Backcrosses were obtained.
Matings from the remaining eight 1st female Backcross matings were more successful. These and subsequent backcrosses gave equal recovery in both sexes. Combined data for the 2nd through 6th female Backcrosses:

\[
\begin{array}{ccc}
\text{Ba} & \text{Ba}^+ \\
\text{female} & 1160 & 829 \\
\text{male} & 1225 & 931 \\
\end{array}
\]

CHROMOSOME III:

Original Matings:
If. or (ST) D. pseudoobscura females x wild type #12 D. persimilis males
Ih. or (ST) D. pseudoobscura females x wild type #15 D. persimilis males
Ij. or (ST) D. pseudoobscura females x wild type #21 D. persimilis males

Results:
1st female Backcross: In all three there appeared to be an excess of or\(^*\) over or progeny:

<table>
<thead>
<tr>
<th></th>
<th>or</th>
<th>or*</th>
</tr>
</thead>
<tbody>
<tr>
<td>If</td>
<td>1181</td>
<td>1453</td>
</tr>
<tr>
<td>Ih</td>
<td>508</td>
<td>656</td>
</tr>
<tr>
<td>Ij</td>
<td>463</td>
<td>686</td>
</tr>
</tbody>
</table>

Subsequent Backcrosses:
If. Equal recovery - 2nd B.C. 714 799
3rd B.C. 2374 2413

Ih. Equal recovery 2nd Backcross - 167 164

No further matings.

Ij. While results of the 2nd female Backcross indicated possible preferential recovery of or\(^*\), subsequent backcross generations gave approximately equal recovery:

<table>
<thead>
<tr>
<th></th>
<th>or</th>
<th>or*</th>
</tr>
</thead>
<tbody>
<tr>
<td>2nd B.C.</td>
<td>419</td>
<td>543</td>
</tr>
<tr>
<td>3rd B.C.</td>
<td>618</td>
<td>691</td>
</tr>
<tr>
<td>4th B.C.</td>
<td>705</td>
<td>769</td>
</tr>
</tbody>
</table>

Backcrosses
If and Ij. Numerous successful male Backcrosses were obtained from 1st female Backcross progeny. In these and subsequent male Backcross generations there was approximately equal recovery of or and or\(^*\).

CHROMOSOME IV:

Original Matings:
Im. in hk j C( inv. IV)/1 D. pseudoobscura females x wild type #15 D. pers. males
Io. in hk j C( inv. IV)/1 D. pseudoobscura females x wild type #21 D. pers. males

Results:
Cy or Cy*

Backcrosses: Im. Equal recovery 1st female Backcross:

\[
\begin{array}{cc}
\text{Cy} & 228 \\
\text{Cy}^* & 245 \\
\end{array}
\]

Combined data: 3214 3316

male Backcrosses: Several successful backcrosses were obtained from the 3rd female Backcross progeny of Io. These and the 2nd male Backcrosses gave approximately equal numbers of Cy and Cy\(^*\) progeny. Combined data: 419 \(\frac{364}{364}\)

X CHROMOSOME - RIGHT ARM:

Original Matings:
Wild type AH D. pseudoobscura males x se D. persimilis females

Result: 1st female Backcross - only one successful mating.

<table>
<thead>
<tr>
<th></th>
<th>se</th>
<th>se*</th>
</tr>
</thead>
<tbody>
<tr>
<td>61</td>
<td>59</td>
<td></td>
</tr>
</tbody>
</table>

No successful 2nd female Backcross matings.

X CHROMOSOME - LEFT ARM:

Original Matings:
Iff. y sn v co sh D. pseud. females x wild type #12 D. persimilis males
IIe. y xn v co sh D. pseud. females x wild type #21 D. persimilis males
Ii. Pt y sn v mbl D. pseud. females x wild type #21 D. persimilis males
(F1 females mated to y sn v co sh D. pseudoobscura males)
Is. Pt y sn v mbl D. pseud. females x wild type #19 D. persimilis males

Dominant marker followed. (Pt males almost always y sn v)
IIe. 1st Backcross only - Excess of $y^+$ over $y$ progeny among both females and males.

<table>
<thead>
<tr>
<th>females</th>
<th>males</th>
<th>females</th>
<th>males</th>
</tr>
</thead>
<tbody>
<tr>
<td>300</td>
<td>216</td>
<td>91</td>
<td>124</td>
</tr>
<tr>
<td>516</td>
<td>215</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Also a somewhat higher recovery of $sh^+$ over $sh$ among the progeny.

<table>
<thead>
<tr>
<th>sh females</th>
<th>males</th>
<th>sh females</th>
<th>males</th>
</tr>
</thead>
<tbody>
<tr>
<td>250</td>
<td>161</td>
<td>141</td>
<td>179</td>
</tr>
<tr>
<td>411</td>
<td>320</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

It. Excess of $+$ over $y$ in $v$ progeny.

<table>
<thead>
<tr>
<th>females</th>
<th>males</th>
</tr>
</thead>
<tbody>
<tr>
<td>91</td>
<td>55</td>
</tr>
<tr>
<td>147</td>
<td>35</td>
</tr>
</tbody>
</table>

Conclusion: There appears to be no evidence for any instance of meiotic drive incorporated into either of the two species.

Continuing our work on the oxygen effect in Drosophila zygotes (see Ulrich & Würgler, DIS-33) a more detailed analysis of the influence of environmental gas conditions before and during irradiation has been made. In earlier experiments we have exposed eggs (which were 10 - 20 minutes old) to an air or a nitrogen current 1 minute before and during the 3 minutes lasting irradiation. In the experiments reported here the influence of prolonged pretreatment and change of gas atmosphere during irradiation has been tested; the age of eggs at the beginning of irradiation (3 minutes; 1000 r; 50 kV; 10 mA) was again 10 - 20 minutes. A wild stock (Berlin wild) was used.
a) nitrogen treatment without irradiation

Unirradiated controls in air showed an embryonic mortality of 6.4%. 1 - 7 minutes nitrogen treatment increased the mortality linear with the duration to 8.7%. Beyond 8 minutes mortality increased more steeply. After 20 minutes in nitrogen 55.7% of the embryos died. Straight lines calculated by linear regression for these data were used to correct the following results (two different lines being calculated from (1) the interval 1 - 7 minutes and (2) the interval 8 - 20 minutes. In irradiation experiments anoxia was never extended over more than 8 minutes.

b) irradiation with nitrogen pretreatment

With 1 minute pretreatment and irradiation in nitrogen we found an embryonic mortality of 54.9% (n ~ 739). Longer (up to 5 minutes) and shorter (1/2 minute) pretreatment did not significantly change the results (summarized pretreatment experiments: 55.1%). The same was true even if there was no pretreatment at all: 54.7% mortality (n = 2488).

If the irradiation in air was preceded by exposure to nitrogen for 5 minutes, the mortality (84.1%; n = 1419) was not different from that without pretreatment (85.6%; n = 2426).

c) change of gas environment during irradiation

The rapid exchange of gas between egg and environment demonstrated under b) allowed for a change of the gas conditions within the egg during the 3 minutes lasting irradiation. In a first series X-rays were applied without an interval 1 minute (either the first, second, or third) in nitrogen and 2 minutes in air; in a second series 2 minutes in nitrogen and 1 minute in air. As compared to 3 minutes irradiation in air practically the same decrease of embryonic mortality was found in the 3 experiments of series 1. The same was true in series 2, where the decrease was more pronounced. Therefore the results of each series are summarized. Thus an embryonic mortality of 77.2% (1 minute N₂) and 69.2% (2 minutes N₂) was found. This agrees well with the expectation of 77.2% and 68.0%. This expectation is derived from the hypothesis of independent realization of radiation-damage caused under aerobic and anaerobic conditions. The calculation of the exact data were based on the findings that dose-action curves in air (ULRICH 1960) and nitrogen (WÜRGLER 1960) can be approximated by curves of the form y = 1 - e⁻ᵏD (y = mortality; D = dose).

d) conclusions

1.) The replacement of gas inside D.m. zygotes by N₂ is achieved within a few seconds.

2.) A nitrogen pretreatment up to 5 minutes has no influence on the embryonic mortality induced by irradiation of 10-20 minutes D.m. eggs.

3.) If during part of the irradiation time the air atmosphere was replaced by nitrogen, the embryonic mortality decreased proportionately to the length of the N₂-fraction. This agrees with the assumption of independent realization of the radiation-damage caused under aerobic and anaerobic conditions.

(Work supported by Schweiz. Nationalfonds zur Förderung der wissenschaftlichen Forschung.)

Zimmering, S. and H. J. Muller. Studies on the action of the dominant female-lethal Fl and of a seemingly less extreme allele, Fl⁸. Tests were made to determine whether the female lethal Fl (Muller and Zimmering, 1960, Genetics 45: 1001-1002) still acts as a complete lethal when present in pseudo-males having two Fl-containing X-chromosomes and two third chromosomes containing tra ("transformer of sex," Sturtevant, 1945, Genetics 30: 297). It was found that the sex transformation failed to save the lives of these flies. Similarly, in flies heterozygous for Fl, the viability of pseudo-males (XX, but homozygous for tra) was as much reduced by the dominant action of Fl as was the viability of their non-transformed sisters that had the same X-chromosome composition but were heterozygous for tra.

Tests of the genetic factors determining the dominant female-lethal effect of Fl in crosses of our y v stock ("bl20") have shown that all the major chromosomes (X, II, and III) of this stock play an important and synergistic role in producing the effect. In daughters of females heterozygous for some or all of these three chromosomes a little or no dominant lethal effect was produced except when all three of the chromosomes were present together in the given mothers. The effect was a maternal
one, tending to kill the heterozygous F1 (but not the non-F1) daughters of all classes, provided that the mothers contained these intensifiers in all three chromosomes, in at least single dose. The intensifiers themselves were partially dominant, in that mothers homozygous for them gave a higher lethality of daughters heterozygous for F1 than did mothers heterozygous for them. When virgin females carrying the intensifiers are kept at a comparatively high temperature (35°C) for 36 or more hours prior to egg laying, the mortality of their heterozygous F1 daughters, derived from eggs laid at 25°C within the next three days, relative to that of their brothers, is considerably reduced (in the cases studied, from about 96% to 80%). Little reduction of mortality is produced when the exposure to warmth is allowed to last only 24 hours. Other cases of genes that have a maternal effect in killing daughters but not sons have been reported by Redfield (1924, 1926), Gowen and Nelson (1942), Gowen (1949), and Bell (1954), but in these cases there was no finding of a primary female-lethal gene, corresponding to F1, that had to be present in the female that was herself subject to the lethal action.

In the experiment on pseudo-males the stock that had been used to provide the tra gene (our stock "J22") had had females with attached X's and males whose single X-chromosome contained w^a. The crosses of this stock unexpectedly showed that the w^a-containing chromosome also carried a allele of F1. We are, for reasons to be given below, denoting this as F1^a (a symbol superseding our earlier, unpublished designation, F1^2). It was found that compound females, one of whose X-chromosomes carried F1 and the other F1^a, invariably died. However, when crosses were made of F1^a males to stocks y v ("bl20") and w ("b69"), which on crossing to F1 males had given a high mortality of daughters, i.e., a high dominance of F1, no such lethality occurred among these daughters. That is, F1^a, unlike F1, failed to act as a (partially) dominant lethal. That this difference was not sufficiently explained by autosomal modifiers was proved by experiments in which the autosomes were appropriately substituted by the aid of chromosomes having inversions and markers. Similarly, parts of the X far from F1 were ruled out. It was further found that F1^a, unlike F1, is not, when in its original setting, lethal even in the female homozygous for it, despite its lethality when "in compound" with F1. F1^a does sometimes act as a lethal to females homozygous for it, however, when taken out by crossing over from its original genetic setting, but the number and loci of the modifying genes here involved have not been worked out.

In the crosses that gave rise to homozygous F1^a females it was found that these females are invariably sterile (hence the superscript s). Their abdomens remain unenlarged, like those of homozygous fes females, while they appear normal in other outward respects. Like the lethality of F1, the sterility of F1^a is to a certain extent and under some conditions dominant, inasmuch as heterozygous F1^a females are found in some crosses to have a high frequency of sterility. Such sterility has not thus far been observed among heterozygous F1 females. Further studies are however needed to determine definitely whether there are differences in the action of F1 and F1^a when they are in exactly the same genetic setting.

Whereas F1 arose within Inversion-49, F1^a is in an X-chromosome of normal structure. It must therefore have arisen as a result of a spontaneous mutation independent of that which produced F1. Both these genes have been found by linkage tests to be slightly to the left of oc. F1^a has been located more exactly as lying between cm and ct, nearer to cm, inasmuch as only 2 out of 10 tested crossovers between cm and ct proved to have been between the loci of cm and F1^2, while the rest were between the loci of F1^2 and ct, thus placing F1^a at approximately 19.1 in the X-chromosome map.

Thus far, tests for female lethality have been made in our laboratory, by Robert Baum and by Marcia Henning, of a considerable number of our stocks in which the X-chromosome of the male had been kept confined to males by having them always crossed to females with attached X's. Thus far, no cases of female lethality have been found other than those in which the original F1 allele had been present as a result of the common origin of the F1-containing region of the given X-chromosome with that of the X-chromosome of the stock in which F1 had first been discovered. These results suggest that mutations of the type in question are comparatively rare.

(Work supported by a grant from the U.S. Public Health Service (Contract RG-5286(C3)).)
Browning, Luolin S. and Edgar Altenburg. The weighing of dehydrated Drosophila as a counting method. The counting of many thousands of individual flies is one of the major technical problems involved in experiments in which there is a low incidence of the phenomenon being studied -- for example, visible mutation rates at specific loci and pseudo-allelic crossingover. A method which eliminates this task without the use of special equipment and furnishes counts reliable within a standard error of about 3% has been developed.

Flies are grown by the "vat method" (D.I.S. #33, p. 177), from 8,000 to 20,000 offspring being obtained from several transfers of the same parents (usually 200 to 400 parents). Since the flies that hatch first in a vat may be twice as large as those hatching later, due largely to better cultural conditions, it was found necessary to establish a rigid routine for emptying, examining, and handling the flies. Three examinations of the etherized offspring are made per vat -- on the 14th, 17th and 20th days -- after which the vat is discarded. After each examination, the offspring are immediately killed with ether, placed in an open milk bottle in an incubator at 55°C and kept for at least 24 hours. All the flies from a given vat are eventually added to the bottle, and after dehydrating, the bottle is transferred to a dehumidifier until a convenient time for weighing on an analytical balance. Flies kept for as long as three months in the dehumidifier showed only a slight change in weight. It was found that various mutant strains in laboratory use had different weights and sex ratios, and although this variation was not great, it was calculated by actual counts for different experiments. The table below shows the results of such a sampling, together with the weights after dehydration of each sample of counted flies coming from a single vat. (The female parent flies in this experiment carried w^a in one X chromosome and w^Bx in the other, and were also heterozygous for y and ap, as well as for the Cy and Ubx inversions in the second and third pair of autosomes; the male parents were Basc males containing sc w^a B.)

<table>
<thead>
<tr>
<th>No.</th>
<th>Weight (Grams)</th>
<th>Weight No. per Gram</th>
<th>No.</th>
<th>Weight (Grams)</th>
<th>Weight No. per Gram</th>
</tr>
</thead>
<tbody>
<tr>
<td>4,637</td>
<td>1.2920</td>
<td>3,560</td>
<td>3,903</td>
<td>0.8543</td>
<td>4,580</td>
</tr>
<tr>
<td>2,455</td>
<td>0.7533</td>
<td>3,260</td>
<td>2,184</td>
<td>0.4851</td>
<td>4,500</td>
</tr>
<tr>
<td>1,973</td>
<td>0.8127</td>
<td>2,436</td>
<td>1,563</td>
<td>0.4139</td>
<td>3,780</td>
</tr>
<tr>
<td>2,797</td>
<td>1.0342</td>
<td>2,700</td>
<td>1,563</td>
<td>0.4139</td>
<td>3,780</td>
</tr>
<tr>
<td>2,072</td>
<td>0.6431</td>
<td>3,220</td>
<td>1,724</td>
<td>0.3969</td>
<td>4,320</td>
</tr>
<tr>
<td>2,406</td>
<td>0.7513</td>
<td>3,290</td>
<td>2,091</td>
<td>0.4749</td>
<td>4,420</td>
</tr>
<tr>
<td>13,543</td>
<td>4.2324</td>
<td>3,200</td>
<td>11,465</td>
<td>2.6251</td>
<td>4,570</td>
</tr>
</tbody>
</table>

There is a range in yields among the twelve sample vats of from 2,200 to 8,500 flies, with a range in flies per gram of from 2,700 to 3,980. The average number of flies per gram for all the vats is 3,450±113, or 3,450±3.3%. Thus in 67% of all experiments involving 50,000 or more flies, the total number of flies (obtained by multiplying the weight of dehydrated flies by 3,450) would be expected to be in error by no more than 3.3%, and by no more than 6.6% in 95% of such experiments.

It is sometimes desirable to scan rapidly large numbers of flies for rare mutants in one sex but without separating them as to sex. The table shows that the number of females represented by one gram of dehydrated males and females can be reliably estimated. For example, the sex ratio based upon a count of about 25,000 flies (columns one and four in the table) was 0.54 females: 0.46 males, 13,543 females being included in a total weight of 5.3508 grams of both males and females. This indicates that one gram of both males and females contained 1,845 females, a figure very similar to that of 1,863 females obtained when the average number of males
and females contained in one gram of both males and females (3,450) based upon a count of over 50,000 flies is multiplied by the sex ratio factor of 0.54.

Mickey, G.H. Nigrosine as an aid for staining brain and salivary gland chromosomes. We have found that the addition of nigrosine (alcohol soluble -- National Aniline) to aceto-orcein stain enhances the staining of chromosomes remarkably. Larval ganglion chromosomes are stained intensely enough to be examined and photographed without the use of phase contrast microscopy. The procedures are as follows:

1) Aceto-orcein stain is prepared by dissolving one gram of orcein dye (Allied Chemical) in 100 ml of 45% acetic acid in an Erlenmeyer flask, with reflux condenser attached, and heating for one hour without boiling. The solution is cooled, filtered, and stored in refrigerator.

2) Aceto-nigrosine stain is prepared by heating 100 ml of 50% acetic acid to boiling point, adding 4 gms of alcohol soluble nigrosine and stirring constantly. It should be boiled for 3 - 5 minutes, or until it acquires a highly viscous consistency. When cooled, it should be filtered, using a water pump filter. Store in closed glass vessel in the dark at room temperature.

3) Aceto-orcein-nigrosine stain for salivary slides is made by mixing 1 ml of aceto-nigrosine and 9 ml of aceto-orcein stain. This must be filtered before each use!

4) Aceto-orcein-nigrosine stain for brain slides is made by mixing 4 ml of aceto-nigrosine and 6 ml of aceto-orcein stain. Must be filtered before each use!

The chief drawback of the stain is the tendency for the nigrosine to precipitate, which necessitates frequent filtering. A temporary squash preparation sealed with cover glass wax will improve with aging a few hours to several days, but should be made permanent if it is to be kept for a longer time. We use the dry ice technique for removing the cover glass and mount in euparal. The stain is permanent and does not fade.

The stain produces superb salivary gland slides, the fine bands on the chromosomes showing very distinctly.

Moyer, S. E., R. E. Comstock and L. H. Baker. Efficient procedures for culturing Drosophila in disposable paper containers. Arnold (Amer. Biology Teacher 19:248-251 and DIS-32 p. 166) described a method for utilizing plastic-lined paper containers for culturing Drosophila melanogaster. We have modified this basic plan to enable greater ease and speed in routine handling of large numbers of containers.

Covers for the containers are now available with a half moon clear plastic window. Ventilation holes, if needed, can be quickly punched with a "pin cushion" made from a rubber laboratory stopper and well spaced common pins.

The medium is anchored in the bottom of the container by a 4" x 4" non-sterilized 8 ply surgical gauze sponge stapled to the sides. This prevents the medium from falling on the flies while the container is inverted during anesthesia.

Anesthesia is accomplished by applying several drops of ether on a small cotton plug stapled into a 1/4 inch hole punched in the side of the container. Since this plug may be an attraction as a site for pupation, it may be desirable to locate it near the top of the container. Further diversion of the larvae from the plug may be achieved by limiting the amount of medium to slightly more than needed to cover the gauze on the bottom. In this way, larvae are attracted to the remaining gauze above the level of the medium. However, ether does not kill pupae occasionally lodged in plugs and possible physiological effects seem negligible.

Although anesthesia is slow with this system, waiting can be eliminated by initiating anesthetization well in advance of anticipated use of a particular culture. There is no danger of over-etherization to flies remaining in containers for an extended period of time prior to examination.

The chief advantage of using these paper containers is in the time and money saved. The cost of the unit and the time required for assembling it by the above procedures is considerably less than the time and cost of labor for washing and sterilizing bottles. Also, each of these containers supports a larger population for a longer period of time than does a half pint bottle. Finally, the containers can be stacked on top of each other for maximum utilization of storage space for Drosophila cultures.
von Borstel, R. C. and Margaret M. Fine. A medium suitable for hatchability and eclosion tests.

Since charcoal agar is a nuisance to mix and since it results in reduced viability of Drosophila larvae, another agent was sought that would give satisfactory contrast to the Drosophila culture medium for hatchability testing.

The obvious choice was fruit juice and it was found that frozen grape juice concentrate works well. The flies cling to the medium with a tenacity that decries the change of name from amelophila. Egg production and survival are high, and eclosion tests as well as hatchability tests are possible from the same vial.

The food formula used at Oak Ridge corresponds closely to that used at the California Institute of Technology (Lewis, DIS-34, 117, 1960). To prepare the hatchability medium, one 12-ounce can of frozen grape juice is stirred into every two liters of the Drosophila food and the mixture is then poured into vials. This food is just firm enough for egg hatchability testing, and it may be desirable to add more agar if some other food formula is used.

We presume that other fruit juice concentrates could be used as well, and appropriately dark wines could possibly be used, but local regulations are such that experimentation along these lines at Oak Ridge has been severely restricted.

PERSONAL AND LABORATORY NEWS

James Divelbiss is joining the staff of the Biology Department, Westmar College, Le Mars, Iowa in September 1961, and plans to establish a Drosophila research laboratory. Since library facilities in the area of genetics are limited he would appreciate receiving any available reprints, new or old.

Edward C. Keller, Jr., has moved from The Pennsylvania State University to The University of North Carolina at Chapel Hill in The School of Medicine, Department of Biochemistry and Nutrition.

Maxi E. Krawinkel, curator of stocks and head technician of the Purdue University Drosophila laboratory for the past five years, was killed in an automobile accident while on vacation in Michigan on January 22, 1961. She has been buried near her home in Bern, Switzerland. A memorial book collection bearing her name has been established in the Biology Library, Purdue University.

MATERIALS REQUESTED OR AVAILABLE

The inbred temperature lines of D. melanogaster derived from a wild population near State College, Pennsylvania, that are described in the stock list of University Park, Pennsylvania (DIS-34) will be maintained until further notice is given in D.I.S.

James Divelbiss is currently engaged in a pseudoallelic investigation of the brown locus. He would appreciate receiving stocks of any brown alleles except the following which he already has: bw¹, bw⁸¹, bw⁷⁵, bw⁵⁹, bwM⁵⁸, bwM⁵⁹ and bwAm. For his address see entry in Personal and Laboratory News.

ANNOUNCEMENTS

Drosophila melanogaster Stock Centers

The National Science Foundation is now supporting two stock centers for the maintenance of strains of Drosophila melanogaster. One is located at the California Institute of Technology, Pasadena, California and is under the direction of Professor E. B. Lewis while the other one is located in the Division of Chemotherapy of The Institute for Cancer Research, Philadelphia 11, Pennsylvania and is under the direction of Dr. I. I. Oster. The nucleus of both centers will consist of duplicates of the 800 basic stocks hitherto only maintained in Pasadena. These centers will serve
as a source of supply for virtually any research needs which might arise regardless of field of genetic interest, 2) provide insurance against loss of all or part of their respective collections by some unforeseen catastrophe, 3) allow the centers to make replacements of each other's stocks in the event any have broken down and 4) provide the "marker" and tester stocks and other combinations of mutations useful in research and teaching.

In addition to the 800 basic stocks, the Center in Philadelphia will maintain approximately 1600 other strains. These will include 600 stocks representing the major portion of the strains currently maintained by Professor H. J. Muller at Indiana University, 200 strains which had been maintained by Dr. J. Schultz in the Department of Genetics of The Institute for Cancer Research, and 800 strains consisting of other useful mutations, combinations of them, and multiple alleles of loci of unusual interest obtained from other laboratories or synthesized by the Center.

We would like to suggest that other workers should contribute useful stocks for inclusion in the collections. The main requisites for acceptance of such strains are that they be held in a combination not requiring selection, they represent new loci, alleles of unusual interest, improved balancers, etc., and they be free of mites. Stocks of mutants which overlap wild-type or of biochemical mutants which have no morphological effect should contain an RK1 marker mutant to serve as a check on the possibility of contamination. In order to avoid the inclusion of too many stocks of questionable usefulness in the permanent collection, consultations with the Subcommittee on Drosophila Stocks will be held from time to time concerning the advisability of adding any of these newly contributed stocks or of eliminating old ones. Those who wish to contribute stocks should send a complete description of the stock to the Center ahead of time preferably on 3 x 5 cards. This will facilitate evaluation of the stock and provide the basic information needed for the stock records.

When a stock has been improved (for example, by introducing a more efficient balancer, or a more useful combination of mutants) the old stock in general will be discarded and replaced by the new one. However, no mutant type, balancer, nor any chromosomal rearrangement will be deliberately discontinued without notice being given in the Drosophila Information Service at least one year in advance.

Requests for stocks for research purposes will be filled as promptly as possible. As heretofore, there will be no charge for this service. It is requested that the receiver return the empty plastic vials in the original mailing carton.

Suggestions for improving the stockkeeping service are welcomed and can be addressed to either Center or to the Subcommittee on Drosophila Stocks.