DROSOPHILA

Information Service

40

January 1965

Material contributed by
DROSOPHILA WORKERS

and arranged by
E. NOVITSKI

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Prepared at the
DEPARTMENT OF BIOLOGY
UNIVERSITY OF OREGON
EUGENE, OREGON

DROSOPHILA INFORMATION SERVICE

Number 40

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Ytterborn, K. Sweden, Stockholm
Yuki, S. Japan, Kobe
Zambruni, L. Italy, Milano
Zimmer, K. G. Germany, Karlsruhe

MATERIALS REQUESTED OR AVAILABLE

DuWayne C. Englert, Department of Zoology, Southern Illinois University, Carbondale, Illinois, would like to receive any reprints that are available in order to build up the departmental library.

C. A. Mourão, Departamento de Biologia Geral - Faculdade de Filosofia, Ciências e Letras, São José do Rio Prêto, São Paulo, Brasil, would be grateful to obtain reprints on speciation in Drosophila and reprints on systematics of Drosophila.

John C. Neeley, Oregon State University, would appreciate a triploid stock with free X-chromosomes from anyone with such a stock.

F. Mainx, Institut f. Allgemeine Biologie, University of Vienna, Wien IX. Schwarspanierstr. 17, would appreciate obtaining strains of Megaselia scalaris (=Aphiochaeta xanthina) from different places as well as strains of other species of Phoridae easily bred in the laboratory.

ANNOUNCEMENT

SEVENTH DROSOPHILA RESEARCH CONFERENCE: May 8 and 9, 1965

In accordance with the decision reached at the 6th (Madison, Wisconsin) Drosophila Conference, the next meeting will be held in Seattle, Washington. I have chosen May 8th and 9th as reasonable dates. However, there is still time to change, and I invite any suggestions and objections both as to date and as to the format of the meetings.

Any such suggestions may be sent to:

L. Sandler
Department of Genetics
University of Washington
Seattle, Washington

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Part IV

I. Herskowitz

344 pp., 3305 references, 1957 through 1962
$4.95 hard cover edition
McGraw Hill Book Company

AMHERST, MASSACHUSETTS: AMHERST COLLEGE

Wild Stocks

1 Oregon-R: generation 438 on 64i22, inbreeding
2 Oregon-R: mass culture, from $ #1 at generation 100
3 Oregon-R: mass culture, from $ #1 at generation 200
4 Oregon-R: mass culture, from $ #1 at generation 300
5 Oregon-R: mass culture, from $ #1 at generation 400
6 Oregon-R-C
7 Oregon-S
8 Samarkand 204: inbred for 204 generations; mass culture since 53h4
9 Samarkand 204-125: from $ #8, inbreeding, generation 125 on 64i22
10 Samarkand 204-100: mass culture, from $ #9 at generation 100

Chromosome 1

11 B
12 B Bx^3/y w f:=
13 Basc
14 Basc/f·=
15 Bx
16 cm
17 cm ct^6
18 cm ct^6 sn^2_3
19 cm ct^6 sn^3
20 cm ct^6 sn^3 oc/y f:=
21 cm ct^6 sn^3_4 sn/y f:=
22 cm ct^6 sn_4
23 cm ct^6 sn_4 oc ptg/y f:=
24 cm ct^6 sn^5 sn/y f:=
25 cm ct^6 sn^5_{34e}/y f:=
26 cm ct^6 sn_{36a}
27 cm ct^6 sn_{36a}/y f:=
28 cm ct^6 sn^3_2 sn^2 oc ptg/y f:=
29 cm sn^3
30 cm sn^3
31 cm sn^3;Ubx^{130} e^s/$T(2;3)Xa^6$
32 ct^6
33 ct^n oc/y f:=
34 ct^n oc/y f:=
35 ct^n sn^3 sn^2/y f:=
36 Ext/FM6
37 f car; Ubx^{130} e^s/T(2;3)Xa
38 f od car
39 g
40 $g_2$sd
41 g^2 pl
42 g^3_3
43 g_{50e}sd
44 g_{53d}
45 g_{53d}
46 g_w sd
47 g^3
48 lz^3_3 m/y f:=
49 lz^3_{36a} y/y f:=
50 m f
51 oc/y f:=
52 oc_2ptg/y f:=
53 pn^{39k} f B/In(1)AM
54 r^{51} At f In49 w^a sc^8/oc ptg
55 ras_2dy
56 ras^2 m/y f:=
57 rb^2
58 rux^2_2
59 rux^2_1 vs
60 sc^8
61 sd_{58d}
62 sd^{58d}
63 sn/y f:=
64 sn_2oc/y f:=
65 sn^2_3
66 sn^2_3 oc ptg^3/y f:=
67 sn^3_3
68 sn^3_3 g^{53d}
69 sn^3_4 oc/y f:=
70 sn^3_4
71 sn^3_5 oc ptg^3/y f:=
72 sn^3_{34e}oc ptg/y f:=
73 sn^3_{36a}
74 sn^{36a}/y f:=
75 un od
76 v
77 v fw^{53d}
78 v g^{53d}_2 sd
79 v wy^2 g^2
80 vs
81 vs cm
82 w
83 w/+·=
84 w_a sn^5/y f:=
85 w^a
86 w^e
87 w^e_2sn/y f:=
88 wy^2
89 wy^2_2 g_2
90 wy^2_2 g^3
91 wy^2_2 g_4
92 wy^2_2 g_{53d}
93 wy^2_2 g^{53d}
94 wy^8 g^{53d} sd
95 y ct^6 f
96 y ct^6_{53d} ras^2 f
97 y g^2 sd/+·=
98 y ras_2 f
99 y ras_{50k} f/B·Basc
100 y_2 sn^3 ras^2_4/y f:=
101 y_2 sn^3 ras_4 m/B·Basc
102 y_2 sn^3 ras^a m/ClB
103 y^2 w^a cv sn^{55a} v/+·=

Chromosome 2

104 a px or
105 b
106 b vg bw^{45a} sp^2 or^{45a}
107 Bl/Cy, bw^{45a} sp^2 or^{45a}
108 Bl/Roi, bw^{45a} sp^2 or
109 Bl L^2/Cy, sp^2

110 bw
111 cl_2
112 cl^2 px sp/T(Y;2)E
113 cn bw
114 ho
115 L^4
116 ltd
117 net_{49h} b cn bw
118 or
119 sca l(2)C/SM5, Cy 2^{lt} sp^{45a}
120 sp^2 or
121 vg_{-B}
122 vg_{-D}/Cy_2 L^4 sp^2
123 vg^{sp}_{sp} $_2$/Roi, bw^{45a}_{45a} or
124 vg_{nl}
125 vg_{no}
126 vg_{np}
127 vg^{45a}_{or}/Roi, bw^{45a} sp^2
128 vg^{45a}_{or}/Roi, bw^{45a} sp^2

Chromosome 3

129 bar-3
130 bv
131 cu
132 D/Gl
133 D^3 Sb/Payne
134 Dl^7/Ubx^{130} e^s
135 Gl Sb/LVM
136 h
137 h st
138 h th_s st p^p cu sr e^s (hes)
139 p^p
140 ri p^p/T(Y;2;3)F, st
141 rsd
142 ru h th st cu sr e^s ca (rucuca)
143 ru h st cu sr e^s ca (ruc-th)
144 ru h st p^p_s ss Su^3 -ss e^s
145 ru st ss ca (rusca)
146 se h
147 se ss
148 se ss Su^3-ss
149 sr Dl e^{61e5}/DCxF
150 ss
151 ss bx Su^2-ss
152 th
153 TM1/D^3
154 TM1/Gl
155 TM1/Ubx^{130} e^s
156 tx

Chromosome 4

157 ci_D ey^R_D
158 ci_2/ey^D
159 ey^2
160 spa^{pol}

Multiple Chromosomes

161 ras^4_{bw} $^{45a}_{45a}$ m/y f:=;Bl^{45a}_{sp}/Cy, $^2_{or}$
162 +/y f:=;vg^{51h25};se^{50k}
163 +/y f:=;bw;e;spa^{pol}
164 bw;e
165 bw;e;spa^{pol}
166 cn bw;e
167 net^{sp}_{51h25} $_2$ or^{45a}_{50k}; ru bv
168 vg_{51h25};se_{50k} ^{60h}
169 vg_{51h25};se_{50k} e^{pol}
170 vg ;se ;spa^{pol}
171 Cy, sp^2/Pm, dp b;DCxF, ru_2 h ca/Sb sr
172 Cy sp^2/Pm, dp b; H/Sb sr
173 Ubx^{130} e^s/T(2;3)Xa

Deficiencies

174 Df(1)g^1_{-B}, f B/In(1)AM
* Df(2)vg_{-D} in stock 122
* Df(2)vg in stock 123

Inversions

* In(1)AM in stocks 54, 174
* Ins(1)Basc in stocks 13, 14, 99, 101
* In(1)ClB in stock 102
* In(1)FM6 in stocks 36, 179, 180, 181, 182, 183, 184, 185, 186
175 $In(1)sc^{260-22}_{J1}$, sc^{260-22}
176 $In(1)sc^{S1}$;Del^{24}_8
* Ins sc^{V1} 49 sc^8 in stock 60
* In(2)bw^{V1} (Pm) in stocks 171, 172
* Ins(2)Cy in stocks 107, 109, 119, 122, 161, 171, 172, 177
177 $In(2)S^{960}$/Cy, S^e E-S
* Ins(2L)t, (2R)Cy in stocks 108, 123, 127, 128
* Ins(3)DCxF in stocks 149, 171
* In(3)Mo, Sb sr in stocks 171, 172
* Ins(3)Payne(LVM) in stocks 133, 135
* Ins(3)TM1, Mé ri sbd^1 in stocks 153, 154, 155
* Ins(3)Ubx^{130} in stocks 31, 37, 134, 155, 173

Translocations

* T(y;")E in stock 112
* T(Y;2;3)F in stock 140
178 T(2;3)gl^{63d29}
* T(2;3)Xa in stocks 31, 37, 173

Triploids

179 cm ct^{6} sn^{3}/FM6, y^{31d} sc^{8} dm B & FM6
180 cm ct^{6} sn^{4}/FM6, y^{31d} sc^{8} dm B & FM6
181 ct^{n} sn^{3}/FM6, y^{31d} sc^{8} dm B & FM6
182 g^{53d} sd/FM6, y^{31d} sc^{8} dm B & FM6
183 v m/FM6, y^{31d} sc^{8} dm B & FM6
184 vs cm/FM6, y^{31d} sc^{8} dm B & FM6
185 wy^{2} g^{2}/FM6, y^{31d} sc^{8} dm B & FM6
186 wy^{2} g^{53d} sd/FM6, y^{31d} sc^{8} dm B & FM6

URBANA, ILLINOIS: UNIVERSITY OF ILLINOIS
Behavior Genetics Laboratory, Department of Psychology

Forward Selection

1. Negative geotaxis - over 140 generations of selection for performance in mass screening maze.
2. Positive geotaxis - over 140 generations of selection for performance in mass screening maze.

Reverse Selection

3. Negative reverse - over 90 generations of reverse selection for performance in mass screening maze, beginning at Generation 52 of forward selection for negative geotaxis.
4. Positive reverse - over 90 generations of reverse selection for performance in mass screening, beginning at Generation 52 of forward selection for positive geotaxis.

Inbred (Brother-Sister)

5. Negative geotaxis - two separate lines with over 30 generations of inbreeding beginning at Generation 110 of forward selection for negative geotaxis.
6. Positive geotaxis - three separate lines with over 30 generations of inbreeding beginning at Generation 110 of forward selection for positive geotaxis.

CARBONDALE, ILLINOIS: SOUTHERN ILLINOIS UNIVERSITY
Department of Zoology

No change from Stock List in DIS 38:13 except for the following correction:

Stock No. 10 should read b vg bw instead of B

MADISON, WISCONSIN: UNIVERSITY OF WISCONSIN
Department of Zoology

Wild Stocks

1 Canton-S
2 Oregon-R

Chromosome 1 (X)

11 B
12 g^2
13 pn^8
14 sc^8 B
15 sc^{S1} Y/y sc & y^a f:=/sc^8·Y
16 sc^{S1} B InS w^a sc^8("Basc")
17 w
18 w_{c2} m f
19 X y^- v bb^-
20 y ac sc pn & y f:=
21 y w^a
22 y cv m f car & y f:=
23 y cv v f car
24 y·Dp(y^+ sc^{V1})& y f:=
25 y_+ w sn^3
26 y sc^{V1}·Y^S/y In49 v·Y^L & y f:=/ y sc^{V1}·Y^S
27 y_2 sc^{S1} B f In49 $_a$v &$_8$y f:=
28 y_{S1} sc^8 B InS w^a sc^8("y Basc").
29 y sc^8 sn^3 w_{31d}
30 y l^{451}/FM6, y sc^8 dm B
31 y_S w^a/Yw
32 Y^S·X In EN ptg oc sn^5·Y^L & sc ct^n ptg car·sn^{X2} In49 y(no free Y)
33 ("Tester-1")y ac pn w rb wy^2 g^2 & y f:=;sc^{19i}/Cy
34 ("Tester-2")y^2 w^a cm wy^2 g^2 car & y f:=;sc^{19i}/Cy
35 ("Tester-3")y rb cm ras^2 g^2 & y f:=; sc^{19i}/Cy

Sterilizer ("sz") Stocks (36-45)

36 ("sz+")Y^{Lc}/X·Y^S
37 ("sz w")Y^{Lc}/w·Y^S
38 ("sz bw")Y^{Lc}/X·Y^S;bw
39 ("sz e")Y^{Lc}/X·Y^S & y v f·=;e
40 ("sz bw e")Y^{Lc}/X·Y^S;bw;e
41 ("sz c")Y^{Lc}/X·Y^S & y v_Sf·=;c
42 ("sz lz f")Y^{Lc}/lz^3 f·Y^S & y v f·=
43 ("sz lz f")Y^{Lc}/lz^3 m f·Y^S & y v f·=
44 ("sz m f")Y^{Lc}/m f·Y^S & y v f·=
45 ("sz y w")Y^{Lc}/y w·Y^S & y ct^6 f·=

Chromosome 2

61 b nub pr
62 bw
63 cn
64 cn bw
65 dp_{cm2}
66 dp_o cl
67 dp_T
68 dp_{tx} Sp cn bw sp/s^2(ls^+_v) Cy, InL cn bw sp
69 dp_{tx} b/SM5, al^2 Cy lt_2 sp^2
70 dp^{tx} Sp pr cn bw sp/S ^{v1} ls Cy, InL pr cn bw sp
71 dp_{v2}/SM5, al^2 Cy lt^v sp^2
72 dp^{tx}
73 S M(2)S7 bw^D/dp^{txI} Cy, InsO pr cn^2 sp
74 vg
75 vg bw

Chromosome 3

81 e
82 st
83 tra/T(2;3)Mé

Chromosome 4

91 Cat/ci^D
92 ci gvl ey^R sv^n
93 spa^{pol}

Multiple Chromosomes

101 bw_{S1};e
102 sc^{S1} B InS w^a sc^8;SM1, al^2 Cy sp^2/ dp b Pm^1 ds^{33k}, c Sb/Ubx^{130} e^s
103 X^+/y f:=;ca K-pn

MADISON, WISCONSIN: UNIVERSITY OF WISCONSIN
Departments of Genetics and Medical Genetics

Wild Stocks

a1 Canton-S
a2 Crimea
a3 Oregon-R (Chicago)
a4 Oregon-R (Madison)
a5 Oregon-$R\text{-}I_{EL2}$
a6 Oregon-$R\text{-}I^{EL2}$
a7 Samarkand
a8 Swedish-B
a9 Urbana

Chromosome 1 (X)

b1 B
b2 br w^{e} ec rb t^{4}/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
b3 car
b4 cm
b5 cv f
b6 ec
b7 f
b8 ma-l
b9 pa/FM4, y^{31d} sc^{8} dm B
b10 pn^{2}
b11 sc cv v dwx/FM6, y^{31d} sc^{8} dm B
b12 sc cv v f B/y f:=
b13 sc eg cv ct^{6} v g^{2} f/FM3, y^{31d} sc^{8} dm B l
b14 sc^{S1} B InS w^{a} sc^{8}(Basc)
b15 sc^{S1} B InS w^{a} sc^{8}/y f:= (Basc/y f:=)
b16 sn^{3}
b17 t^{2} v f
b18 v
b19 w_{a}
b20 w_{a2}(Inbred Sydney)
b21 w^{co}
b22 w_{col}(Inbred Sydney)
b23 w^{col} (Inbred Sydney)
b24 w^{e}
b25 w_{h}(Inbred Sydney)
b26 w^{sat} (Inbred Sydney)
b27 w f
b28 y
b29 y cv_{S1} y f car/Y
b30 y sc^{S1}_{6} car odsy f g^{2} dy y ras^{2} sn^{3} ct^{8}_{J1} cm rb ec w pn l sc^{+}_{M1}/lJ1 $sc^{+}_{J1(+)}$ In49 ptg oc B^{+}/lJ1 $^{+}$·Y
b31 y^{2} cv_{S1} y f
b32 y^{2} sc^{S1} B InS w^{a} sc^{8}(Bascy = y Basc)

Chromosome 2

c1 al dp b pr c px sp(apl)
c2 al dp b^{3} pr cn vg $c_{2}a$ px bw mr sp/S^{2} Cy lt^{+} pr^{+} Bl cn^{2} L^{4} sp^{2}(twelvepl)
c3 b
c4 b cn bw
c5 b pr vg
c6 c
c7 cn
c8 cn bw_{D}
c9 cn bw^{np}
c10 cn vg^{np} bw
c11 Df(2)MB/SM1, al^{2} Cy sp^{2}
c12 dp
c13 In(2LR)102, ds^{w} sp/SM1, al^{2} Cy sp^{2}
c14 mi/Pm^{2}

Chromosome 3

d1 bx 34^{e}
d2 cd
d3 e_{4}
d4 e wo ro
d5 Gl Sb/Ins LVM
d6 h
d7 Ly Dfd kar ry^{26}/In(3)MRS, M34 ry^{2} Sb
d8 ru_{1} h th st cu sr e^{s} ca(rucuca)
d9 ry^{1}
d10 ry^{2}
d11 se
d12 ss
d13 st
d14 Ubx^{130}/D-138

Chromosome 4

e1 bt^{D}/ci^{D}
e2 Ce/spa^{Cat}_{R}
e3 ci ey
e4 ey^{Cat}/ci^{D}
e5 spa^{Cat}/ci^{D}

Multichromosomal

f1 cv;In(3)MRS, M34 ry^2 Sb/Ubx^{130}(1;3)
f2 sc cv f;In(3)MRS, M34 ry^2 Sb/Ubx^{130}(1;3)
f3 v;tra/C(3)X(1;3)
f4 y f:=;ca K-pn(1;3)
f5 y f:=;ru h th st cu sr e^s ca(1;3)
f6 sc^{S1} InS_1 w^a sc^8;Cy/Pm, ds^{33k}; Sb/Ubx^{130}(1;2;3)
f7 v;cn;st(1;2;3)
f8 y;bw;c;ey(1;2;3;4)
f9 y f:=;bw;e;ci ey^R(1;2;3;4)
f10 bw;st(2;3)
f11 bw^D;st(2;3)
f12 cn bw;e(2;3)
f13 Cy, sp^2/Pm, dp_2b;H/Sb sr(2;3)
f14 SM1, al^2 Cy sp^2/Pm, ds^{33k};Ubx^{130}/ Sb(2;3)
f15 bw;e;ci ey^R(2;3;4)

Attached-XY

g1 $Y^SX{\cdot}Y^L$, In(1)EN, Y^S B y·Y^L/pn

g2 $Y^SX{\cdot}Y^L$, In(1)EN, Y^S B y·Y^L/ y^2 su-w^a w^a bb

X with Y arm attached

h1 $X^+{\cdot}Y^S/Y^{LC}$;bw
h2 f·Y^S/Y^L & sc v f
h3 y ct^6 f·Y^L/Y^S and y f:=

Altered Y

i1 sc·Y·B(B^SY^L·bb$^+Y^S$y$^+$)/y v & y^2 su-w^a w^a bb

Attached 3

j1 A(3L)3, ri;A(3R)3, sr

Translocations

k1 T(1;3)9, cu kar ry^{26} l^{26} Sb Ubx/+ & y f:=
k2 T(1;3)9, cu kar ry^{26} l^{26} Sb Ubx/ru h th st cu sr e^s ca & y f:=;ru h th st cu sr e^s ca

Special Lethals (Amino Acid Mutants) See Novitski, DIS 37:51

x1	Alaful-1	x6	Glufultyrless-1	x11	Tyrproless-1
x2	Gluful-1	x7	Glufultyrless-2	x12	Tyrproless-2
x3	Gluful-3	x8	Glufultyrless-3	x13	Tyrproless-3
x4	Gluful-4	x9	Tyrless-1	x14	Glufultyroproless-1
x5	Glufulproless-1	x10	Tyrless-2	x15	Phenylfultyrless-1

NEW HAVEN, CONNECTICUT: YALE UNIVERSITY
Department of Biology

The stock list remains essentially the same as that appearing in DIS 38:10, with the following changes:

Stock Additions

Chromosome 1

31a g^2 pl/FM3, y^{31d} sc^8 dm B l

Translocation

160 T(Y;2)C/pr cn

Corrections

145 Df(1)$w^{55i3.2}$ spl/In(1)sc^{S1} dl-49, v f B
147 Df(1)w^{59k13} spl(sn^3)/dp(1)w^V, cb

Removed from List

61 y^2 w^a m_4f
144 Df(1)s^{m4L}, $y_{rst}{}^{3R}$/y, Dp(1;3)49^{a7}
148 Df(1)w^{59k13} spl/In(1)sc^{S1} dl-49, v f B

CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGO
Department of Zoology

Note: Only stocks not commonly carried in other laboratories are listed.

Wild type

1 Chicago wild-type

Chromosome 1

2 Bx^{3} ma-l
3 Bx^{46}
4 fu^{57a}/FM-1
5 l(1)J1, sc^{J1}/Del(1)24
6 lix^{5}
7 ln^{8} pn
8 pn g^{2}
9 pn^{2} rb
10 sc^{10-1} pn
11 sc/y Hw
12 sc z^{59d15}
13 sc z^{1}
14 sc z^{m}
15 w^{e} bb^{1}/y f:=
16 y ac br pn w^{e} spl
17 y ac z ec ct
18 y z^{a}

Chromosome 2

19 bw^{D}
20 bw^{75}
21 bw^{81}
22 bw^{5-}/Cy cn^{2} L^{4} sp^{2}
23 In(2R)bw^{v34}, Cy/al dp b Bl c px sp
24 In(2LR)lt^{m3}
25 T(2;3)lt^{m29}
26 pr ltd

Chromosome 3

27 ru h th st cu sr e^{s} ca

Chromosome 4

28 spa^{Cat}/ci^{D}

Inversion-X

29 In(1)AM/M(1)o f
30 In(1)EN, y/y f:=
31 In(1)rst^{3}, y w^{4} rst^{3} car bb
32 In(1)sc^{4}, y sc^{4} f v cv w^{a}/y w
33 Ins(1)sc^{4} dl-49 sc^{8}, y sc^{4} dl-49 w^{a} sc^{8}/y^{4} f:=
34 Ins(1)sc^{7} S, y sc^{4} B InS/sc^{8}·Y/y f:=
35 In(1)sc^{8}, sc^{8}
36 In(1)sc^{260-14}, sc^{8}
37 In(1)sc^{S1} sc^{8}, y sc^{S1} car m w^{a} sc^{8}/ In(1)dl-49, y w lz^{s}
38 In p(1)sc^{V1} v/Ins(1)sc^{8} dl-49, y^{31d} v f B sc^{8}
39 In(1)w^{m4}, w^{m4}
40 In(1)y, y

Deficiencies - Duplications - X Chromosomes

41 w^{-59k13} spl/Dp(1;3)w^{Vco}
42 Df(1)w^{258-45}, y w^{-} spl dm; Dp(1;3)w^{Vco}/ y w f
43 Df(1)w^{358-45}, y w^{-}; Dp(1;3)w^{Vco}/y w f
44 Dp(1;2R)$w^{51b?}$; y w rst^{3}/y w f
45 Dp(1)$w^{vc6094b}$/Y^{S} w y·Y^{L} y^{+}/y w/sc^{8}·Y
46 Dp(1;3)$w^{m264-58a}$/+; y w f Y^{L}·Y^{S}/y w/ sc^{8}·Y
47 Dp(1;3)$w^{m264-58a}$, Su-V/Su-V; Y^{S} w y·Y^{L} y^{+} /y w
48 Dp(1;3)$w^{m264-58a}$, Su-V^{+}/Su-V^{+};Y^{S} w y·Y^{L} y^{+}/y w

Reversed Acrocentrogenic

49 B^{S}·sc^{8}/Ins(1)sc^{7} AM

Ring-X

50 X^{c1}, y/sc^{8}·Y/y v f car
51 X^{c1}, y v/y w f:=

Reversed Acrocentric

52 RA, y ac sc pn -- In(1)sc^{8}/In(1)sc^{8} (C.O.J-3) y ac sc w^{a}/sc^{8}·Y, y

X with Y fragments Attached

53 FR-1, Y^{S} y cv v f/y f:=
54 y Hw·Y^{S} y^{+}/Y^{cL}/y w^{a}:=
55 FR-1, Y^{S} y cv v f/sc^{8}·Y/y v f car

Attached X-Y; no free Y

57 y^2 su-w_1^a w_S^a $Y^L \cdot Y^S$/y/O
58 y w f $Y^L \cdot Y^S$/y w/O
59 y w spl $Y^L \cdot Y^S$/y w/O

Altered Complete Y's

60 $B^S Y$/y f/y^2 su-w^a w^a
61 sc^8 Y, y^{54e} ac^{54e}/y v; bw
62 Y w^+ Co/y_1 w^a
63 $sc^8 \cdot Y$: bw^{54k}/y v; bw

Y^S Fragments

64 Y_S^{cS}: bw^+ bb^+/g^2 $B \cdot Y^L$/y v bb; bw
65 Y_S/g^2 $B \cdot Y^L/y$ f:=
66 Y_S: y^+ bb^+-5/$B \cdot Y^L$/y w
67 Y_S: y^+ bb^+-6/g^2 $B \cdot Y^L$/y v bb; bw
68 Y_S: y^+ bb^+-7/g^2 $B \cdot Y^L$/y w
69 Y_S: y^+ bb^+-8/y_1 g^2 $B \cdot Y^L$/y v bb; bw
70 $Y_S \cdot Y^S$/sc $sc^8 \cdot Y^L$/y f:=
71 $Y^S \cdot Y^S$#2/y v f $\cdot Y^L$/y_1 f:=
72 $sc^{VI} \cdot Y^S$/y v f bb$\cdot Y^L$/y f:=

Y^L Fragments

73 $sc^{S1} \cdot Y^L$/y ac w^a ct^6 f$\cdot Y^S$/y f:=
74 $sc^{S1} \cdot Y^L$#2/y ct^6 f$\cdot Y^S$/y w
75 Y^{cL}/y ct^6 f$\cdot Y^S$/y w^a:=
76 Y^L-13/y ct^6 f$\cdot Y^S$/y v bb; bw
77 Y^{cL}-15/y f$\cdot Y^S$/y w

Multichromosomal

78 In(3LR)Ubx^{130}, $_2Ubx^{130}$ e^s/Xa
79 SM-1, al Cy sp^2/In(2LR)102 ds^w sp^2; $_2$Ins(3LP,3RC)Sb e^s/Ubx e^s
80 sp^2; st_S C_3 G ca/TM-1, Mé ri
81 Su-V; Y^S w^+ y$\cdot Y^L$ y^+/Y/y w
82 y/$sc^8 \cdot Y$; ru h th st p^p cu sr e^s
83 w; Cy/Pm; CxD/Sb, In(3R)Mo
84 y; In53A, al^2 Cy^+ sp^2; Ubx^{130} e^s/Xa
85 y w/Y^S f y$\cdot Y^L$ y^+; sv^n
86 Y^S: y^+ bb^+-5/+/B$\cdot Y^L$; Cy/Pm; CxD/Sb, In(3R)Mo
87 Y^S: y^+ bb^+-7/g^2 B$\cdot Y^L$/+; Cy/Pm; CxD/Sb, In(3R)Mo

ROCHESTER, NEW YORK: UNIVERSITY OF ROCHESTER
Department of Biology

Wild Stocks

120 Canton stocks
Oregon-R (3 inbred strains)

Chromosome 1

ct^K
y^2 w^a v
y ct f . 797
In(1)bb^-, y sl^2 bb^-/AM
In(1)m^K, y^2 w^a
In(1)sc^8, In(1)m^K
In(1)w^{m4}
In(1)EN, y/y f:=
In(1)rst^3, y rst car bb
In(1)49, In(1)B^{M1}, y sc v cu-X/FM_4

Chromosome 2

L^K
Pm^K/Cy L^4
Ps Sco/Cy L^4
Sco/Cy L^4

Chromosome 3

Drop/Ins(3L)(3R)
Pr Drop/Ins(3L)(3R)
rucuca

Multichromosomal

y;bw bw^+
y v f;Y^{bw+};bw
y;$sc^8 \cdot Y$;bw
y ct v;ru h

Translocations

$T_1(1;2)^K$
$T_2^1(1:3)^K$ (1096)
$T_2^2(1;3)^K$, ru ca (1698)
$T_2^2(1;3)^K$, ru e^s ca (1697)
$T_2^2(1;3)^K$, th st
$T_3^2(1;3)^K$, sc
$T(2;3)$, C^K/Pm;Sb_4
$T(2;3)$, Mo^K/Cy L^4
T(3;4)(Dubinin 2)/DCXF

STONY BROOK, NEW YORK: STATE UNIVERSITY OF NEW YORK
Department of Biological Sciences

Wild Stocks

001 Oregon-R
002 Swedish-B

Chromosome 1

101 w_a
102 w
103 w m f
104 y ct^{6} ras^{2} f
105 f/y^{+} ^{6}
106 ec ct^{6} car/ClB
107 Ins(1)sc^{S1} sc^{8}, B w^{a}

Chromosome 2

201 al dp b pr cn c px sp/Cy sp
202 al dp b pr cn c px sp/Cy pr cn sp
203 ap^{49}/Cy
204 b vg
205 bw
206 dp
207 Ins(2L,2R)Cy bw^{V2}/al dp b pr cn c px sp
208 c px sp

Chromosome 3

301 e^{11}
302 se
303 ru h th st cu sr e^{s} ca
304 bar-3

Chromosome 4

401 ey

Multichromosomal

501 Cy pr cn/Pm; H/Sb-C
502 Cy sp/al dp b pr cn c px sp; ci ey^{R}
503 dp;e
504 pr cn;by

Translocations

601 T(2;3)Mé/ru h th st cu sr e^{s} Pr ca
602 T(2;3;4)bw^{V30k18}/Ins(2LR)Cy

Tumor Stocks

901 tu bw; er^{+} (Su-er)$^{+}$
902 tu A_2 (Milan)
903 tu B_3 (Milan)

SYRACUSE, NEW YORK: SYRACUSE UNIVERSITY
Department of Zoology

Wild Stocks

34 strains each derived from a Syracuse wild inseminated female
9 strains each derived from a South Lancaster, Mass., wild inseminated female
3 Florida strains

Polygenic crossveinless strains

10 strains selected directly from progenies of individual wild inseminated females from various places
6 strains selected indirectly (sensitivity to crossvein defect production at 36.5°C), then directly
det, selected

EMPORIA, KANSAS: KANSAS STATE TEACHERS COLLEGE

Wild Stocks

Oregon-R
Canton-S

Chromosome 1

y
w_a
w_{Bwx}
w_e
w_h
w_{sat}
W^6
ct_3
sn_{37h}
lz^2
ras^2

v
m
s
g
f
y ct^6 ras^2 f
su^2-s v
w m
w_h m f_3
w sn^3 m
sc $S1^1$ B InS w^a sc^8

Chromosome 2

dp
b
cn
en

vg
L^2
bw
al b c sp^2
b vg

Chromosome 3

ve
se
th
st_{11}
e^{11}
se ss k e ro

Chromosome 4

ey^2

pol

Multichromosomal

v;bw
SM^1;Cy/Pm;Ubx^{130}/Sb

Attached-X

lz^3/y f:=
f fu/y f:=

Translocations

lt/T(Y;2)A
T(1;4)B^s/y f:=
ri p^p/st, T(Y;2;3)F

EAST LANSING, MICHIGAN: MICHIGAN STATE UNIVERSITY
Department of Zoology

Wild Stocks

Oregon-R
Urbana
Crimea
Samarkand
Swedish-B
Canton-S

Chromosome 1

w_a
w_e
w
w m f
w m
br

cm
g
f
m
pn
rb
y
Basc
m^D/FM3

Chromosome 2

bw
dp
cn bw
Cy/Pm
vg

pk cn
SD
b pr

Chromosome 3

ca
st
se
e se
h th st cu sr e^s ca
(ru lost)

Multichromosomal

bw;st(2;3)
v;bw(1;2)

bw;e(2;3)
w;bw;st(1;2;3)
Cy/Pm;H/In(3r)Mo, Sb, sr(2;3)
y;bw;e;ci ey^R(1;2;3;4)

Attached-XY

Y^S w y·Y^L y^+/O/y w/O

Attached-X

y m f/y w f:=

Closed-X

X^{c1}, y v/y w f:=

LINCOLN, NEBRASKA: UNIVERSITY OF NEBRASKA
Zoology and Physiology Department

Virus-caused CO_2 sensitivity: several stocks.

Dly (delayed recovery from CO_2 anesthesia, see McCrady and Sulerud, 1964): several stocks.

NAPLES, ITALY: UNIVERSITY OF NAPLES
Istituto di Biolologia Generale e Genetica

Wild Stocks	Chromosome 1	Chromosome 2	Chromosome 3
Bisignano	B	b	cd
Canton	sc^{S1} B InS w^{a} sc^{8}	b cn vg	Sb/+
Lecce	v	bw	Sb/In(3R)1(3)Na
Oregon-R	w_{a}	cl	se
Pavia	w_{bf}	cn	st
Roma	w_{bl}	cn bw	
Sciolze	w_{co}	Cy/Pm	Multichromosomal
	w_{i}	pr	w;vg
	w_{t}	pr en	
	w	vg	
	y		

LYON, FRANCE: UNIVERSITÉ DE LYON
Faculté des Sciences

Wild Stocks

Oregon-R	Lyon	Champetières (inbred)	Algérie

SYDNEY, NEW SOUTH WALES, AUSTRALIA: UNIVERSITY OF SYDNEY
Department of Animal Husbandry

Wild Stocks

3 strains from N.S.W. and Victoria

Chromosome 1	Chromosome 2	Chromosome 3
$In_{2}rst^{3}$	b	e^{11}
pp_{bl}	b j	
w^{bl}	Cy/Pm	
w	j	
y	net	
	vg	

Multichromosomal

sc^{S1} B InS w^{a} sc^{8};Ins SM1, al^{2} Cy Sp^{2}/dp b Pm ds^{33k};C Sb/Ubx^{130} e^{s};pol

TURKU, FINLAND: UNIVERSITY OF TURKU
Department of Genetics

Wild Stocks

1 Canton-S
2 Oregon-K
3 Oregon-R-S
4 Samarkand (Inbred)

Chromosome 1

101 B
102 bb
103 bi ct g^2
104 br w^e ec_{rb} t^4/ FM1, y^{31d} sc^8 w^a lz^s B
105 Bx
106 car
107 cm ct^6 sn^3 & y f:=
108 ct^6
109 cv
110 cv dx sn
111 cv lz/ClB
112 cv sn
113 cv sn lz/ClB
114 ec
115 ec ct v f
116 ec cv
117 f
118 $f_2$car
119 g_2
120 g_2 f
121 g_2 f B & y
122 g^2 f sd
123 In(1)dl_4-49, y $f^n a_4$
124 In)1)sc_8, y sc^8
125 In(1)sc^{S1}, sc^{S1L}
126 Ins(1)sc^{S1R}, S, sc^8, w^a B
127 In(1)w^{m4}
128 lz/FM3, y^{31d} sc^8 dm B l
129 lz m/ClB
130 m
131 m g^2
132 m wy g^2
133 ras^2
134 rb
135 rb cx
136 rsc & y f:=
137 s
138 sc
139 sc cv
140 sc cv v f
141 sc ec cv ct^6 v g^2 f/y^2 sc^{S1} B, In-49, v w^a sc^8
142 sc w cv
143 sd_3
144 sn^3
145 spl
146 t^4
147 v
148 w_{ch}
149 w^{ch} wy
150 wy
151 y
152 y ac v
153 y cv
154 y In49 B^{M1} & y f:=
155 y In49 f car & y f:=
156 y sn^3 bb
157 y w
158 y w cv
159 z

Closed-X

160 X^{c2}, cv v f/ClB

Chromosome Y

191 f·Y^S/Y^L
192 In(1)w^{m4} & extra Y
193 sc^8·Y/y ac sc oc ptg & y f:=
194 v f_2B XY/ y^2 su-w^a w^a bb
195 X·Y^L/Y^S & X·Y^L/ X·Y^L (Neuhaus)

Chromosome 2

201 al^2 Cy, InL_4 lt^3_2/b pr Bl lt^3 cn^2 $InCyR_4$ L^4 sp^2
202 ap^4/Rvd, In2LR
203 Bl L^2/Cy
204 bw_2
205 cn_{tx} InCyR cg sp^2/InsNS px sp
206 dp_{38k} Sp cn^2/S^2 Cy cn^2 (homoz. InCyR)
207 ds^{38k}/Cy(2L), $dp^2{}_3$b pr
208 In(2L)Cy, al^2 ast^3 b pr (Cy not present)
209 NS, b mr/Cy
210 pr cn ix/SM5, al^2 Cy lt^v sp^2
211 rl
212 stw_2^2
213 stw^2 vg
214 vg

Chromosome 3

301 Bd^G/In(3R)C, l(3)a
302 bv_{34e}
303 bx^{34e} bxd^{121}/TM1, Mé ri sbd^1
304 ca bv
305 D^3 Sb/InLP Dfd InRP ca
306 e
307 Gl Sb/LVM
308 h th st_{cp} in ri_{p^p} sr e^s ca/In(3LR) Ubx^{130}, $ri_{Ubx^{130}}$ e_{ca} ("hinca")
309 In(3LR)Ubx^{130}, ri Ubx^{130} e/TM1, Mé ri p^p sbd^1
310 In(3R)$Ant_{B}p^B$, ss^a/TM1, Mé ri sbd^1
311 In(3R)Dl^B, st Dl^B/In(3R)P^W, st l(3)W ca
312 In(3R)P^{FLA} (homozygous)
313 Me, InL Sb/ru h D InsCXF
314 Pc/T(2;3)Mé
315 (ru) h th st cp in ri p^p ss^a bx^3 sr e^s/TM1, Mé ri sbd^1
316 ru h th st cu sr e^s ca ("rucuca")
317 ru h th st cu sr e^s Pr ca/T(2;3)Mé
318 Sb Ubx/T(2;3)Xa
319 Scx/TM1, Mé ri sbd^1
320 se app
321 se rt^2 th/Mé, InL
322 ss bxd e^s/T(2;3)Xa
323 st sr H^2 ca/In(3R)P^W, st l(3)W ca

324 th st cp in ri p^p
325 th st cp in ri p^p bx sr e^s/TM1, Mé ri p^p sbd^1 ("thrie")
326 th st cp in ri p^p ss bxd sr e^s/TM1, Mé ri p^p sbd^1
327 W Sb/InsCXF

Chromosome 4

401 ci_W
402 ci^W
403 spa

Multichromosomal

501 bw; e
502 Cy/Pm; D/Sb (MI)
503 In(3)T(2;3)$Antp^{Yu}$/Cy; Sb

Deficiencies

601 Df(2)MS-4/SM1, al^2 Cy sp^2
602 Df(2)MS-8/SM1, al^2 Cy sp^2
603 Df(2)MS-10/SM1, al^2 Cy sp^2
604 $Df(2)rl^{10a}$ lt cn/Pm ds^{33k}

Translocations

701 T(Y;2)B/b c^3
702 T(Y;2)C/cn^3
703 Bl T(2;3)A; ru h D TA ss e^s/Payne
--- T(2;3)$Antp^{Yu}$ (see 503)
704 al T(2;3)101 sp^2/Cy L^4 sp
705 ru h T(2;3)101 e^4 ro ca/Payne, Dfd ca
--- T(2;3)Mé (see 314, 317)
706 T(2;3)rn (several stocks with different marker combinations)
--- T(2;3)Xa (see 318, 322)

Triploid

801 y^2 sc w^a ec/FM4, y^{31d} sc^8 dm B;cn;h

KWANGJU, KOREA: CHUNNAM NATIONAL UNIVERSITY
Department of Biology

Wild Stocks

1 Chiri (Korea)
2 Kwangyang (Korea)
3 Kwangju (Korea)
4 Naju (Korea)
5 Oregon-s
6 Oregon-R
7 Tolsan (Korea)

Chromosome 1

1 B
2 cm
3 ec
4 rg
5 sc cv v f
6 svr
7 v
8 w
9 w^{ch}
10 y

Chromosome 2

11 b
12 b vg
13 bw
14 bw ba
15 Bl/Cy, bw^{45a} sp^2 or bw^{45a}
16 Bl L/SM, al^2 Cy lt^v sp^2
17 c
18 Cy/Pm
19 ex
20 pd
21 pr
22 rh
23 vg

Chromosome 3

24 aa h
25 ca
26 cv c sbd^2
28 D/Gl
29 gl
30 h
31 ru
32 st
33 th
34 th st cp

Chromosome 4

35 bt
36 ci
37 ci gvl bt
39 ey
40 pol
41 spa

Attached-X

42 br ec/y^{3d}

Inversions

43 sc^{S1} B InS w^a sc^8

VARANASI 5, INDIA: BANARAS HINDU UNIVERSITY
Department of Zoology

Wild Stocks

a) Canton-S
b) Oregon
c) Kerala
d) Cuttak
e) Varanasi

Chromosome 1

ClB
X^{c2}
y
w_i
w_a
w_{bl}
w_e
w_{co}
w_h
w
82 - y sc^{S1} B In49 ct^{ns} sc^{8}

Chromosome 2

b pr vg
vg
g 49 - dp^{txl} Sp ab^{2}/S^{2} ls Cy_1 In Cy L
B - fes ms(b)cn sp/dp^{txl} Cy^{1} cn^{2}
135 - S fes Sp ms ta cn mr crs/dp^{txl} Cy^{1} cn^{2}
g 67 - $ls_T dp^{1}$ Sp ms ta cn crs/S^{2} Cy Bl cn^{2} L^{4} sp^{2}
g 45 - dp^{T} Sp cn bw sp/S^{2} (1st) Cy, In L cn bw sp
Cy D

Chromosome 3

se cu
se^{a} h
ss
Ly/D_3
es
st

Chromosome 4

ey^{2}
ci^{w}

Multichromosomal

fs 13 - y^{+} ac^{+} sc^{8} Y/y B;bw^{D};st^{+} ♂
& y f:=;bw^{D} ♀

102 - Y^{S} X InEN In49 $y \cdot Y^{L}$;st (no free Y)

KALYANI, WEST BENGAL, INDIA: KALYANI UNIVERSITY
Department of Zoology

Wild Stocks

a1 Calcutta University (Originally from Oregon Stock)

Chromosome 1	Chromosome 2	Chromosome 3	Chromosome 4
b1 y	c1 vg	d1 se^{a} h	e1 ey^{2}
b2 w		d2 ss^{a}	e2 ci^{w}
b3 B			

HOBART, TASMANIA: UNIVERSITY OF TASMANIA
Botany Department

Wild Stock

Canton-S (strain from Tasmania)

Chromosome 1

y^2 ^{61g}apr ec cv ct v f
w
B
sc^{S1} B InS w^a sc^8

Chromosome 2

cn bw
fj wt/Xa, T(2;3)
wi
bj
J^{34e3}
rh
bw

Chromosome 4

ey^2

Multichromosomal

bw;st
y;Cy/Pm;H/Sb
$v;e^4$ ro
w;cn bw

Special

B 387 $y_8$snoc and y f:=
F 42 sc^8.Y/ac^3

F 117 ("Y^{LC} snocty") Y^{LC}/oc ptg Y ♂ and Y^{LC}/$\frac{y\ ct^n\ oc\ ptg\ car}{y\ ct^1;In49\ sn^{x2}}$ ♀

F 266 sc^8 Y/snocy ♂; and $Sc^8Y/\frac{y\ ct^n\ oc\ ptg\ car}{y\ ct^1\ In\ In49\ sn^{x2}}$ ♀

F 267 sc^8Y/$snocy^+$ ♂; and $Sc^8Y/\frac{y\ ct^n\ oc\ ptg\ car}{y\ ct^1\ In\ In49\ sn^{x2}}$ ♀

VEPERY, MADRAS 7, INDIA: MADRAS VETERINARY COLLEGE
Department of Animal Genetics

Wild Stocks

Oregon-K
Madras

Chromosome 1

1 m
2 sc^{S1} B InS w^a sc^8
3 v
4 w
5 w m

Chromosome 2

6 b cn bw_2
7 Cy Bl L^2
8 dp b cn bw
9 vg

Chromosome 3

10 e

Attached X

11 sc^{S1} B InS w^a sc^8 & y v f:=
12 sc ct v f car & y v f:=

Multichromosomal

13 bw;st
14 vg;e_{S1}
15 y sc^{S1} In49 sc^8;bw;st

MÜNSTER (WESTF.), GERMANY: INSTITUT FUR STRAHLENBIOLOGIE DER UNIVERSITÄT

Wild Stocks

normal (Berlin wild)

Chromosome 1

sc^{8}·Y/X^{c2} y_{x2}B & y f:=
In-49 v sn^{3} B & y_{L} f:≡
Y^{S}.X In EN v y·Y^{δ} (sc^{8} ? y) (no free Y)

Chromosome 2

b pr vg

Multichromosomal

y sc^{S1} In-49 sc^{8};bw;st
y sc^{S1} In-49 sc^{8};bw;st p^{p}

TÜBINGEN, GERMANY: MAX-PLANCK-INSTITUT FÜR BIOLOGIE

Stocks listed in DIS 38:25 with the following numbers are no longer kept in culture:
2, 4, 6, 8, 10, 15, 16, 18.

New Stock: sc^{8} bb w^{61a}

SANTIAGO, CHILE: UNIVERSIDAD DE CHILE, DEPARTAMENTO DE GENÉTICA
Instituto de Biologia "Juan Noé"

Wild Stocks

1 Chillán
2 Oregon R-C
3 Rapel
4 Santiago
5 Swedish free of inv.
6 Valdivia

Chromosome 1

7 B
8 Bx
9 my
10 v
11 w
12 w m f
13 y

Chromosome 2

14 b
15 b vg
16 bw
17 dp
18 L^{2}
19 L^{2}/Cy
20 S/Cy;En-S
21 vg

Chromosome 3

22 D/Gl
23 e^{11}
24 Gl Sb/LVM
25 se
26 st

Chromosome 4

27 ey^{2}

Multichromosomal

28 dp;e^{11}
29 w;vg

NEW DELHI, INDIA: INDIAN AGRICULTURAL RESEARCH INSTITUTE
Department of Botany

Wild Stocks

1 Oregon-K

Chromosome 1 (X)

1 B
2 sc^{S1} B InS w^{a} sc^{8}
3 y
4 y sc^{S1} InS sc^{8}
5 y sc^{S1} B In49 ct n^{s} sc^{8} (Binsety)

Chromosome 2

1 vg
2 cn bw
3 b cn bw
4 dp b cn bw
5 dp b cn
6 Cy cn bw
7 bw
8 Cy/Bl L^{2}
9 fes ms b cn Sp/Cyo
10 fes ms cn Sp/Cyo
11 fes Sp ms ta_{2} cn mr(Crs)/Cyo
12 dp^{tx} Sp ab_{2}/S^{2} ls_{1}Cy, In(Cy)L
13 dp b L^{4} Pm^{1}/dp tx^{1} Cy, o^{5} pr cn^{2}
14 ls dp Sp ms ta_{4} cn Crs/S^{2} Cy lt^{3} pr^{+}
15 dp^{tx} Sp Cn bw Sp/S^{2}(ls^{+})Cy In(L)cn bw Sp

Chromosome 3

1 e
2 st

Chromosome 4

1 ey

Multichromosomal

1 bw;st
2 y sc^{S1} InS sc^{8};Cy/Bl L^{2}
3 y sc^{S1} InS sc^{8};bw;st
4 y sc^{S1} InS sc^{8};cn bw
5 y sc^{S1} InS sc^{8};vg
6 y sc^{S1} InS sc^{8};dp b cn bw
7 sc^{S1} B InS w^{a} sc^{8};Cy
8 y sc^{S1} In49 v_{4} sc^{8};dp^{h} b bw/dp^{txl} Cy Pl ^{8}Bl cn^{2} L^{4} Sp^{2}
9 sc^{8} Y/y B & y f;= Bw^{D}

Attached X

1 sc^{S1} B InS w^{a} sc^{8} &y v f:=
2 sc^{S1} f In49 v w^{a} sc^{8} & y f:=
3 y sc^{S1} f In49 v w^{a} sc^{8} & y f:=

Altered Y with mutants in X-chromosome

1 lj^{1} Y/l j^{1} sc^{j1}(+)In49 v Ptg oc^{2} B^{M1}/y sc^{S1} car odsy f g_{1} dy_{8}v ras^{2} sn^{3} sc^{6} cm rb ec w Pn^{1} sc^{8} (Maxy) ct

BAYFORDBURY, HERTFORD, HERTS, ENGLAND: JOHN INNES INSTITUTE

Wild Stocks

1 Bayfordbury
2 Hampton Hill
3 Samarkand
4 Teddington

Inbred Lines

5 Oregon (v marker), 229
6 b pr, 127

Chromosome 1

7 v
8 y w

Chromosome 2

9 b pr vg

Multichromosomal

10 Cy L^{4}/Pm;H/Sb

ROMA, ITALY: CITTÀ UNIVERSITARIA
Istituto di Genetica

Wild Stocks

A1 Oregon
A2 Marzi

Chromosome 1 (X)

B 1 B
B 2 car^{S1} bb
B 3 sc^{8} B InS w^{a} sc^{8}
B 4 pn^{R}
B 5 r/y f:=
B 6 sc cv v f B/y f:=
B 7 sc z ec
B 8 sw
B 9 w^{a}
B10 w^{Bwx}
B11 w^{cf}
B12 w^{cp}
B13 w^{r}
B14 w^{cf}
B15 w^{e}/y f:=
B16 w^{e} dy/y w:=
B17 en w/y f:=
B18 y^{1}
B19 y^{59}
B20 y ac sc pn/y f:=
B21 y cv v g/y f:=
B22 y fa^{n} sn^{3}
B23 y sc w^{col} spl f/In(1) rst^{3}, rst^{3} f
B24 y$_{2}$w^{a} spl rb
B25 y^{2} su-w^{a} w^{a} bb/y w spl rb
B26 y^{2} v ma-l^{cf}
B27 y^{2} w^{31d}
B28 y v^{of} w^{a} f sin/y f:=
B29 y^{1259}/y^{2} su-w^{a} w^{a} bb/sc^{8}·Y
B30 XX/O;X·Y

Chromosome 2

C1 b cn c bw
C2 b cn v bw
C3 Bl L/Cy
C4 Bl L$^{v}_{lt}$/SM5, al^{2} Cy sp^{2}
C5 bw^{D}
C6 bw
C7 cn bw
C8 Sp J$_{v}$L^{2} Pin/SM5, al^{2} Cy lt^{v} sp^{2}
C9 Tft/Cy

Chromosome 3

D1 ca K-pn
D2 Gl Sb/LVM
D3 H/Sb sr In(3R)Mé
D4 H^{2}/In(3R), Vno, Vno^{s}
D5 ru h st cu sr e^{s} ca
D6 se ss k e^{s} ro
D7 st sr e^{s} ro ca
D8 st C3G ca/In(3LR)Ubx^{130}, Ubx^{130} e^{s}

Chromosome 4

E1 ci ey^{R}
E2 pol

Multichromosomal

F 1 bw;st(2;3)
F 2 lys re;ss(2;3)
F 3 Cy/Pm;Sb/Ubx(2;3)$_{R}$
F 4 sc cv v f B;ci ey^{R}(1;4)
F 5 y;pol(1;4)
F 6 y f;ci ey^{R}(1;4)
F 7 y;ru h th st p^{p} cu sr e^{s}(1;3)
F 8 y^{2} cho^{2};lys re(1;2)
F 9 y;bw;st(1;2;3)
F10 al L^{4} Cy sp/Pm;H/Sb sr In(3R) Mé(2;3)

Triploid

G1 y^{1259}/FM4, w f/FM4, w f

Translocations

H1 T(1;4)B^{S}(16 A1), y^{2} cv v B^{S} car/y f:=
H2 T(2;3)bw^{V4}, bw^{V4}/Cy

Inverted X Chromosomes

I 1 In(1)dl-49, v^{of} f
I 2 In(1)dl-49, w lz
I 3 In(1)dl-49, y Hw m^{2}/fa N^{22a}
I 4 In(1)dl-49, y Hw m^{2} g^{4}/Df(1)N$^{8}_{4L\ 8R}$
I 5 In(1)sc^{8}, sc^{8}, y sc^{4+8} cv v f/y f:=
I 6 In(1)sc$^{7}_{8}$, AM^{8}
I 7 In(1)sc^{8}, sc^{8}
I 8 In(1)sc^{8}, dl-49, y^{31d} w^{a}
I 9 In(1)sc^{8}, dl-49, y^{31d} v^{of} f/y f:=
I10 In(1)w$^{m4}_{m4L}$/Df(Y)Y^{bb}
I11 In(1)w$^{m4}_{3R}$, rst^{51b7}, y w^{-} rst^{3}/Dp(1;2R)w^{51b7}/y w f:= Y
112 In(1)481(12E-F;14B), y bb^{1481}/FM6, y^{31d} sc^{8} dm B/sc^{8}·Y

Deficiencies & Duplications

L1 Df(1)N^{8}
L2 Df(Y)Y^{bb-}
L3 Dp(1;2R)w^{51b7}
L4 Dp(1;1)B^{S}(RMG), y w^{a}·B^{S}/sc^{S1} dl-49, v

X Chromosomes with A Y Arm Attached

M1 X·Y^{L}(C-2), y cv v f car bb^{-}·Y^{L}
M2 X·Y^{S}(A-3), sc$_{S}$cv v·Y^{S}
M3 Y^{S}X·(FR-1), Y^{S} y cv v$_{S}$f
M4 Y^{S}X·(P-7), In(1)EN, Y^{S} y f

Attached - XY

N1 XY^{L}·X^{S}(108-9), y^{2} su-w^{a} w^{a} Y^{L}·Y^{S}
N2 XY^{S}·Y^{L}(115-9)$^{+}$, y^{2} su-w^{a} w^{a} Y^{S}·Y^{L} y
N3 XY^{S}·Y^{L}(110-8)$^{+}$, y^{2} su-w^{a} w^{a} Y^{S}·Y^{L} y
N4 XY^{S}·Y^{L}(129-16)$^{+}$, y^{2} su-w^{a} w^{a} Y^{S}·Y^{L} y

N5 $Y^{S}X{\cdot}Y^{L}$, Ins(1)EN, dl-49, Y^{S} car f v $y{\cdot}Y^{L}$

Altered Y

P1 $Y^{Su\text{-}Var}$

P2 $Y{:}bw^{+}(Y^{L}\ bw^{+}{\cdot}bb^{+}\ Y^{S})$
P3 $Y^{C}{:}bw^{+}/X;bw$
P4 $B^{S}\ Y\ y^{+}/y^{2}\ su\text{-}w^{a}\ w^{a}\ bb$
P5 $B^{S}\ Y\ y^{+}/y^{2}\ su\text{-}w^{a}\ w^{a}\ bb$
P6 $B^{S}Y\ y^{+}(B^{S}Y^{L}{\cdot}bb^{+}Y^{S}\ y^{+})$

BLINDERN, NORWAY: UNIVERSITY OF OSLO
Institute of General Genetics

Wild Stocks

1 Amherst iso-
2 Canton-S
3 Florida 26-24
4 Florida 10
5 Formosa, Japan
6 Lausanne-S
7 Oregon
8 Oregon R-C
9 Oregon iso-
10 Oslo
11 Samarkand (inbred)
12 Swedish-b-6 (Swedish-b cleaned of inversions)
13 Woodbury

Chromosome 1

14 Bx^{3}
15 ec ct^{6} v g^{3}/ClB
16 f
17 fa
18 f B od car & y f:=
19 f od sy car
20 g f car & y f:=
21 od car
22 r^{39k} f B/In(I)AM
23 sc cv v f
24 $sc^{Bwx}w$
25 sc^{S1} B InS w^{a} sc^{8}
26 w
27 w m f
28 w^{a}_{Bwx}
29 w
30 y
31 y ec ct^{6} v f
32 y pn
33 y w

Altered Y's

34 sc^{8} Y;w
35 y sc^{8} Y;sc^{S1} B InS w^{a} sc^{8}

Chromosome 2

36 al^{2} Cy, InL, lt^{3}/ b pr Bl lt^{3}_{4} In Cy R L^{4} sp^{2}
37 al dp b pr c px sp
38 al dp b pr^{2} c px^{3} sp/Cy^{4} al^{2} lt^{3} L^{4} sp^{2}
39 b j pr cn
40 bw
41 $cn\ bw^{5}$
42 dp^{11f26}
43 $fj\ px^{49k}$ sp
44 j^{50e5}
45 j^{2}
46 L^{2}/Cy
47 M(2)z/Cy L
48 M(2)50j7/Cy^{4}, al^{2} lt^{3} L^{4} sp^{2}
49 Pm/Cy^{4}, al^{2} lt^{3} L^{4} sp^{2}
50 pr^{37b} en
51 S/Cy
52 vg

Chromosome 3

53 cp^{4}
54 e^{3} wo ro
55 gl^{3}
56 Gl Sb/LVM^{r}
57 jv Hn^{2} h
58 ma^{42a} fl
59 p
60 ri
61 se
62 st
63 st e^{2}
64 ve

Chromosome 4

65 ar/ey^{D}_{R}
66 $ci^{2}ey$
67 ey^{2}

Multichromosomal

68 sc^{S1} $_{8}$B InS w^{a} sc^{8};cn bw;e^{11}
69 bw;st
70 cn bw;e^{11}
71 cn bw;ru h st p^{p} ss e^{s}
72 Cy/Pm;H/C Sb_{R}
73 y;bw;e;ci ey

Deficiencies and Translocations

74 $Df(1)N^{8}_{2}/dl\text{-}49$, y Hw m^{2} g^{4}
75 Df(2)Px^{2}, bw sp/Cy L
76 T(2;3)Cy

BUENOS AIRES, ARGENTINA: UNIVERSIDAD DE BUENOS AIRES
Facultad de Ciencias Exactas y Naturales, Laboratorio de Genética I

The same stocks as in the Laboratory of Genetics of the Argentine Atomic Energy Commission.

CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGO

D. americana

Wild Stocks

1 Independence
2 Anderson

Others

3 cn
4 o

D. texana

5 New Orleans

D. virilis

Wild Stocks

6 Pasadena lethal-free
7 Texmelucan

Chromosome 1

8 ap^{40e}
9 w^{50112}
10 y^{40a} dy
11 y^{40a} ec ch^{2} dy

Chromosome 2

12 b bk dt
13 va

Chromosome 3

14 gp^{2}
15 S/+
16 sv
17 sv t tb gp^{2}
18 sv tb gp^{2}
19 tb
20 tb gp^{2}

Chromosome 4

21 cd

Chromosome 5

22 B^{4} pe^{Jap}
23 es B^{3}
24 es pe
25 pe
26 ru
27 ru st B^{3} pe^{Jap}
28 ru st es mh B^{3}
29 ru st mh
30 st B^{3}
31 st es pe^{Jap}
32 st mh
33 st pe
34 st pe^{Jap}

Multichromosomal

35 b;pe^{Jap}
36 b;th gp^{2};cd;pe
37 b;sv t tb gp^{2};cd pe
38 b;sv t tb gp^{2};pe
39 cd;B^{3} pe
40 cd;es pe
41 cd;pe
42 cn;pe
43 pe;gl
44 "scute"(II);pe^{m3}
45 S/+;B^{3} pe
46 t;cd
47 v^{48a};pe
48 v^{48a} w;pe
49 v^{48b};pe
50 w;cd;pe

SYRACUSE, NEW YORK: SYRACUSE UNIVERSITY
Department of Zoology

D. subobscura

wild strain C from A. Prevosti
crossveinless polygenic strain v.t.i. from A. Prevosti

D. simulans

6 wild strains from Florida

D. willistoni

2 wild strains from Florida

Additional Wild Strains

D. funebris
D. hydei
D. pseudoobscura
D. virilis
D. willistoni

BALTIMORE, MARYLAND: JOHNS HOPKINS UNIVERSITY

a1 D. funebris
a2 D. hydei
a3 D. simulans
a4 D. simulans, La-3
a5 D. simulans, La-4
a6 D. simulans, Lima, Peru, a^{5}
a7 D. simulans, Lima, Peru, a^{10}
a8 D. simulans, New Orleans
a9 D. simulans, South Africa
a10 D. virilis

EAST LANSING, MICHIGAN: MICHIGAN STATE UNIVERSITY
Department of Zoology

D. robusta: East Lansing 1964
D. melanica: East Lansing 1964
D. immigrans: East Lansing 1964
D. virilis: California
D. virilis: Yale
D. pseudoobscura: Yale
D. pseudoobscura: Rochester
D. funebris: Yale

NEW YORK CITY, NEW YORK: COLUMBIA UNIVERSITY
Department of Zoology

D. equinoxialis

Wild Strains; geographical

Brazil (3 strains)
British Guiana (2)
Mexico (1)
Puerto Rico (1)
Trinidad (2)
Venezuela (4)

Sex-Ratio

Puerto Rico (1)

D. paulistorum

Wild Strains; geographical (by races)

Amazonian race

British Guiana (3)
Colombia (2)
Panama (1)
Trinidad (2)
Venezuela (2)

Andean-Brazilian race

Brazil (12)
Colombia (3)

Transitional strains

Andean-Brazil (9)

Calypso race

British Guiana (1)
Trinidad (1)

Centro-American race

Panama (1)

Guianan race

British Guiana (1)

Orinocan race

British Guiana (4)
Panama (2)
Venezuela (2)

D. persimilis

Wild: 3 strains

D. pseudoobscura

Wild: 3 strains

D. willistoni

Wild Strains, geographical

Brazil (4)
Puerto Rico (1)

Sex-Ratio

Brazil (1)

LEXINGTON, KENTUCKY: UNIVERSITY OF KENTUCKY
Department of Zoology

D. affinis: Lexington, Kentucky
D. putrida: Lexington, Kentucky
D. robusta: Lexington, Kentucky
D. busckii: Lexington, Kentucky
D. hydei: Lexington, Kentucky

ST. LOUIS, MISSOURI: WASHINGTON UNIVERSITY
Department of Zoology

D. robusta

Notation of arrangements as in Carson, H. L., 1953 Genetics 38:168.

Stocks homozygous for gene arrangement

Stock designation	X	2	3	Stock origin
Standard	SS	SS	SS	New York
BF-5	11	3S	SS	Big Fish Lake, Minnesota
TA	32	11	S1	Lake Tsala Apopha, Florida
St e-17	11	1S	SS	
C-66 (R1-C)	1S	3S	SS	Chadron, Nebraska

Other Species

D. americana Meramec Standard
D. buzzatii (various)
D. carsoni Bridgewater, Vt. 1957
D. colorata Petoskey, Mich.
D. crucigera Mt. Tantalus, Oahu, Hawaii
D. euronotus (various)
D. immigrans (various)
D. lacertosa UTL No. 25313, Sapporo, Japan
D. micromelanica (various)
D. melanica (various)
D. melanura (various)
D. mercatorum (various)
D. nigromelanica (various)
D. paramelanica (various)
D. repleta (various)
D. stalkeri (various)

LONDON, ENGLAND: UNIVERSITY COLLEGE LONDON
Department of Zoology

D. subobscura

Inbred Lines

B, K & NFS (structurally homozygous) & several others

Wild Stocks

Edinburgh
Galilee

Chromosome 1

Bx

m ct bnt v sc
oc

pm ct sn
v w sc/v w^{co} sc
y

Chromosome 2

ma int mj
th ma mop

Standard order

s th int ey wt
s th int ey mj
th ma

Inverted order

ni

Chromosome 3

ho
nt fd^{M}
+/gs
nt

Chromosome 4

pl pp pt
sj otp pl pp

Chromosome 5

ch ar
ch cu
ch/Ba ant
ch/Ba$_{2}$st
ch kk^{2}/Ba rn

D. hydei vg
D. simulans Jer +

SYDNEY, NEW SOUTH WALES, AUSTRALIA: UNIVERSITY OF SYDNEY
Department of Animal Husbandry

D. ananassae (as in DIS 39:67)
D. montium (as in DIS 37:58)
D. nebulosa " "
D. persimilis (as in DIS 37:58)
D. pseudoobscura " "
D. simulans " "

OSAKA, JAPAN: OSAKA UNIVERSITY
Medical School, Department of Genetics

D. virilis

Wild Stocks

1 Kaidema (Japan)
2 Pasadena (USA)

Chromosome 1

3 v^4
4 w^a
5 y

Chromosome 2

6 eb

Chromosome 3

7 cn

Chromosome 4

8 cd

Chromosome 5

9 st B^3 pe
10 st es

Multichromosomal

11 v;es (1;5)

D. simulans

Wild Stocks

1 A (USA)
2 B (USA)

Chromosome 1

3 v
4 y w

Chromosome 2

5 net
6 net b py sd pm

Chromosome 3

7 jv se
8 jv st se
9 H^h pe

LYON, FRANCE: UNIVERSITÉ DE LYON
Faculté des Sciences

D. funebris
D. busckii

SÃO JOSÉ DO RIO PRÊTO, SÃO PAULO, BRAZIL: GOVÊRNO DO ESTADO DE SÃO PAULO
Faculdade de Filosofia, Ciências e Letras, Deptº de Biologia Geral

D. sturtevanti - Jamaica, Cuba, Porto Rico, Guadalupe, Santa Lúcia, Trinidad, Colômbia, Peru, Mexico, Honduras, Panama, Guiana Britanica, Brazil (Amazonas, Pará, Maranhão, Espírito Santo, Guanabara, Paraná, Rio Grande do Sul, Mato Grosso, Goiás).

SANTIAGO, CHILE: UNIVERSIDAD DE CHILE
Instituto de Biología "Juan Noé", Departamento de Genética

D. busckii: Chile (La Serena)
D. camaronensis: Chile (Azapa)
D. funebris: Chile (La Serena, Valdivia, Tierra del Fuego y Punta Arenas)
D. gasici: Chile (Arica), Bolivia (Cochabamba), Colombia (Bogotá)
D. gaucha: Brazil (M. Capoes, C. de Jordan and Taimbas), Argentine (Córdoba)
D. hydei: Chile (Camarones, and El Tabo), Bolivia (Cochabamba)
D. immigrans: Chile (El Tabo and Valdivia)
D. mercatorum: Chile (Arica)
D. mesophragmatica: Bolivia (La Paz), Perú (Machu-Picchu, Cuzco)
D. pavani: Chile (Copiapó, Vallenar, La Serena, El Tabo, Viña del Mar Olmué, Bellavista, Arrayán, Los Alpes, Colbúm, Los Quenes, Chillán), Argentine (Mendoza)
D. simulans: Perú (Lima), Chile (Arica)
D. viracochi: Perú (Machu-Picchu), Colombia (Bogotá)
D. virilis: Chile (Santiago)

SAPPORO, JAPAN: HOKKAIDO UNIVERSITY
Faculty of Science, Department of Zoology

D. auraria: race A; Sapporo(1), Okoppe(1), Oshoro(1), Fuyushima(1)
D. lutea: Sapporo(1)
D. virilis: Sapporo(3), Okushiri(1)
D. immigrans: Sapporo(1)
D. funebris: Sapporo(2), Akan(1)
D. brachynephros: Sapporo(1), Nopporo(1)
D. unispina: Sapporo(1)
D. nigromaculata: Sapporo(2), Uryu(1)
D. testacea: Naebutoro(1), Uryu(1)
D. busckii: Sapporo(1)
D. histrioides: Sapporo(2)
D. histrio: Sapporo(1), Fuyushima(1)
D. ananassae: India(1)
D. sordidula: Sapporo(15)
D. sp. like sordidula: Nopporo(1), Doya(1)
D. lacertosa: Sapporo(2), Fuyushima(5), Doya(2)
D. moriwakii: Nopporo(1), Yamada-Onsen(1)
D. sp. like okadai: Nopporo(1)
Parascaptomyza pallida: Yamada-Onsen(2)

KWANGJU, KOREA: CHUNNAM NATIONAL UNIVERSITY
Department of Biology

D. auraria: Type C (5 strains)
D. rufa
D. brachynephros
D. angularis
D. immigrans
D. suzukii
D. bizonata
D. virilis
D. nigromaculata
D. busckii

BAYFORDBURY, HERTFORD, HERTS, ENGLAND: JOHN INNES INSTITUTE
Department of Genetics

D. simulans

TURKU, FINLAND: UNIVERSITY OF TURKU
Institute of Genetics

D. simulans

Wild Stocks

Chromosome 3

jv se st pe
st pe

VARANAS, INDIA: BANARAS HINDU UNIVERSITY
Department of Zoology

Wild Stocks

(a) D. bipectinata (Calcutta)
(b) D. ananassae (Howrah)
" " (Andhra)
" " (Kerala)
" " (Mughalsarai)
" " (Bhagalpur)
(c) D. malerkotliana
(d) D. nasuta
(e) D. immigrans

Mutants of D. ananassae

Chromosome 1

y vs v
y_a
w

v
ct
vs

Chromosome 2

cu b se
cu se
b se
cu b
ic b
ic se
b
cu
se
ic
cu bw

blo
a
ss

Chromosome 3

px pc stw
px pc
stw pc
stw px
stw
px
pc

Unlocated mutants

dct
sp
ci
arch

CHANDIGARH, PUNJAB, INDIA: PANJAB UNIVERSITY
Department of Zoology

D. melanogaster (4 strains)
D. takahashii
D. kikkawai
D. nepalensis
D. suzukii
D. malerkotliana (2 strains)
D. ananassae (3 strains)
D. bipectinata (2 strains)
D. jambulina
D. punjabiensis
D. immigrans

BERLIN-DAHLEM, GERMANY: UNIVERSITÄT BERLIN
Institut für Genetik der Freien

72 D. funebris - wild
73 D. busckii - wild
74 D. hydei - wild
75 D. simulans - v
76 D. virilis - wild

KALYARI, WEST BENGAL, INDIA: KALYARI UNIVERSITY
Department of Zoology, Genetics Laboratory

D. ananassae

Wild Stocks

1 Kalyani (Kln Cp)
2 Ichapur (I Pr)
3 Ranaghat (Rgt)
4 Calcutta (Cal po)
5 Dhakuria (Dka)
6 Sahaganj (Sgnj)

OSAKA, JAPAN: OSAKA UNIVERSITY
Medical School, Department of Genetics

D. ananassae (USA)
D. funebris (Japan)
D. hydei (Japan)

TÜBINGEN, GERMANY: MAX-PLANCK-INSTITUTE FÜR BIOLOGIE

For complete list, see DIS 39:73.

The following species listed in DIS 39 are no longer kept in culture:
D. miranda
D. paulistorum

New Species

D. bifurca
D. fulvimacula
D. nigrohydei
D. repleta, glass

Mutant stocks of D. hydei listed in DIS 39 with the following numbers have been lost or discarded: 14, 15, 16.

New Stocks of D. hydei

Chromosome 1

1 cherry-tomato
2 cherry-tomato-2
3 forked
4 forked-2: In(1)
5 miniature-63i
6 miniature-63j
7 prune
8 tomato
9 tomato-2
10 tomato-rood
11 vermilion-Tubingen-3 (homozygous inviable)
12 white-mottled-1: Tp(1)
13 white-mottled-2: In(1)
14 white-mottled-3: In(1)
15 Notch-Tübingen-1: Df(1)
16 Notch white-deficience: Df(1)
17 yellow-Tubingen-2

Y-Chromosome

18 tube-proximal
19 tube-distal

LOUVAIN, BELGIUM: THE UNIVERSITY
Janssens Laboratory for Genetics

D. americana
D. subobscura (Belgium)
D. subobscura (Küssnacht)(Standard Homozygous)
D. virilis

NEW MUTANTS

Report of P. T. Ives

e^{60h}: $ebony^{60h}$ Ives, 60h. Arose spontaneously in the vg;se stock and has a medium strong phenotype.

gl^{63d29}: $glass^{63d29}$ Ives, 63d29. Induced simultaneously with and not yet separated from a T(2;3) by Cobalt-60 gamma rays. Eye much reduced in size, nearly colorless, with a typically glassy surface. The developmental viability of homozygous $T(2;3)gl^{63d29}$ is generally low when in competition with heterozygotes but is very good in pure culture.

Report of B. H. Judd

w^{zm}: white-zeste mottle The description of this mutant in DIS 39:60 stated that the white locus change is located left of w^a; this is incorrect. Fine structure analysis shows that the change in the white locus is located right of w^a and probably right of w^{ch}.

w^{zl}: white-zeste light Fine structure analysis of this mutant shows the change to be located to the right of w^a and probably also to the right of w^{ch}. The description in DIS 39:60 stating that the change is left of w^a is incorrect.

Report of Kalyani University

held out Spontaneous from wild stock collected from Kalyani after 4th generation. Wings held out horizontally. Recessive; linkage group not yet established.

Report of G. H. Mickey

vs^{61j}: vesiculated 61j Mickey 61j10. 1-16.3. Arose from wild type male treated with radio frequency energy. Uniform expression in both males and females. Originally reported as bw^{61j} (DIS 38:28) and symbol changed to bu-w^{61j} (DIS 39:58). Determined as allele of vesiculated by M. J. Fahmy (personal communication) RK2.

Report of H. J. Muller

spx, In:spreadex Chromosome 1; derived from X-rayed spermatozoon; recessive; wings spread widely apart and often directed somewhat downwards, as in Dichaete; abdomen of female tends to be narrow and shrunken but usually has sufficient fertility for maintenance as homozygous stock; arose simultaneously with inversion that is judged by its crossing over properties to be similar in length to In49 but with both of its breaks somewhat to the right of those of In49. Existing stocks, transmitted to Oster, consist of spx, In alone and y^2 spx, In f car.

Tul: Turned-up-like Of spontaneous origin, chromosome 1; heterozygote with wings like those of heterozygous Tu, that is, slightly but usually definitely turned up near ends but not twisted; hemizygote and homozygote more extreme, viable and fertile, with wings usually wrinkled, also as in Tu, and sometimes held somewhat apart; not located exactly but between g and f at about 50 (±3). By a curious coincidence our stock "Maxy-Tu" (f36 of our DIS 31 list), which was constructed with the intention of having Tu in the non-multiple-recessive X and which was thought to have been so made, was much later found not to have Tu as planned but to mimic this result by reason of having Tul, unknown previously to the tests in question, in the homologous chromosome.

We: Wedge eye Of spontaneous origin, chromosome 3; lethal when homozygous; viability, fertility and classifiability good when heterozygous. Eye reduced to a small vertical wedge with the point downwards, much smaller than homozygous Bar. Location one to several units to the right of claret, i.e. near right end of III, so as to cross over fairly often from Dichaete when that is associated with CXF Inversion complex. Exists in well-balanced form in stock containing In3RC e 13e in chromosome homologous to its own.

gy: gouty legs Of spontaneous origin, chromosome 4; recessive, viable, fertile, and usually classifiable as homozygote; shortening and thickening of the legs, more especially of the metatarsi of the hind legs, which are often swollen. This mutant behaves as an allele of a previously undetected recessive feature of Dominant-eyeless, inasmuch as the compound of the latter with gy manifests the gy phenotype in at least as extreme form as that of homozygous gy. It is therefore very probable that gy is located close to the point in IV, identified by Bridges, at which the break occurred into which the transposed section of chromatin associated with Dominant-eyeless was inserted.

Report of H. L. Plaine and Sister M. Baptista Aubele

ove: overetherized Plaine and Aubele, 1964b. Recessive on second chromosome; not localized. Spontaneous in one male of Swedish-b erupt strain; recovered in isogenic progeny derived from this male. Wings held perpendicular to longitudinal axis of body, giving the appearance of flies which have been overetherized. Wings of newly emerged flies expand to normal position within one hour ($\bar{X}$ = 45.8 min., standard error = 2.08); permanently elevated to perpendicular position within thirty minutes to one hour after expansion ($\bar{X}$ = 36.9 min., standard error = 1.13). Flies capable of weak wing vibrations but incapable of flight. Lack of coordination of first two pairs of legs apparent from time of emergence; morphology of legs apparently normal. Completely penetrant; uniform expression in both sexes. Mutant viable; females fertile; males successful in mating with mutant females but lack or have lowered mating ability with females of wild-type and other mutant strains. Maintained in homozygous condition in Swedish-b erupt background and in heterozygous state balanced over dp^{txl} Cy, Ins05 pr cn^{2}.

Report of J. B. Spofford

Su-V: Suppressor of Variegation Spofford, 61c. 3-41.3 Pub. 1962, Genetics 47: 986-987. Isolated from $Dp(1;3)w^{m264\cdot 58a}$ stock. Reduces mutant effect (sometimes completely) of w, rst, fa, spl, nd and dm variegation of the corresponding loci within $Dp(1;3)w^{m264\cdot 58a}$. Also reduces w variegation of $In(1)w^{m4}$ and rst variegation of $In(1)rst^{3}$. Enhances sc variegation of $In(1)sc^{4}$ and y variegation of $In(1)y^{3p}$. Semicominant, heterozygotes distinct. Maternal effect, Su-V/+ offspring of Su-V/Su-V clearly distinguishable from Su-V/+ offspring of Su-V/+ mothers and the latter clearly distinguishable from +/Su-V offspring of +/+ mothers. Slightly less fertile as homozygotes. Viable in both sexes.

Report of Ruby M. Valencia

fw^{wr}: furrowed Valencia, 1959 1-38.3. The mutant described in DIS 33:100 as wr: wrinkled has been found to be an allele of furrowed.

sd^{spa}: scalloped-spatula Valencia, 1959 1-51.5 The mutant described in DIS 33:99 as spa: spatula has been found to be an allele of scalloped. Interacts with Bx^r when both are in heterozygous condition. Cytological examination showed chromosome probably normal, although there was an undefinable "disturbance" around the locus.

En-sd: Enhancer of scalloped Valencia, 1963 When sd^{spa} (spatula) females were mated to Beadex (Philadelphia stock b 16), a few of the sons were found to be almost wingless, while the rest were typical spatula. When these wingless males were mated to attached-X females, one-half of the sons were wingless, thus indicating an autosomal dominant enhancer of scalloped. The autosome has not yet been identified. En-sd does not enhance Bx or Bx^r.

DROSOPHILA SPECIES

NEW MUTANTS

hydei

Report of O. Hess and M. M. Green

to: tomato Hess 63g4 1-48.0 (locus by Green). Eye color bright reddish orange darkening to dull brown with age. Phenotype inseparable from that of previously described ch (1-115) of Spencer. X-ray induced as single ♂. RK1

to^2: tomato-2 Green 63k5 1-48.0. Inseparable from to. X-ray induced as single ♂. RK1

to^3: tomato-3 Green 63k7 1-48.0. Inseparable from to. X-ray induced as single ♂. RK1

to^4: tomato-4 Gloor 62g 1-48.0. Inseparable from to. X-ray induced as single ♂. RK1

pn: prune Green 63j31 1-13.4. Dark maroon eye color. Possible homologue of pn in D. melanogaster. X-ray induced as single ♂. RK1

f: forked Gloor 64c 1-86± (locus by Green). Bristles twisted and gnarled as in f of D. melanogaster for which it is good homologue. X-ray induced in ♀ as single F_1♂. RK1

f^2: forked-2 Green 6311 In(1). Bristles forked; phenotypic allele of f. Crossing over between m and ch reduced to about 18%; expected 50%. Crossing over to left of m nearly normal. Homozygous ♀♀ inviable. X-ray induced as single ♂. RK2 Lost.

f^3: forked-3 Green 6315 In(1). Bristles forked; phenotypic allele of forked. Crossing over reduced over entire X. Between m and ch crossing over reduced to 3-5%; expected 50%. To left of m, crossing over rare, only occasional doubles found. X-ray induced as single ♂. RK2

Sp: Spread Green 63j25 1-?. Wings held at angle from body. Occasionally overlaps wild-type. Probably localized to right of m. X-ray induced as single ♂. RK2

w^{m1}: white-mottled-1 Green 6318 Tp(1) Eye color dark red mottled. X-ray induced as single w^{m1}/w female. Salivary cytology (by A. S. Mukherjee) suggests X heterochromatin including NOR inserted into section 17A (Berendes Map).

w^{m2}: white-mottled-2 Green 63l12 In(1) Eye color yellow-orange mottled. Strongly modified toward wild by additional Y. w^{m2} ♂♂ low viability, very late hatching. X-ray induced as single w^{m2}/w female. Salivary cytology (by A. S. Mukherjee) suggest pericentric inversion with one break in heterochromatin, one break just after doublet 17A1,2. Also includes an insertion of heterochromatin at 9A,B.

w^{m3}: white-mottled-3 Green 64b12 Tp(1) Eye color of heterozygous $w^{m3}/w_{♀♀}$ between that of w^{m1} and w^{m2}. Homozygous females and hemizygous males phenotypically nearly normal. X-ray induced as single w^{m3}/w female. Salivary cytology (by A. S. Mukherjee) shows insertion of heterochromatin at 17A as in w^{m1}.

w^{63l}: white-63l Green 63l12 1-19 Eye color snow white. Malpighian tubules and testes sheath also w. X-ray induced as single ♂. RK1. Lost.

w^{64a}: white-64a Green 64a1 1019. Inseparable from w^{63l}. Recovered as single w lt/$w^{+}_{♀}$ from X-rayed wild-type ♂. RK1. Discarded.

w^{64b}: white-64b Green 64b2 1-19. Inseparable from w^{63l}. Recovered as single w lt/$w^{+}_{♀}$ from X-rayed wild-type ♂. RK1. Discarded.

N^{t1}: Notch-Tübingen-1 Green 63l5 1-22.3. Df(1). Phenotype homologous to Notch in D. melanogaster. Homozygous and hemizygous lethal. Recovered as single female. X-ray induced.

Df(1)w, N: white Notch Deficiency Hess 64g25 Df(1) Notch phenotype inseparable from N^{t1}; $N/w_{♀♀}$ show phenotype of white, indicating that the deficiency also includes locus of white. Homozygous and hemizygous lethal. X-ray induced, irradiation of $+/+_{♀}$ which was crossed with w/Y♂. Recovered as single F_1 female. Salivary cytology (by A. S. Mukherjee) shows loss of bands from 16C-17A inclusive (Berendes' map).

ch^{t}: cherry-tomato Hess 63b22 1-115.7. Inseparable from ch. X-ray induced as single ♂. RK1

ch^{t2}: cherry-tomato-2 Green 63k4 1-115.7. Inseparable from ch. X-ray induced as single ♂. RK1

g^{63k}: garnet-63k Green 63K7 1-32.5. Inseparable from g. X-ray induced as single ♂. RK1. Discarded.

v^{t1}: vermilion-Tübingen-1 Hess 62b20 1-7.4. Inseparable from v. X-ray induced as single ♂. RK1

v^{t2}: vermilion-Tübingen-2 Hess 63c6 1-7.4. Inseparable from v. X-ray induced as single ♂. RK1

v^{t3}: vermilion-Tübingen-3 Green 63k30 1-7.4. Inseparable from v. X-ray induced as single ♂, homozygous $_{♀♀}$ inviable, possibly In(1). RK1

m^{63i}: miniature-63i Green 63i31 1-59.6. Inseparable from m. X-ray induced single ♂. RK1.

m^{63j}: miniature-63j Green 63j4 1-59.6. Inseparable from m. Complementary with m and m^{63i}; ♀♀ m^{63j}/m and m^{63j}/m^{63i} near but separable from wildtype. ♀♀ m/m^{63j} gave no m^+ crossovers in 1594 progeny indicating m^{63j} not dy allele. X-ray induced as single ♂. RK1.

y^{t1}: yellow-Tübingen-1 Hess 621 1-38.8. Inseparable from y. Homologue of y^1 of D. melanogaster, i.e. yellow body and bristles. X-ray induced as single ♂. RK1

y^{t2}: yellow-Tübingen-2 Hess 62i30 1-38.8. Inseparable from y. X-ray induced as single ♂. RK1.

y^{64a}: yellow-64a Green 64a30 1-38.8. Inseparable from y. X-ray induced as single ♂. RK1. Discarded.

y^{64b}: yellow-64b Green 64b6 1-38.8. Inseparable from y. X-ray induced single ♂. RK1. Discarded.

y^{db}: yellow-dark bristle Green 63j30 1-39.0. Yellow body color somewhat darker than that of y, and black bristles. Equivalent to lt of D. hydei and y^2 of D. melanogaster. ♀♀ y/y^{db} and lt/y^{db} are y^+ in phenotype; therefore y^{db} functional allele of both y and lt. ♀♀ y/y^{db} gave 2 y^+/4, 152 progeny with y^{db} localized to the right of y, presumably allelic to lt. RK1. Lost.

subobscura

Report of A. Pentzos-Daponte

Cherry mutants in a wild population of Drosophila subobscura in Northern Greece. During the course of a study the aim of which was the genetic analysis of the chromosomal polymorphism of D. subobscura in the wild, flies of a vernal and autumnal population had been trapped on the Sheikh-Sou hill (altitude 120 m), in the outskirts of Thessaloniki. Among the captured flies there were few individuals the eye-color of which was "cherry-like". These flies were isolated and cultured for several generations. Subsequently, virgin females from the "cherry-like" cultures were mated with males of a cherry stock. Neither the F_1 nor the F_2 of these crosses have shown any sign of segregation; hence it is assumed that the originally captured "cherry-like" flies were genuine cherry mutants.

Kaneko, A. Hokkaido University, Japan.
Differences in the shape of spermatheca of robusta group species in Japan. With one text-figure.

The identification of closely related species may be made by careful examinations of slight differences of external characters. A study of the male genitalia is to be the most satisfactory way to separate the closely related species, while the structure of female ovipositor varies to some extent. The differences in shape of the spermatheca sometimes help in identification of species.

In Japan, Drosophila belonging to robusta group had been divided into the following four species: D. sordidula, D. lacertosa, D. moriwakii and D. okadai. Kaneko, Tokumitsu and Takada separated D. sp. like sordidula from D. sordidula as a new species. Although the two species were very similar in external characters and genitalia, they were different in both karyotypes and sexual isolation. More recently, one more species very close to D. okadai was found by Kaneko, as a new member of robusta group. It is not always possible to identify accurately female members of robusta group, namely D. okadai and D. sp. like okadai, some of D. sordidula and D. sp. like sordidula and D. lacertosa, on the bases of external characters and egg-guide. The shapes of spermathecae in the six species of robusta group as mentioned above, varied remarkably from one another, as shown in the Figure. In living and alcohol-fixed specimens, the degree of transparency in spermathecae of these six species is to be arranged in descending order: 1) D. sp. like sordidula 2) D. sordidula, 3) D. moriwakii, 4) D. sp. like okadai, 5) D. lacertosa and 6) D. okadai. However, the spermatheca of D. okadai is not always transparent. Of course, after preparation (boiling the abdomen in 10% sodium hydroxide, clearing in phenol, soaking in oil of creosote, mounting by balsam), the spermathecae of all species became very pellucid and the internal structure can be easily observed. No apical round hollows were found in D. okadai, D. moriwakii and D. sp. like sordidula. Such differences in spermathecal shape as described above are helpful in identifying the female in robusta group in Japan.

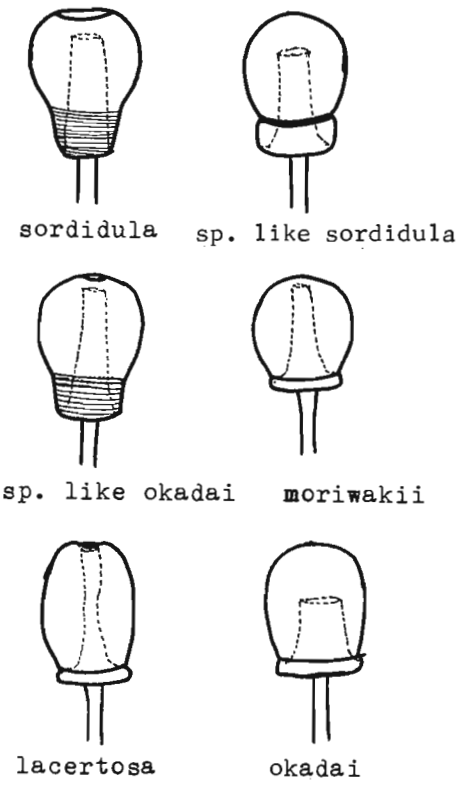

Figure: showing spermathecae of six species in robusta group

Norton, I. L. and J. I. Valencia. Oak Ridge National Laboratory. Tenn.
Genetic extent of the deficiencies formed by combining the left end of $In(1)y^4$ with the right end of $In(1)sc^9$.

The salivary gland chromosome break points of $In(1)y^4$ are between 1A8 and 1B1 on the left and between 18A3 and 4 on the right (i.e. = In(1) 1A8-B1;18A3-4). $In(1)sc^9$ is broken between 1B2 and 3 on the left and between 18B8 and 9 on the right (i.e. = In(1)1B2-3;18B8-9). Thus, the recombinant, $In(1)y^{4L},sc^{9R}$ is deficient for salivary bands 1B1 and 2 as well as 18A4 through 18B8.

This recombinant is y (y^{def} or y^4 ?) and ac^{def}, but it survives in combination with l(1) J-1. To check the genetic extent of the proximal deficiency, $In(1)y^{4L},sc^{9R}$ was made heterozygous to the following mutants: fu and fu^{59} (59.5), hdp (59.5), bkl (59.9), obl (60.1), crk (60.1), ton^2 (60.1), bk^2 (60.6), thl (60.7) sby-61 (60.8), pph-61 (61.0), smd (61.1), coc (61.5) and meg (61.9). Mutant phenotypes were obtained only with sby-61, smd, and coc. Thus Df(1)18A4-B8 is approximately one unit long; furthermore the relative position of pph-61 is in error. We wish to express our gratitude to M. G. and O.S. Fahmy for generously supplying us with the selection of mutants that so completely cover this region.

Hess, Oswald. Max-Planck Institut für Biologie, Germany. Mutation and localization studies in the Y chromosome of D. hydei.

During the growth stage of the primary spermatocytes five sites of the Y chromosome of D. hydei form loops, which in principle are organized in the same way as the loops of lampbrush chromosomes in Amphibian oocyte nuclei. According to their different morphology the five loops are called threads (F), pseudonucleolus (P), clubs (K), tubular ribbons (T), and noose (S). For the localization the long arm of the Y chromosome is divided into ten equal segments which are designated 1-10 starting at the kinetochore. The loci of the loops have been mapped as follows (fig. 1): threads in segment 10 or 9, pseudonucleolus in segment 9 or 8, tubular ribbons in segment 2 or 3, clubs in segment 1, and noose on the short arm.

By X irradiation the following X.Y-translocation chromosomes have been induced:

a) T(X,Y), 340/7: $Y^{(S,K,T)}$. w 1t

b) T(X,Y), 340/2: $Y^{(S,K)}$. w 1t

c) T(X,Y), 290/1: $Y^{(S)}$. w 1t

d) T(X,Y), 340/10: w 1t.$Y^{(F)}$

The four translocation chromosomes possess a complete euchromatic arm of the X which is marked by white light; the heterochromatic arm is lost. The translocated Y chromosome fragments differ in respect to the loops which they possess. Translocations a, b, and c have kinetochores from the Y chromosome, translocation d from the X chromosome. Males and homozygous females have normal viability; males without an additional complete Y chromosome are sterile.

Besides these X.Y-translocations one Y-autosome translocation and one Y fragment have been induced by X rays:

e) T(Y,A), vermilion-1: $Y^{(S,K,T)}$.A and A.$Y^{(P,F)}$

f) $Y^{(S,K,T)}$

The translocation chromosomes of the autosomal translocation segregate independently, because the autosomal break point lies next to the kinetochore. Both translocation chromosomes of this reciprocal translocation can, therefore, be crossed into other stocks. The Y fragment includes segments carrying the organizers for the tubular ribbons, the clubs, and the noose. The distal part of the long arm containing organizers of threads and pseudonucleolus are lost.

In addition, two mutants of one of the loops, the threads, have been induced by X-irradiation (fig.2). In the first mutant, called "tube-proximal", the normally compact proximal sections of the threads are changed into tubes, whereas the distal diffuse sections are unchanged (fig. 2b). In the second mutant, "tube-distal", the distal sections are changed into two knots of narrow tubes and the proximal compact sections are not altered (fig. 2c). It has proved possible to build up stocks in which all the males have Y chromosomes carrying the mutation tube-proximal, and stocks in which all the males have the mutation tube-distal.

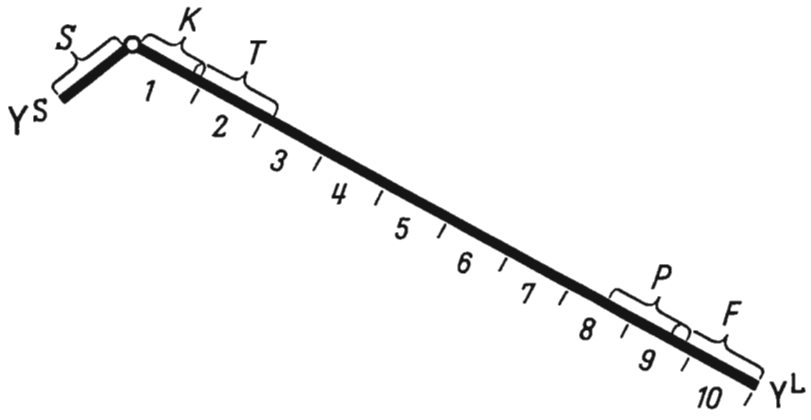

Fig. 1: Diagram of the Y chromosome of D. hydei, showing the loci of the five spermatocyte loops. Explanations: S, noose; K, clubs; T, tubular ribbons; P, pseudonucleolus, F, threads; Y^S, Y^L, short and long arm.

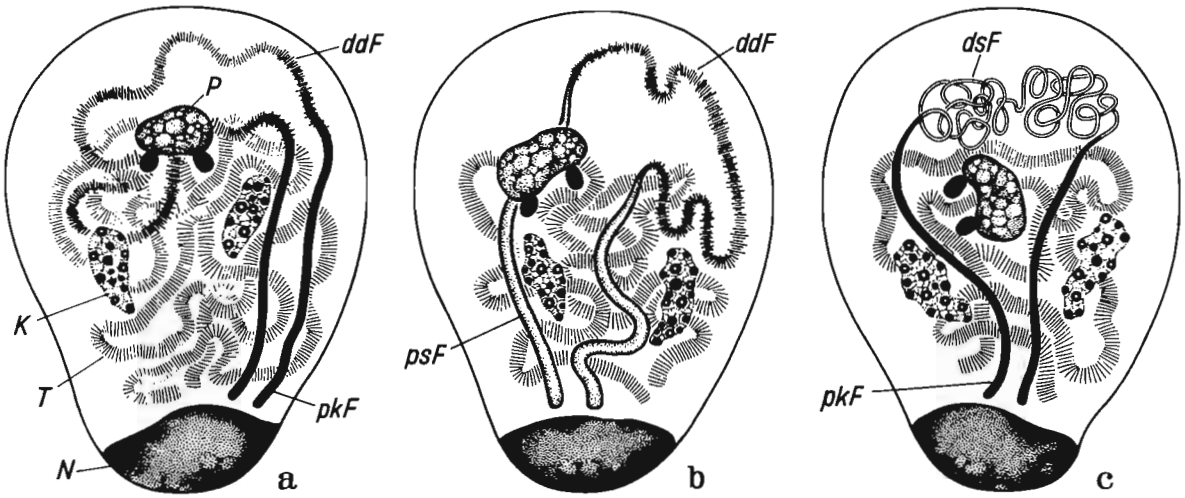

Fig. 2: Loops in the spermatocyte nucleus of normal (a), tube-proximal (b) and tube-distal, (c) male. Explanations: ddF, diffuse distal (normal), dsF, tubular distal sections (tube-distal) of the threads; pkF, proximal compact (normal), psF, proximal tubular sections (tube-proximal) of the threads; N, nucleolus. For other abbreviations, see fig. 1.

Pelecanos, M. University, Thessaloniki, Greece. The mutagenic effect of the duration of treatment with diethyl sulphate on previously starved adult males.

Previous communications have shown the importance of diethyl sulphate as a mutagenic agent. (Pelecanos 1962, Pelecanos and Alderson, 1963). Moreover, the mutagenic activity of above mentioned chemical has been studied in detail by the same workers and the data obtained are already in press.

The present report describes the results of some preliminary experiments the aim of which was twofold:

1. Study the effect of the duration of treatment upon the yield of mutations induced by the feeding of diethyl sulphate.
2. Assess whether a prior starvation treatment of the adult males would alter the frequency of induced mutations.

In all our previous experiments, newly emerged Oregon-K males were treated immediately after collection, while in the present two series of experiments, they have been either fed for 24 hours in an ordinary medium, or starved for 24 hours before the treatment. The same diethyl sulphate solution has been used throughout each experiment despite the different rate of hydrolysis over the different periods of treatment. The method used for feeding the flies has been described elsewhere (Pelecanos and Alderson 1963). After the treatment the males were tested for sex-linked recessive lethal mutations by the Muller-5 method. Each male was individually mated to two females for three days; only the first brood is recorded here.

The results are gathered in Tables I and II.

Table I shows that when a 24 hour starvation preceded the treatment, there was essentially a linear relation between the duration of treatment and the mutagenic effect. On the contrary, when newly emerged males have been fed for 24 hours in an ordinary laboratory food medium

before treatment, the data obtained (Table II) are entirely different. There is no sign of linearity as regards the relation between the treatment's duration and the yield of mutations induced; furthermore, the frequency of mutations itself is significantly lower in all three cases where data so far available, allow comparisons. (For 12 hr, 24 hr, and 36 hr. treatments, the χ^2 values and the probabilities are respectively: $\chi^2_1=4.25$ $P<0.05$, $\chi^2_2=6.6$ $P=0.01$, $\chi^2_3=12.9$ $P<0.001$).

Table I. The mutagenic response to adult feeding of diethyl sulphate, when newly emerged males have been starved for 24 hours before treatment.

	Duration of Treatment	No. of chromosomes tested	No. of lethals	lethals per cent
	12 hours	1,079	68	6.30
Concentration of	24 hours	743	84	11.30
D.E.S. 0.5%	36 hours	567	117	20.63
	48 hours	1,040	282	27.10

Table II. The mutagenic response to adult feeding of diethyl sulphate, when newly emerged males have been fed in an ordinary medium for 24 hours before treatment.

	Duration of Treatment	No. of chromosomes tested	No. of lethals	lethals per cent
	12 hours	786	17	2.16
Concentration of	24 hours	1,141	33	2.89
D.E.S. 0.5%	36 hours	987	85	8.61

Note: D.E.S. stands for diethyl sulphate (ethyl sulphate).

The most likely explanation appears to be that when previously fed, the flies do not eat enough from the treatment medium which obviously they dislike. It is also reasonable to assume that flies resist better to hunger when the treatment's duration is shorter; our results are clearly in favor of such an explanation. Thus, a previous to the treatment starvation appears to be an important factor which has to be considered when using diethyl sulphate as a mutagen by the adult feeding method.

References: Pelecanos, M. Induced öogonial lethals. DIS 36:107, 1962.
Pelecanos, M. and T. Alderson. The mutagenic response to adult feeding of diethyl sulphate in Drosophila. DIS 37:116, 1963.

Narda, R. D. Panjab University, India.

The role of various male stimuli during mating and insemination in D. malerkotliana Parshad and Pika, 1964.

The direct observations on the courtship behavior of males in Drosophila malerkotliana reveal that the male approaches the female, taps the tarsi of her middle-leg with that of his fore-leg, vibrates his wings, circles in the case of non-receptive female and postures at her rear end. To study the extent of the role played by each act in preparing the female for coition, as well as to find out the effect of light, experiments were designed eliminating the various factors one by one. Ten four-day old virgin females and 10 males of the same age but either without fore-leg tarsi or wings were kept for 48 hours in a half-pint milk bottle with standard Drosophila food at 25± 1°C in a room with fluorescent tube lighting. The light was eliminated by running the experiment in closed cardboard boxes which were early checked for the purpose. Ten trials for each set of experiment along with a control were run simultaneously. After the required period the females were checked for insemination through the presence of sperms in their spermathecae and ventral receptacles. Whereas the normal males inseminated 42% females in light, only 7.293% were fecundated in total darkness. Further, since the percentage insemination again increased to 39.796 when 1/4 pint milk bottles were used the possibility of vision as a major stimulant is ruled out. It rather helps in bringing the mates together. The removal of fore-leg tarsi in males decreased the insemination to

29.348% as against 44.706% in the control. The wingless males fecundated only 7.071% females as compared to 39.175% in the control. Again, when tarsiless and wingless males were kept with the normal females, not even a single was fecundated. These studies indicate that tapping and wing vibration of the male, of which the latter seems to be more important, are the essential factors to stimulate the female for copulation. The other acts like sight, circling and posturing seem to be of accessory nature.

Parshad, R. and K. K. Duggal. Panjab University, India. Drosophilidae of Kashmir, India.

Collections of Drosophilidae made from three places, viz., Srinagar, Pahalgam and Gulmarg, in the Kashmir Valley from June 18, 1963 to July 22, 1963, revealed twenty species, two belonging to the genus Scaptomyza and eighteen to Drosophila. Of the latter, one each belonged to the subgenera Dorsilopha and Pholadoris, eleven to the subgenus Sophophora and the remaining five to the subgenus Drosophila. Three out of these, D. (Pholadoris)ebonata, D. (Sophophora) epiobscura and D. (Drosophila) pentaspina are novo while Scaptomyza pallida, S. graminum, D. busckii, D. helvetica, D. pulchrella, D. brachynephros, D. testacea and D. curviceps are being reported for the first time from India. The frequency distribution of the various species at the three places is given in Table 1.

Table 1. Frequency distribution of the various species of Drosophilidae in the Kashmir Valley

S. No.	Name of the species	Frequency distribution at						Total no. of flies	
		Srinagar 5300 ft. above sea level		Pahalgam 7000 to 8000 ft. above sea level		Gulmarg 8900 to 9400 ft. above sea level			
		♀	♂	♀	♂	♀	♂	♀	♂
1.	Scaptomyza pallida	-	-	-	-	9	5	9	5
2.	Scaptomyza graminum	-	-	-	-	3	2	3	2
3.	D. busckii	-	-	2	1	-	-	2	1
4.	D. ebonata sp. novo	5	16	-	8	-	-	5	24
5.	D. helvetica	-	-	3	27	3	3	6	30
6.	Drosophila epiobscusa sp. novo	-	-	6	21	12	46	18	67
7.	D. bifasciata	-	-	167	744	110	221	277	965
8.	D. melanogaster	111	165	2	4	2	-	115	169
9.	D. nepalensis	7	8	58	203	-	-	65	211
10.	D. suzukii	20	82	282	1488	5	6	307	1576
11.	D. pulchrella	-	-	-	1	-	-	-	1
12.	D. ananassae	-	1	-	-	-	-	-	1
13.	D. kikkawai	28	21	-	-	-	-	28	21
14.	D. rufa	-	2	-	5	-	1	-	8
15.	D. jambulina	-	2	-	-	-	-	-	2
16.	D. brachynephros	-	-	-	1	-	-	-	1
17.	D. curviceps	-	-	1	-	1	6	2	6
18.	D. immigrans	37	42	9	6	-	-	46	48
19.	D. testacea	-	-	-	1	-	-	-	1
20.	D. pentaspina sp. novo	1	10	-	12	-	1	1	23
	Grand total	209	349	530	2522	145	291	884	3162

Bochnig, V., H. J. Belitz and H. Nöthel
Institut für Genetik der Freien Universität Berlin. Increase in frequency of a detrimental in a laboratory wild stock.

The Berlin wild-stock of D. melanogaster has been reared in our laboratory for more than 30 years. In 1958 we derived from this stock some sublines which have since been kept in mass cultures. All of them had good viability. At that time we found in one of these sublines 3 out of 21 males tested to have a detrimental in heterozygous condition in the second chromosome. Of these 3 males, 8 balanced stocks (1/Cy) were established, 6 of them behaved as lethals and 2 as semilethals. These 8 factors were allelic. But in the 28 combinations necessary to test the allelism the frequency of surviving compounds varied greatly, and was in most cases higher than expected, even in crosses between lethals. The survivors were entirely sterile, however. As these and further experiments suggest (Belitz, in preparation), the genetical background seems to determine whether the factor in question acts as a lethal or as a semilethal and whether or not the fitness of the heterozygotes is high. Beginning in 1961, fecundity, fertility and longevity in the same subline decreased, giving at last a stock with extreme low viability. A new genetical analysis yielded the following results: 85 males were tested for second-chromosome detrimentals. In 83 males each second chromosome contained a semilethal, in the other 2 one second chromosome had a lethal, the other one a semilethal. These factors, so far tested, proved to be allelic to the detrimentals found in 1958. But the newly isolated homozygous animals are fertile in some cases. Third-chromosome detrimentals were looked for in 57 males, but no one was found.

Krimbas, C. B. College of Agriculture, Greece. Further data on inversion polymorphism of D. subobscura in Greece.

Some data suggested the possibility of seasonal changes in frequencies of structural types of chromosome E in Parnes population (Greece) of D. subobscura (Krimbas, 1964, Evolution-in press). In order to investigate this point it has been decided to study big samples of this population in spring and late summer. An important spring sample (N=216) has been studied by analyzing the males' genotype, crossing them to homozygous females for Standard order in all their chromosomes.

In chromosome A,A2 showed a net increase in frequency compared to the preceeding years. J3+4 was not found this time. The chromosome E frequencies were similar to the late summer ones of last year, in this way disproving a cyclical seasonal change. In chromosome U,UI+2+7 showed a net increase in frequency, while in chromosome 0,0ST decreased.

Chromosomes A2 seem to have size decreasing capacities in regard to AST. Also UI+2+4 seem to be size increasing, while UI+2+7 size decreasing. These differences are still not statistically significant to the 5% level. Only the genotypes for chromosome U showed a net departure from Hardy-Weinberg ($0.01 < P < 0.001$) in having heterozygotes more than expected and less homozygotes. Fitnesses have been estimated (ratio between observed and expected) for the six main genotypes: UI+2/UI+2=0.42,UI+2+4/UI+2+4=0.78,UI+2+7/UI+2+7 = 0.25, UI+2+4/UI+2= I.2I, UI+2+7/UI+2+4 = I.05,MMM UI+2+7/UI+2 =I.46. A fitness surface has been constructed with these values, which showed a maximum at the point UI+2 freq=0.35;UI+2+4 freq=0.40 and UI +2+7 freq=0.25, $\bar{W}$=0.9962. The actual population lies quite near to the maximum (UI+2 freq= 0.32,UI+2+4 freq=0.48,UI+2+7 freq=0.20, $\bar{W}$=0.9929). This shows that our fitness estimates are not very far from reality.

Fujii, S., Kanehisa, T. and Ohnishi, M.
Kobe University, Japan. Biochemical analysis of "Freckled-type melanotic tumor" inducible fraction.

After the co-working of Kanehisa with Prof. Barigozzi and co-workers, Universita Di Milano, the 0.3 M NaCl-eluted fraction which can induce "Freckled-type tumor", from D.E.A.E. cellulose column-chromatography, was tested for the presence of nucleic acids. This fraction has a maximum adsorption at around 280 mμ and minimum at 260 mμ. So far from the fraction from 15 gr. wet weight flies, purified by some organic solvents, DNA or RNA could not be found in this fraction. There is no incorporation of P^{32} in this fraction, though the other two fractions from the chromatography incorporate this isotope.

Makino, S., E. Momma, A. Kaneko, T. Tokumitsu and T. Shima. Hokkaido University, Japan. Drosophilidae from seven localities in Hokkaido and Aomori prefecture, summer in 1964.

Collections were made during the period July to August, 1964 in five different localities of Hokkaido: Esan, Doya, Nisama, Gunnai and Toikanbetsu, and in two localities of Aomori prefecture: Okuyagen and Imabetsu. A total of 7,188 specimens were collected by the use of fermented banana, at one hour intervals from sunrise to sunset for three successive days. They were represented by 37 species as given in the accompanying table. Among them, new species designated as Drosophila sp. like sordidula and Drosophila sp. like okadai were found to occur in robusta group. Remarkable was the fact that D. lacertosa constituted 80.9 per cent of all flies collected in Toikanbetsu, the northern part of Hokkaido.

Numerical data of Drosophila species collected in seven localities of Hokkaido and Aomori prefecture, summer 1964.

Localities / Species	Imabetsu	Okuyagen	Esan	Gunnai	Doya	Nisama	Toikanbetsu	Total
Amiota variegata	-	1	-	5	1	1	-	8
Leucophenga quadripunctata	2	-	-	-	-	-	-	2
L. ornatipennis	1	-	-	-	-	-	-	1
L. sp.	1	-	-	-	-	-	-	1
Chymomyza sp.	2	-	-	-	-	-	-	2
Parascaptomyza pallida	-	10	-	-	-	2	-	12
Scaptomyza apicalis	1	-	-	2	-	2	-	5
Drosophila alboralis	-	1	-	-	-	-	-	1
D. sexvittata	-	-	-	1	-	-	-	1
D. trivittata	1	-	-	-	-	-	-	1
D. histrioides	12	40	1	2	8	1	-	64
D. coracina	48	16	-	82	59	186	-	391
D. bifasciata	7	33	-	-	31	44	52	167
D. hilvetica	-	-	-	-	-	-	1	1
D. suzukii	-	-	-	2	-	-	-	2
D. lutea	2	-	-	-	-	-	-	2
D. magnipectinata	-	-	-	1	-	-	2	3
D. auraria race A	12	-	70	-	23	42	-	147
D. auraria race B	90	29	76	626	1208	390	2	2421
D. auraria race C	-	-	-	-	3	-	-	3
D. brachynephros	-	-	1	22	-	22	1	46
D. unispina	2	-	-	-	-	-	1	3
D. nigromaculata	10	-	2	100	91	304	19	526
D. testacea	24	15	1	21	23	68	18	170
D. histrio	4	21	-	4	-	2	-	31
D. funebris	-	-	-	-	9	-	5	14
D. pengi	-	-	-	-	1	-	-	1
D. ezoana	-	-	-	1	-	2	13	16
D. sordidula	-	3	-	6	-	2	-	11
D. lacertosa	36	59	5	259	110	12	2043	2524
D. moriwakii	12	109	-	49	119	12	127	428
D. okadai	-	2	-	68	-	-	62	132
D. sp. like sordidula	8	6	-	-	-	-	-	14
D. sp. like okadai	-	-	-	-	-	-	29	29
D. sp. I	1	-	-	-	-	-	-	1
D. sp. II	-	-	-	1	-	-	-	1
D. sp. III	-	-	-	-	2	-	-	2
D. sp. IV	-	-	-	-	-	3	-	3
D. sp. V	-	-	-	-	-	-	1	1
Total	276	345	156	1252	1688	1095	2376	7188

Solima-Simmons, Angela and Howard Levene. Columbia University. Effect of age and temperature on matings of Drosophila paulistorum.

Previous experiments (Malogolowkin, Solima-Simmons and Levene, in press) led us to check on the effect of temperature and age on sexual isolation. The experiment was performed using Ellens (1957) Chambers with two strains (Simla H and Elena L) of the Amazonian and one (Apoteri Y) of the Orinocan race, all tested against Bahia 6 (Andean-Brasilian) kept for several years in the laboratories.

The flies were aged 5, 10 or 20 days and tested at 20°C and 25°C. The percentages of heterogamic matings are:

days	Simla H			Elena L			Apoteri Y		
	5	10	20	5	10	20	5	10	20
temp.									
20°C	2	0	13	0	5	8	30	33	36
25°C	0	0	0	5	0	3	21	16	17

There seems to be little effect of age but lower isolation at lower temperature.

Previous values obtained in the experiment cited above (ca. 10 days, 25°C) were 0, 3, 10 percent respectively.

For the actual percentage of total matings (heterogamic and homogamic) there is comparatively little effect of age and temperature.

A comparison of the total matings of both the new and the old data give the following percentages:

	Simla H vs Bahia 6		Elena L vs Bahia 6		Apoteri Y vs Bahia 6	
Previous	20	70	18	65	37	97
Present	58	61	63	81	69	79

The results could be explained by a better adaptedness of the Orinocan and especially the Amazonian flies to laboratory conditions. Their mating activity is now closer to the older Bahia 6 strain.

Research supported by NSF Grant NSF-GB-1906.

Miller, Dwight D., Ralph L. Sulerud and Neal Westphal. University of Nebraska. Determinations of D. athabasca Y chromosome types from new localities.

As pointed out by Miller and Roy (1964), D. athabasca has three widespread Y chromosome types: large J-shaped Type 1, medium near-V Type 2, and large V-shaped Type 3 (with X^1 and X^2). Compared with the X-chromosome of this species, which in primary spermatocytes has segments in ratio of about 5:1:4 (Miller and Stone, 1962), Type 1 may be described as having a segment ratio of 5:1:2, 5:3, or 5:2; Type 2 as 3:1:2 or 3:3; and Type 3 as essentially 5:1:4 (like the X). Recent collections of our own, supplemented by those of others who have kindly provided us with specimens and cultures, have made it possible to extend observations of Y chromosome types in this species (for new material we are grateful to Miss Judith Barkley and Dr. Edward Novitski, University of Oregon; Professor Th. Dobzhansky and Mrs. Olga Pavlovsky, Rockefeller Institute; and Dr. Philip T. Ives, Amherst College). The widespread, generally western Type 1 has now been determined from additional western localities: British Columbia (Okaneghan, Victoria), Idaho (Boise), Minnesota (Hallock, Halstad), New Mexico (Raton Pass), Oregon (Eugene, Siuslaw National Forest), and Washington (Mt. Baker National Forest, San Juan Islands). Type 2 has been encountered at Amherst, Massachusetts, and Northfield, Minnesota. A new type, designated Type 5, has been found in material from Seguim Bay, Washington (Olympic Peninsula). This is a nearly V-shaped chromosome intermediate between Types 2 and 3 (segment ratio 4:1:3 or 4:4).

Fedoroff, N. V. and R. D. Milkman. Syracuse University. Induction of puffs in Drosophila salivary chromosomes by amino acids.

In view of the role of small molecules in enzyme induction and the relationship of gene activation to enzyme induction and to puffing, we have been investigating the effects of amino acids on puffing in D. melanogaster salivary chromosomes. Glands are excised from third-instar larvae grown at 18° and incubated separately in Ringer's solution containing the test substance and in plain Ringer's solution. After a given interval, puffing patterns are compared. The production of a puff with 0.03M tryptophan at site 68D on III-L has been reported (Biol. Bull. 127, in press). Puffing at 68D is never observed in controls. It was also reported that methionine and tyrosine appear to have no effects. A study of the comparative effects of d- and l-tryptophan also showed differences. However, although one might expect that a puff would appear either regularly or not at all, d-tryptophan causes smaller and less frequent puffs (20% vs. 85% of nuclei observed) than l-tryptophan. In the case of both methionine and tryptophan there appears to be an increase in the frequency and extent of puffing at site 50F on II-R; this puff, however, shows relatively high variability, and some uncontrolled factor may be involved. In similar experiments, l-histidine produced no significant changes in the puffing pattern. The results are summarized in table below.

Amino acid added	Concentration (moles/liter)	Hours in Ringer's	Results
l-histidine	0.05	1	No significant change
"	0.10	1	"
l-methionine	0.06	1	50F ?
"	0.20	1	50F ?
l-tryptophan	0.03	1/2	No change
"	0.03	1	Puff at 68D (50F ?)
d-tryptophan	0.03	1	Small puff observed at 68D occasionally
l-tyrosine	0.0022	1	No significant change

These results are differential results: immersion in Ringer's solution for the durations noted produced a variety of changes in the puffing pattern. Only those ascribable to the amino acids are reported. Absence of visible puffing does not, of course, imply the absence of local changes in rates of RNA synthesis. The present survey continues in an attempt to learn more about the turning on and off of genes. (Work supported by NSF Grant G-24023 and NSF Undergraduate Research Participation Project).

Slizynski, B. M. Institute of Animal Genetics, G. B. Differential X-ray sensitivity of spermatogonia in Drosophila melanogaster.

Slizynska found that in regard to the effects of irradiation there was a profound heterogeneity between males as well as between the germ cells of individual males in brood. Heterogeneity between the males would arise if at the time of treatment some males have more spermatogonial mitoses than the others. Heterogeneity between the germ cells of individual males can be reduced to the fact that in the testis some spermatogonial cells are in a susceptible stage (metaphase) while the majority of cells are in a resistant stage. The question was studied cytologically and the following results were obtained.

Among 129 2-3 days old males of y w stock there were 25 males in which there were no mitotic divisions in the testes, 23 males had mitoses in the apex cells and 81 males had spermatogonial mitotic divisions in the cysts. Among the males of this last category there were 51 with 4 or less dividing spermatogonial cells, 14 males with 5-7 divisions per testis, and 16 males with 8-16 divisions of spermatogonial cells per testis. Thus the highest sensitivity to treatment in brood is expected to occur in about 12% of males. This figure will be doubled if two last classes of frequency of mitoses are taken as resulting in high sensitivity.

Kojima, K. and M. Dalebroux. North Carolina State. A procedure for analyzing three-point test data when one gene shows low penetrance.

During the Drosophila Research Conference at Madison, Wisconsin (1964), Dr. R. Hillman of Temple University called Kojima's attention to an article in DIS (Tsukamoto; DIS No. 38:91-93). The article deals with an estimation procedure of recombination fractions in three-point test with a low penetrance gene. Dr. R. Hillman applied Tsukamoto's method to his data, and obtained an uninterpretable result.

An algebraic examination starting from the tables in Tsukamoto's article leads his four formulae to the following results:

$$\text{Formula (1)} = \frac{100}{1 + \sqrt{\frac{R_1}{100-R_1}}} \quad \text{, instead of } R_1,$$

$$\text{Formula (2)} = \frac{100}{1 + \sqrt{\frac{R_2}{100-R_2}}} \quad \text{, instead of } R_2,$$

$$\text{Formula (3)} = 50\% \quad \text{, instead of } R_1,$$

and

$$\text{Formula (4)} = \frac{100}{1 + \sqrt{\frac{(100-R_1)\,(100-R_1+yR_1)}{R_1\,(100y+R_1-yR_1)}}} \quad \text{, instead of } R_2.$$

Thus, it seems that the four formulae by Tsukamoto do not give the estimates of recombination fractions as intended. The rest of this note will deal with a correct procedure for estimating two recombination fractions and one penetrance value in three-point test. The formulae for approximate sampling variances of the estimates will be given.

Consider a stock with mutants a, b, and x at three loci. The x is assumed to have a low penetrance. Denoting their wild-type alleles by +'s, an F_1 hybrid of this stock with a wild stock is a/+, b/+ and x/+, and its phenotype is of wild type. When the F_1 (the sex of this individual must be female in the case of Drosophila) is backcrossed to the triple mutant stock, segregation occurs at the three loci. There are eight phenotypic classes. The first problem is to determine whether locus x is located between the a and b loci or at the outside of the a-b segment. A test for this distinction can be performed by the following procedure.

As in Table 1, pool the observed numbers of animals according to four paired phenotypic classes. The difference, YU-ZX, is expected to be zero when the order of the loci is x-a-b and a positive value when the order is a-x-b. The magnitude of the latter value is given at the bottom of Table 1, and the symbols used will be defined later. The departure of YU-ZX may be tested by the following X^2 statistic with one degree of freedom:

$$X_1^2 = 4(YU - 1/2\ M)^2/M$$

where M = YU + ZX.

After the order of the three loci is determined, one can proceed to estimate the values of recombination fractions and penetrance. Symbols to be used are as follows:

p : the recombination fraction between the left-most and center loci.
q : the recombination fraction between the center and right-most loci.
y : the fraction of x/x individuals which appear as wild type (penetrance index).

x-a-b arrangement:

All phenotypic classes, their expected and observed numbers are given in Table 2. Compute the following;

$$A = \frac{n_1 + n_2 + n_3 + n_4}{N}, \quad B = \frac{n_1 + n_2 + n_5 + n_6}{N}, \quad Q = \frac{n_2 + n_4 + n_6 + n_8}{N}$$

The expected values of these quantities are

$$E(A) = p\,(1-y) + 1/2\,y$$

$$E(B) = q$$

$$E(Q) = 1/2\,(1 + y)$$

Thus, the estimates are given by

$$\hat{y} = 2Q - 1$$

$$\hat{p} = (2A-\hat{y})/2(1-\hat{y}) \text{ for the x-a segment}$$

$$\hat{q} = B \text{ for the a-b segment}$$

In other words, y and q can be estimated directly, and the estimate of p is compounded with the estimate of y. The sampling variances for the $\hat{q}$ and $\hat{y}$ are computed by

$$B(1-B)/N \quad \text{for } \hat{q}$$

$$4Q(1-Q)/N \quad \text{for } \hat{y}$$

The sampling variance for the p is more complicated, but approximately given by

$$\frac{1}{N(1-\hat{y})^2}\left[A(1-A) + \frac{(1-2A)^2}{(1-\hat{y})^2} \cdot Q(1-Q) - \frac{2(1-2A)}{(1-\hat{y})}\left\{\frac{(n_5+n_7)(n_2+n_4)}{N^2} - \frac{(n_6+n_8)(n_1+n_3)}{N^2}\right\}\right]$$

a-x-b arrangement:

As in the case of x-a-b, the phenotypic classes, their expected and observed numbers are given in Table 3 for this case. Compute the following;

$$A = \frac{n_1 + n_2 + n_3 + n_4}{N}, \quad B = \frac{n_1 + n_2 + n_5 + n_6}{N}, \quad Q = \frac{n_2 + n_4 + n_6 + n_8}{N}$$

Their expected values are

$$E(A) = p(1-y) + 1/2\,y$$

$$E(B) = q(1-y) + 1/2\,y$$

$$E(Q) = 1/2(1 + y)$$

In this case, the estimates of both p and q are compounded with the estimate of y, and they are

$$\hat{y} = 2Q-1$$

$$\hat{p} = (2A-\hat{y})/2(1-\hat{y}) \quad \text{for the a-x segment}$$

$$\hat{q} = (2B-\hat{y})/2(1-\hat{y}) \quad \text{for the x-b segment}$$

The sampling variance of $\hat{y}$ is given by $4Q(1-Q)/N$, and the approximate sampling variances of $\hat{p}$ and $\hat{q}$ are

$$\frac{1}{N(1-\hat{y})^2}\left[A(1-A)+\frac{(1-2A)^2}{(1-\hat{y})^2}\cdot Q(1-Q)-\frac{2(1-2A)}{(1-\hat{y})}\left\{\frac{(n_5+n_7)(n_2+n_4)}{N^2}-\frac{(n_6+n_8)(n_1+n_3)}{N^2}\right\}\right]$$

and

$$\frac{1}{N(1-\hat{y})^2}\left[B(1-B)+\frac{(1-2B)^2}{(1-\hat{y})^2}\cdot Q(1-Q)-\frac{2(1-2B)}{(1-\hat{y})}\left\{\frac{(n_3+n_7)(n_2+n_6)}{N^2}-\frac{(n_4+n_8)(n_1+n_5)}{N^2}\right\}\right],$$

respectively.

(This work was partly supported by Public Health Research Grant GM 11546 from the Division of General Medical Sciences. The junior author is a member of the European Atomic Energy Community).

Table 1. x-a-b or a-x-b

Paired Phenotypes	x++ and +ab	x+b and +a+	xa+ and ++b	xab and +++	Total
observed numbers	X	Y	Z	U	N

$E(YU-ZX)=0$ for x-a-b

$E(YU-ZX)=p(1-p)(1-2q)(1-y)N^2$ for a-x-b

Table 2. x-a-b arrangement

Phenotypes	Expected Numbers	Observed Numbers
x + b	1/2 pq(1-y)N	n_1
+ a +	1/2 pq N + 1/2q(1-p)yN	n_2
x + +	1/2 p(1-q)(1-y)N	n_3
+ a b	1/2 p(1-q)N+1/2 (1-p)(1-q)yN	n_4
x a +	1/2 q(1-p)(1-y)N	n_5
+ + b	1/2 q(1-p)N+ 1/2pq yN	n_6
x a b	1/2(1-p)(1-q)(1-y)N	n_7
+ + +	1/2(1-p)(1-q)N+ 1/2 p(1-q)yN	n_8
Total	N	N

Table 3. a-x-b arrangement

Phenotypes	Expected Numbers	Observed Numbers
+ x +	1/2 pq(1-y)N	n_1
a + b	1/2 pq N + 1/2(1-p)(1-q)yN	n_2
+ x b	1/2 p(1-q)(1-y)N	n_3
a + +	1/2 p(1-q)N + 1/2 q(1-p)yN	n_4
a x +	1/2 q(1-p)(1-y)N	n_5
+ + b	1/2 q(1-p)N + 1/2 p(1-q)yN	n_6
a x b	1/2 (1-p)(1-q)(1-y)N	n_7
+ + +	1/2 (1-p)(1-q)N + 1/2 pq yN	n_8
Total	N	N

Spiess, E. B. University of Pittsburgh. D. persimilis from Humboldt County, Calif.

During July, 1964, persimilis was collected from the Redwoods Forest of Humboldt County, California (town of Weott). With 188 chromosomes identified the frequencies of arrangements from chromosome III are as follows: Klamath 78.2%, Mendocino 13.8%, Humboldt* 3.8%, Standard 1.1%, Cowichan 1.5%, Whitney 1.1%, and Unknown* 0.5%. We are tentatively designating the more common arrangement of two heretofore rare or unknown arrangements as "Humboldt" since we discovered it first in our cultures, took photographs with its heterozygote KL/HU*, and corresponded with Professor Dobzhansky (Rockefeller Institute) as to the arrangement's identity with that described by him (with C. Epling, 1944, Carnegie Institute Washington Publ. #554). Professor Dobzhansky agreed that this arrangement was likely to be his Humboldt arrangement. The exact banding pattern will be reported soon, but briefly it is an independent inversion of the Standard sequence of approximately the same length as the well known KL but displaced more proximally by about 15 bands than KL. The "Unknown*" arrangement however is a single step inversion from KL (overlapping) and had not been observed at the time of our correspondence with Dobzhansky, so that the correct naming may be decided later. In fact this latter "Unknown" may well be identical with Dobzhansky's Humboldt and the arrangement we designated above as "Humboldt*" may be a new arrangement. In either case, the frequency of the latter is much higher than observed before (3.8%); if it is truly a newly formed arrangement, it can hardly be ephemeral to the population.

Whitten, M. J. University of Tasmania. Factors affecting penetrance of an eye mutant in D. melanogaster.

Penetrance of witty (DIS 38:31) in the homozygous state depends both on background modifiers and the environment. A novel method, involving the truncated normal distribution, and utilizing the fact that asymmetric flies are produced, has been applied to measure the genetic and environmental contributions to penetrance.

Initially it was thought that wi arose spontaneously in a Cy j stock. However the evidence suggests that all individuals in the stock were homozygous for wi and that penetrance was reduced to near zero by the large complement of modifiers reducing the activity of wi.

Removal of certain modifiers on the same linkage group as wi results in the dominant form. Penetrance of this form is then dependent on modifiers on Chromosome 3 and 1 and (or) 4.

It is believed that wi first occurred as a dominant in a natural population and a system of modifiers (dominance modifiers) was selected to reduce it to recessivity. Subsequently the penetrance of wi was reduced to near zero by the accumulation of further modifiers (penetrance modifiers). It has not yet been determined whether the two classes of modifiers are mutally exclusive.

Carmody, George. Columbia University.
Two unusual strains of the D. willistoni sibling species group.

Two new strains of the D. willistoni sibling species group have been found that provide a potential bridge between the gene pools of D. equinoxialis and D. paulistorum. The first strain was collected near Girardot, Colombia. It produces fertile offspring with the following three strains of D. paulistorum: Angra (Andean-South Brazilian Race), Maranguape (Andean South-Brazilian Race), and Salvador (Centro-American Race). This last cross produces fertile offspring only when Girardot females are mated with Salvador males; the reciprocal cross produces no offspring at all. No offspring are produced in crosses of Girardot with 13 geographical strains of D. equinoxialis. However, crosses with three other D. equinoxialis strains (Belém 0, Içana, and Puerto Rico) produce offspring which are sterile - both males and females.

The second strain, called Belém K, was collected in Belém, Northeastern Brazil. It is completely interfertile with the Girardot strain. Belém K produces fertile offspring with at least six geographical strains of D. equinoxialis: Belém 0, Içana, Puerto Rico, Simla I, Skeldon, and Tefé. Crosses of Belém K with ten other strains of D. equinoxialis give no offspring. Belém K is interfertile with only one strain of D. paulistorum: Maranguape. No other cross between Belém K and D. paulistorum produces even sterile offspring.

Preliminary sexual isolation data that was gathered using the "male choice" method shows both strains about as isolated from D. paulistorum as from D. equinoxialis, with the Girardot strain slightly more highly isolated from D. equinoxialis than from D. paulistorum and the Belém K strain slightly more isolated from D. paulistorum than from D. equinoxialis. This high sexual isolation together with the fact that these two strains are geographically separated suggests that those crosses which can be obtained in the laboratory almost certainly do not occur in nature.

The salivary chromosomes of these two strains show polymorphism for inversions in chromosomes II and III. The banding pattern of chromosome III is unique in each of these two strains and both are different from that of either D. paulistorum or D. equinoxialis. In addition the salivary chromosomes of the hybrids have many inversions in all three chromosomes. Chromosome pairing in these hybrids is good, with only short regions apparently unpaired.

Mapping of the inversions and unpaired sections is currently in progress.

Research supported by NSF Grant GB-1906 to Prof. Howard Levene and NIH Grants 2TI-GM216-05 and 5TI-GM216-06.

Mukai, T. National Institute of Genetics, Japan.
Position effect of spontaneous mutant polygenes controlling viability in D. melanogaster.

Spontaneous polygenic mutations affecting viability were accumulated under the minimum pressure of natural selection in 104 second chromosomes which were derived from a single second chromosome. In Generations 25, 32, 52, and 60, homozygous viabilities of these chromosome lines were estimated. In addition the viabilities of flies carrying random combinations of these chromosomes were estimated and the correlation coefficient between the sum of homozygote viabilities and the corresponding heterozygote viability was calculated for each generation. The estimated values were positive and highly significant.

On the other hand, in Generations 32 and 60, the viabilities of heterozygotes between these experimental chromosomes and a chromosome supposed to be identical to the original chromosome were estimated and the correlation coefficient between homozygote and heterozygote viabilities was calculated for each generation. The results showed significantly negative values. From these results it can be said that newly arising mutant polygenes clearly show overdominance in homozygous genetic background when the chromosomes carrying them are combined with the original normal chromosome, but are heterozygously deleterious in trans-phase heterozygotes. These phenomena are significant in clarifying the mechanism by which natural populations carry genetic variation.

The results are highly reliable because those conclusions were drawn on the basis of counting approximately 3 million flies. (This work has been supported by PHS grants GM-7836 and RH-34).

Sakaguchi, B. and S. Kobayashi. National Institute of Genetics, Japan. Morphological observations of "sex-ratio" agents of D. willistoni and D. nebulosa.

It has been demonstrated by Poulson and Sakaguchi (1961) that the infectious agents responsible for the maternally transmitted "sex-ratio" condition (SR) in D. willistoni and D. nebulosa are minute spirochetes, presumably Treponema. In order to investigate the form, fine structure and life cycles of the SR agents, the SR spirochetes, electron microscopical observations are now underway. The results so far obtained may be briefly described.

Females of SR strains of D. willistoni and D. nebulosa were used as a source of the SR spirochetes. The hemolymphs taken from the females were either diluted with Drosophila Ringer or treated with 2% phosphotungstic acid (for negative stain). The diluted hemolymphs were placed on grids and then fixed by osmium tetroxide vapor.

The forms of the SR spirochetes are very variable; the two species of the spirochetes cannot be clearly distinguished (Figure 1). The size of the spirochetes can be estimated to be of

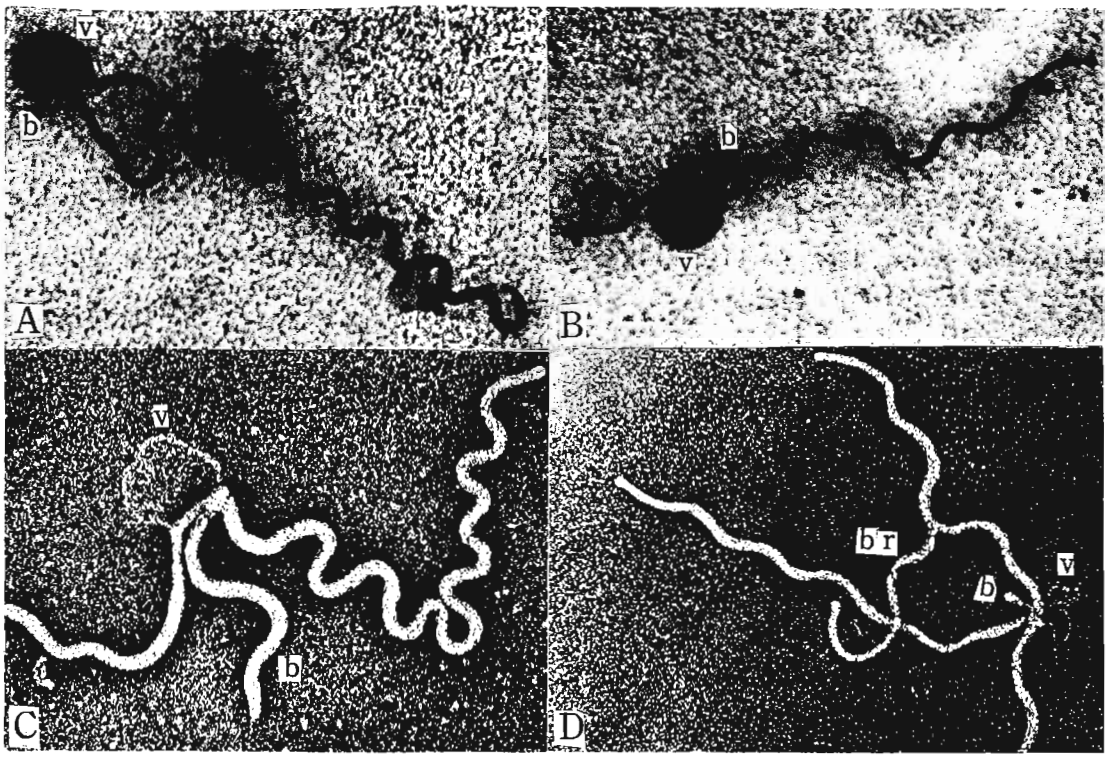

Figure 1. Electron micrographs of SR agents of D. willistoni and D. nebulosa. A and C: SR agent of D. willistoni (A: fixed by osmium vapour, X 12,000; C: negative stain, X 16,000). B and D: SR agent of D. nebulosa (B: Fixed by osmium vapour, X 12,000; D: negative stain, X 13,000). b: bud, br: branch, v: vesicle.

the order of about 8 to 16 microns in length and 0.15 to 0.08 microns in diameter. In most instances the spirochetes show irregular undulations running along the filaments, as represented in the photographs. A striking morphological feature is a vesicle ranging from 0.8 to 1.2 microns in diameter. These vesicles may occur at any point along the filament. Furthermore, they form one to four minute granules (buds) which may develop into young spirochetes. It is suggested that the vesicles play an important role in the cycle of duplication.

It is interesting that the filaments are occasionally branched. This feature has so far been found in no other spirochete species. In some spirochetes treated with 2% ammonium sulfate the filament appeared to be double coiled, each coil approximately 5 millimicrons thick.

(This work has been supported by Grant GM-10238 from U. S. National Institute of Health).

Kessler, Seymour. Columbia University. Mating speed and mating preference in two species of Drosophila.

Ethological (sexual) isolation in Drosophila is believed to result from an interaction between two factors; sexual drive and mating discrimination (Mayr, 1946; Spieth, 1951); the exact nature of the relationship between these two factors, however, has never been clearly understood. It has been supposed by Mayr (1963, p. 101) and others that flies, particularly females, that are slow to mate would exercise greater discrimination in mate selection than fast ones. This implies that an inverse relationship may exist between mating speed and discriminatory ability. This position has been tested by means of multiple-choice sexual isolation tests utilizing mutant strains of D. pseudoobscura (gl) and D. persimilis (or) that have been selected both for slow and fast mating speed.

Preliminary results show no support for this position; the isolation indices for tenth generation flies being 0.97 for the fast line, 0.89 for the slow line and 0.86 for controls. Further studies are currently in progress. Research supported by NSF Grant NSF-GB-1906 to Professor Howard Levene and NIH Grants 2TI-GM216-05 and 5TI-GM216-06.

References: Mayr, E. 1946. PNAS, 32:57-59; 1963. Animal Species and Evolution. Spieth, H. T. 1951. Behaviour, 3:105-145.

Kitagawa, O. Tokyo Metropolitan University. Japan. Heterozygous effect of induced recessive lethals accumulated on second chromosomes of D. melanogaster.

More than sixty lethal free second chromosomes were extracted from the heterozygous population of D. melanogaster which was maintained for six months. Male flies with normal viability when homozygous were irradiated by 500 r of X-rays. Thirty four second chromosomes carrying induced recessive lethals were obtained. Double and triple lethals were accumulated in cis-phase on second chromosomes through recombination of females with two or more lethals in trans-phase. In this experiment, 40 normal, 34 one, 15 double and 9 triple lethal strains were used. Viability of wild phenotype flies were determined by the Cy-Pm technique. Following results are obtained:

No. of lethals per zygote	No. of crosses	Mean no. of flies per cross	Preadult viability (Cy/Pm = 1.0000)
0	64	417.7	1.0100±.01924
1	108	702.6	.9942±.01587
2	122	364.7	.9842±.01379
3	102	465.2	.9728±.01352
4	51	523.3	.9580±.01839
5	20	494.4	.9377±.02934
6	6	632.7	.9177±.06658

Hand in hand with the increase of the number of lethals per zygote, the viability decreased rapidly. This synergistic interaction between lethals is very relevant to the problem of the maintenance of genetic loads in natural populations.

Banks, J. L. The Ohio State University. Surface sterilization of D. melanogaster eggs.

To establish axenic cultures of D. melanogaster or to harvest early embryonic stages aseptically, surface sterilization of eggs is necessary. A technique similar to that of Mitsuhashi and Maramorosch, 1963, using 0.1% Hyamine's solution for a period of time up to 5 min. does not interfere with embryonic development.

Reference: Mitsuhashi & Maramorosch. Aseptic Cultivation of Four Virus Transmitting Species of Leafhoppers (Cicadellidae) Contrib. Boyce Thompson Inst. 22(4):165-173, Oct.-Dec.1963.

Plaine, H. L. and Sister M. Baptista Aubele. Ohio State University. Occurence of the erupt effect in the Su-er tu bw; er^+ $Su\text{-}tu^+$ strain of D. melanogaster.

Three series of eggs and larvae of the Su-er tu bw; er^+ $Su\text{-}tu^+$ strain were collected over a twenty-four hour period on the surface of corn-meal-dextrose media and exposed to X-irradiation at ±12 hours of age. The disks of media containing irradiated eggs and larvae were placed on the surface of the same type of media in standard size milk bottles and incubated at 25±1°C. Eyes of adult flies were scored upon emergence as extreme erupt, weak erupt, or normal.

Irradiation was carried out with a Norelco MG 150/10 industrial X-ray unit having a 2.5 mm beryllium window and a maximum output of 150 kv. The delivered dose in air for all series was 1000 r (±5%) at 140 kv, 5 ma, distance 36 cm. Series 1 and 2 received 108-116r/min. with additional filtration of 0.019 mm aluminum, while series 3 was irradiated inside a lucite chamber with no additional filtration at a dose rate of 37-39 r/min.

Evidence of the phenotype characteristic of extreme erupt was found consistently in all of the irradiated series (Table 1), while all series subjected to X-irradiation differed significantly from the non-irradiated controls of the same strain. Further studies as to the nature of this erupt response are in progress.

Table 1: The erupt eye effect in irradiated and non-irradiated series of the Su-er tu bw; er^+ $Su\text{-}tu^+$ strain.

Treatment		Total	Phenotype of eyes (%) Normal	Extreme erupt	Total erupt
X-irradiated 1000 r	series 1	1027	74.6	5.4	25.4
	series 2	594	74.1	5.2	25.9
	series 3	1087	75.8	6.2	24.2
non-irradiated controls		1511	99.7	0	0.3

Taira, T. National Institute of Genetics. Japan. Soluble nucleotides in Drosophila.*

An attempt was made to make a map of soluble nucleotides more complete /nucleotide composition as a function of stage of development/. Among many analytical techniques previously tested, the following combination technique was found to be best: hot 50% alcohol for an extraction, charcoal as an adsorbant for fractionation and paper chromatography and electrophoresis for separation and identification. The treatment with a charcoal adsorbant was done in the cold. A final quantity of nucleotides of at least 0.1 micromoles is required for a clear identification.

In the course of development of Oregon-R, the total relative amounts of nucleotides per dry weight in larvae, pupae and adults are given as 293, 39 and 336 respectively. For convenience the total density units of total extracts at each stage of development is expressed as: $(E_{260} - E_{310})$ x (total volume) /E=extinction/ as being equal to the relative total quantity of nucleotides. As for the individual nucleotides, uridine diphosphate acetyl-glucosamine is detected in pupae and adults, but not in larvae. Oligonucleotides are found at stages from larvae to mid-pupae, but not in adults. Nucleosides, such as uridine and inosine, are detectable in larger amounts in larvae than in adults, but are not found in pupae. Soluble nucleotides in larvae are scarcely detectable. Therefore, it is clear that the value 293 in larvae is represented mostly by non-nucleotide substances, namely the nucleosides described above and other /nucleosides/ in small quantities. On the contrary, in adults soluble nucleotides represent the majority of the value 336. These results suggest: (1) soluble nucleotides in Drosophila begin to appear in pupae but are not found in larvae, and (2) the correlation between the appearance of uridine diphosphate acetyl glucosamine and the disappearance of oligonucleotides in pupae may be very close to cell differentiation of adult organs. In the near future these data and those of typical mutant strains of Drosophila will be published as a map of soluble nucleotides.

*Editing was required to make this paper more readable in English.

Lucchesi, J. C., S. Mills and R. Rosenbleeth. University of Oregon. Relative frequencies of induced TM and RA compound X chromosomes in D. melanogaster females.

Lindsley (1958) has proposed that in males pairing and subsequent (spontaneous) exchange involving X and Y heterochromatin "are conditioned by non-homologous but heterochromatin-specific forces." On the other hand, Lucchesi (1964) has offered arguments supporting the view that in females pairing and subsequent (induced) exchange involving an attached-X and Y chromosomes are conditioned by homology. The present experiment was undertaken to further investigate pairing affinities of X and Y heterochromatin.

Females of the following constitution were obtained:

Ia. $X{\cdot}Y^{L}$, $In(1)sc^{8L}$, EN^{R}, y^{+} f cv $y{\cdot}Y^{L}/X\ Y^{S}{\cdot}Y^{L}$, y $Y^{S}{\cdot}Y^{L}$ B^{S}

b. " $/X\ Y^{L}{\cdot}Y^{S}$, y $Y^{L}{\cdot}Y^{S}$ y^{+}

IIa. $Y^{S}X{\cdot}Y^{L}$, In(1)EN, Y^{S} B f v w $y{\cdot}Y^{L}$ $y^{+}/\ XY^{S}{\cdot}Y^{L}$, y $Y^{S}{\cdot}Y^{L}$ B^{S}

b. " $/\ XY^{L}{\cdot}Y^{S}$, y $Y^{L}{\cdot}Y^{S}$ y^{+}

These were treated with 3000r of X-irradiation, mated to suitable males, and brooded for five 3-day broods. TM chromosomes would result from breaks in the centric heterochromatin of each homologue; RAs would most likely involve a break in the distal X-heterochromatin (I) or in the Y^{S} arm (II) of the inverted chromosomes and a break in the centric region of their homologues (Novitski, 1954).

In order to determine the true number of compounds formed, corrections have to be effectuated that will take into account those compounds which were induced and then lost due to meiotic crossing over. The observed frequencies of TMs and RAs in broods 1 and 2 represent 13% and 15% of the actual number of compounds induced in oöcytes, respectively. In broods 3, 4, and 5, the observed frequencies of TMs and RAs represent 16% and 30% of the compounds actually induced in oögonia. These figures were calculated using the tetrad analyses of Novitski (1951) and Sandler (1954) for compounds and our own tetrad analyses for inversion heterozygote tetrads in which the events producing a compound were superimposed on crossing over. In addition, the assumptions that the tetrad distribution in the inversion heterozygotes and in the TMs is E_0=.05, E_1=.60, and E_2=.35, whereas in the RAs it is E_0=.44, E_1=.12, and E_2=.44, were made. The results of the experiment are given in the following table:

	P	TMs*	RAs*	N**	Freq. TMs	Freq. RAs	Ratio TMs/RAs
I	a.	21	74	109,100	192×10^{-6}	678×10^{-6}	.284
	b.	121	54	61,312	1973 "	880 "	2.241
II	a.	22	6	213,180	103 "	28 "	3.666
	b.	152	61	262,464	579 "	232 "	2.492

* corrected (see text)
** corrected (x4)

Fully realizing that an uncomfortable number of assumptions were made to obtain the tabulated figures, we maintain that some trends are still worthy of notice: (1) The frequency of TMs is greater in those cases where the non-inverted chromosome was $XY^{L}{\cdot}Y^{S}$ (Ib and IIb). This may indicate that Y^{L} of the inversion folds back and preferentially pairs with Y^{L} of the non-inverted chromosome and/or that Y^{S} of the non-inverted chromosome folds back and preferentially pairs with X-heterochromatin to the left of the centromere of the inverted chromosome. In the case of Ia and IIa, the same preferential pairing relationships would make the formation of TMs more difficult. (2) The frequency of RAs is greater when the distal heterochromatin on the inverted chromosome is that of sc^{8L}. This observation cannot readily be explained on the basis of homology and may reflect a greater breakability of X vs. Y-heterochromatin. (3) The higher frequency of RAs induced in IIb than in IIa may be a function of the longer heterochromatic segment available to the left of the centromere of the non-inverted chromosome in IIb. This physical difference apparently masks the fact that Y^{S} may have greater pairing affinity for Y^{S}, in the case of IIa, than it would have for Y^{L}, in the case of IIb. The same trend is seen in Ib vs. Ia. Here, the lessened degree of difference indicates that X-heterochromatin (of sc^{8L})

pairs more readily with Y^S than Y^S would with Y^S. This may not be as peculiar as it may seem if one considers that in normal situations, the X and Y are pairing partners.

This research was performed in Prof. E. Novitski's laboratory and was supported by the NSF URP program.

Lucchesi, J. C. University of Oregon.

The influence of heterochromatin on crossing over in ring/rod heterozygotes of D. melanogaster.

The presence among the Eugene stocks of a large number of tandem metacentric (TM) lines, synthesized for other purposes by Drs. E. Novitski and W. J. Peacock, has afforded the opportunity to study the influence of pericentric heterochromatin on the crossing over properties of ring X-chromosomes. The TM lines had been obtained by irradiating females of the constitution XY, y^2su-$w^a w^a Y^S \cdot Y^L y^+$(Parker)/ XY^L, In(1)sc^4-sc^{8L}, EN^R, y f v(or m) cv $y \cdot Y^L y^+$. Two different inverted chromosomes, derived from two separate stocks of In(1)EN, had been used. TM's bearing one or the other of these inverted chromosomes will be referred to as (A) or (B) in the following presentation (series 17- and 19- of Novitski and Peacock).

Virgin females from each of 5 (A) and 9 (B) TM lines (all heterozygous for the markers y, cv, v (or m) and f) were crossed to males bearing the fertility tester Y-chromosomes of Brosseau; the fertility of ring-bearing sons with each of the various Y-testers determined the amount of Y heterochromatin present in a given TM (and, therefore, in the ring X-chromosomes derived from it), measured in terms of fertility factors. Using the representation for a normal Y + + + + + c + +, where the first five + signs indicate kl-5 through kl-1, c is the centromere and the next two + signs are ks-1 and ks-2, the following distribution was obtained: - - - - - c - - (3), - - - - + c - - (5), - + + - + c - - (1) and + + + + + c - - (5).

Ring chromosomes from each of the above four types were used in the following crosses: X^c(from TM (A) or (B)), y cv v (or m) / f x sc cv v f B / Y. Each cross consisted of 12 or 24 pair matings, brooded for three 3-day broods. Care was taken concerning uniformity in age of females and temperature. The results are presented in the following table:

Ring Type* (kl-1 tokl-5)	Rod ♂♂ c.o. / n.c.o.	Rod ♂♂ % c.o.	Ring ♂♂ c.o. / n.c.o.	Ring ♂♂ % c.o.	Pat. ♂♂	♀♀	Pat. ♂♂ / ♀♀
(A) - - - - -	87 / 1785	4.6	32 / 1340	2.3	77	3405	2.3
+ - - - -	187 / 1635	10.3	55 / 1300	4.1	85	3447	2.5
(B) - - - - -	169 / 1657	9.3	36 / 1302	2.7	87	3127	2.8
+ - + + -	124 / 910	12.0	20 / 590	3.3	70	1681	4.2
+ + + + +	94 / 883	9.6	24 / 612	3.8	67	1683	4.0

* Refers to the number of Y^L factors present in the pericentric heterochromatin of the ring chromosome.

The results of crosses involving rings derived from type (A) TM chromosomes suggest that an increase in pericentric heterochromatin results in an increase in the frequency of crossing over. Furthermore, this increase is of greatest magnitude in region IV which, significantly perhaps, is the region adjacent to heterochromatin in both the ring and the rod chromosomes. Rings derived from type (B) TM's show an increase in crossing over with added heterochromatin of a much lower level of magnitude. The following table presents recombination frequencies for each individual region studied; the rings containing no Y^L fertility factors are used as the reference base; the Table also includes a comparison of one of the (B) rings with the (A) series

Ring type	I (y to cv) Rods	Rings	II (cv to v/m) Rods	Rings	III (v/m to f) Rods	Rings	IV (f to centr.) Rods	Rings
(A) - - - - -	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
+ - - - -	2.02	1.40	2.05	1.95	2.25	1.14	3.84	3.09
(B) - - - - -	1.79	0.96	1.96	0.92	1.7	1.19	3.8	1.72
(B) - - - - -	1.00	1.00	1.00	1.00	1.00	1.00	1.00	1.00
+ - + + -	1.13	1.18	1.40	1.56	1.40	1.10	1.00	1.00
+ + + + +	1.02	1.47	0.93	1.00	1.10	1.84	1.15	1.27

The difference in the results obtained with type (A) and type (B) rings may be explained in the following manner. By chance, or because of the use of different inverted chromosomes in the original TM synthesis, the (B) rings may have a segment of unspecified X or Y heterochromatin large enough to render them less sensitive to additional heterochromatin. This working hypothesis may be tested by a systematic search among type (A) TM's for rings which lack all of the Y fertility factors, yet yield high crossing-over values (of the order of 10%); and conversely, among type (B) TM's for rings yielding low crossing-over values (of the order of 4 or 5%). Cytological estimates of relative size may be useful to determine the presence of unspecified X or Y heterochromatin.

$(XY^L)^C$ chromosomes will be sent to the Drosophila Stocks Center of the Institute for Cancer Research, Philadelphia, from where they would be available to anyone who might want to use them as balancers for special stocks.

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Zambruni, L. University of Milan.

Preliminary chromatographic analysis of the "brown spots" character in Drosophila melanogaster.

Owing to the peculiar manifestation of the bsp character, it seemed useful to investigate if differences in free-ninhydrinpositive substances could be correlated with the changes of metabolic pattern in the female following copulation. According to the method described by Fox et al. (1959), two-dimensional chromatograms of virgin and mated females (5 days old) were obtained. The quantitative analysis of the free-ninhydrin reacting components showed that the tyrosine amount does not change after mating in bsp females, while an increase occurs in the Sevelen females (control). This finding points to a correlation between tyrosine and brown spots formation, because it can be suspected that part of this substance is utilized for the formation of brown pigment.

Holt, Th. K. H., and H. D. Berendes. Genetisch Laboratorium, The Netherlands.

Experimental puffs in D. hydei polytene chromosomes, induced by temperature shocks.

During the entire third larval instar abnormal puffs can be induced in the salivary gland chromosomes by transferring the larvae from 25 to 35°C. for 1 hour. These abnormal puffs (located at 32A, 36A, 48C, 58B, 81B and 85B respectively, according to the cytological map of Berendes, 1963), are also induced in cells of the stomach, midintestine and Malpighian tubules. Also, in salivary glands transplanted from early third instar larvae into the abdomen of adult females, abnormalities in the puffing pattern are induced by temperature shocks. After 3 days of implantation, the flies were shocked for 1 hour, and after 3 weeks of implantation for 1/2 hour. Both experiments revealed the same abnormal puffs as found after treatment of normal larvae.

Momma, E. Hokkaido University, Japan.
The variability of abundance and sex-ratio in natural population of D. nigromaculata.

A preliminary note on a survey of Drosophila population in the University Botanical Gardens investigated during ten successive years from 1953 to 1962 are presented here. Monthly collections except in three years, 1953, 1955, and 1959 were routinely carried out by the same methods with the use of banana traps during seasons. Flies were collected on three successive days near the end of each month. On each day of collection, flies were collected at one-hour intervals from sunrise to sunset. During seven years, 20,820 flies representing 38 species were collected.

D. nigromaculata was one of the two abundant species, of which the other was D. auraria, showing the rate of 26.36%. Sex-ratio of this species was 129. The results are presented in Table 1.

Data in Table 1 seem to indicate that the variability of sex-ratio shows no relation to annual activities. In general, this species showed a trimodal seasonal activity with three peaks in May, July and September. Sex-ratio showed seasonally low in inactive periods between the three peaks. A bimodal diurnal activity with two peaks in the morning and in the evening was observed in this species. In inactive periods between the three active ones, the male diurnal activity decreased especially toward evening.

Table 1: Percentage frequencies of abundance and sex-ratios of D. nigromaculata in Drosophila populations sampled from the University Botanical Gardens at Sapporo during the seven years.

	No. of flies of nigromaculata	Frequency in a given period	Relative frequency	Sex-ratio			
1954	1209	52.4	22.0	126			
1956	846	22.5	15.4	97			
1957	503	15.4	9.2	114			
1958	682	35.4	12.4	137			
1960	586	27.3	10.7	132			
1961	1109	24.2	20.2	153			
1962	554	19.6	10.1	147			
Total	5489	26.4	-	129			
					Sex-ratios during a day		
					Morning	Noon	Evening
May	1215	45.8	22.1	164	176	190	145
June	288	11.6	5.3	88	110	80	63
July	1149	20.4	20.9	112	106	109	120
August	633	11.1	11.5	90	104	90	65
September	1830	47.4	33.3	142	146	120	153
October	374	31.8	6.8	140	138	123	176

Von Halle, E. S. Oak Ridge National Lab., Tennessee.
Localization of E-spl.

In a stock of y^2 su-w^a w^a spl; T(2;3)Xa, E-spl/+ (Welshons, 1956, DIS 30:157) a y^2 su-w^a w^a spl; E-spl non-Xa male was found and a stock was established. E-spl is homozygous viable and appears wild type in the absence of spl. Matings of spl/spl females by spl; +/SM1, Cy; E-spl/ Ubx^{130} males indicated that the mutant is on chromosome 3. A further attempt to localize the mutant was made by crossing spl;E-spl females to ve h th cu bx e^s ro ca males (stock from R. F. Grell). spl/+; ve h th cu bx e^s ro ca/E-spl virgins were then backcrossed to homozygous marker males, and non-spl crossover males were selected and crossed individually to homozygous spl virgins. The progeny were examined for the presence or absence of E-spl. These results indicate that E-spl is close to ro on the linkage map. Ten crossovers between e^s and seven crossovers between ro and ca included no crossovers between ro and E-spl.

Levine, Louis and Seymour Kessler. The City College of New York and Columbia University. Use of the Drosophila maze to study rate of activity.

The Drosophila maze has to date been utilized to study either geotaxis (Hirsch and Erlenmeyer-Kimling, Jr. Comp. Physiol. Psychol. 1962; Dobzhansky and Spassky, P.N.A.S. 1962) or phototaxis (Hadler, Biol. Bull. 1964). However, the maze can also be used to study the rate of activity of different mutants and species of Drosophila, and to select for faster and slower running of the maze.

In preliminary experiments involving the mutant orange (or) of D. persimilis, it was found that some 93% of the flies ran the maze in a 24 hour test period. In the case of the mutant glass (gl) of D. pseudoobscura, some 59% of the flies ran the maze in the same time interval. Data gathered at two hour intervals show that for the or flies, some 90% of the flies that ran the maze did so in the first eight hours. When using gl flies, only some 60% did so during the same period of time.

Selection for fast and slow running of the maze have been initiated. It has been possible to select for increased speed of running the maze in both or and gl flies. However, only in the case of gl flies was the total percent of flies completing the maze in the test period also increased. Results of selection for slow running have not thus far been fruitful. The pattern and percent of running the maze appears to be stable despite six generations of selection.

Experiments are now in progress to test the maze running characteristics of wild populations of these two species and the hybrids between them.

Research supported by NSF grant GB-1906 to Professor Howard Levene of Columbia University.

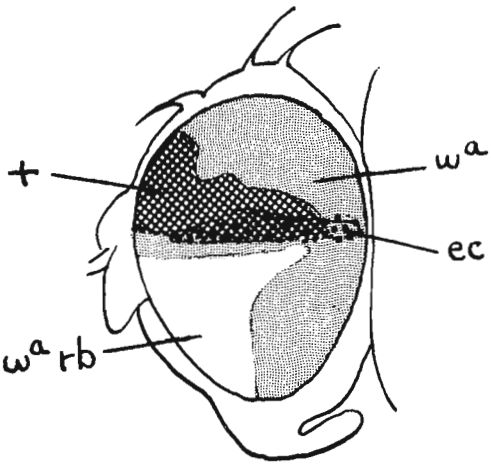
Baker, William K. University of Chicago. A method for the developmental timing of the pattern of variegation.

In order to obtain more critical evidence on the time during development when the pattern of position-effect variegation is laid down in the eye anlage, a genetic scheme was devised in which twin spots resulting from induced somatic exchange and variegated pigmentation caused by position effect could be scored in one and the same eye. The rearrangement causing white variegation was $Dp(w^m)264\text{-}58a$, an insertion of the white region into the base of 3L. Larvae of the following genotype were exposed to X rays 40 hrs. after the eggs were deposited:

$$\frac{Y^S \; y \; w^a \; + \; rb \; + \; f}{+ \; w^a \; ec \; + \; sn \; +}; \quad \frac{Dp(w^m)}{III}$$

A somatic exchange will produce a twin spot in the eye consisting of a patch of "white" (w^a rb) tissue next to a patch of echinus tissue, these spots being located in a background of w^a-pigmentation and normal facet arrangement. The presence of $Dp(w^m)$ will cause an area of wild-type pigmentation in the same eye.

When twin spots were induced by irradiation at 40 hrs. the size and shape of the twin spots were much the same as that of the variegated tissue. The accompanying figure illustrates one such eye. Although the precise shape of the echinus area is difficult to delimit by visual inspection, its general outline is obvious. The areas of different pigmentation can be delimited quite precisely. The ambiguities in the outline of the echinus area prevented the full utilization of the experimental design, which had incorporated the homozygosis for Y^S, in the white member of the twin spot, to see if there was a sector effect of additional heterochromatin when the variegated tissue partially covered both members of the twin spot.



A series of eyes similar to the one pictured have provided the critical evidence that the pigment potentialities of the developing variegated eye anlage are determined during the end of the first larval instar. (Studies supported by an NSF Senior Postdoctoral Fellowship).

van Breugel, F. M. A. Genetisch Laboratorium. The Netherlands. Experimental puffs in D. hydei salivary gland chromosomes observed after treatment with CO, CO_2, and N_2.

Third instar larvae of D. hydei were treated with sublethal doses of carbon monoxide (3 hours in 100 percent CO at 25°C). Salivary glands from treated larvae were inspected for changes in puffing pattern. No changes were found immediately after treatment. However, subsequent exposure to air produced six large puffs not normally seen in larvae. These puffs begin to appear after 15 minutes, attain rapidly their maximum size and revert to normal slowly within several hours under aerobic conditions. They are located at 32A, 36A, 48C, 58B, 81B, and 85B following the chromosome map by Berendes (Chromosoma 14, 1963).

In an attempt to find out whether these puffs are induced by lack of oxygen, larvae were similarly kept for 3 hours in pure (commercial quality) O_2, N_2, or CO_2, or submerged in Ringer solution (30-40 larvae in 1 ml in hermetically sealed container). Whereas pure oxygen had no effect on the puffing pattern either during or after treatment, the remaining agents induced exactly the same puffs in the same manner as CO, i.e., the puffs became visible shortly after exposure to air following treatment. Furthermore, if 3 hours of CO_2 was immediately followed by N_2 (1/2 hour), the puffs did not begin to form until some time after removal from the N_2 atmosphere.

In larvae deprived of oxygen for a shorter time (under 3 hours), these "recovery" puffs will be present for a shorter period also. Tentatively, it may be concluded that these special puffs are somehow connected with respiration. The fact that the same puffs are obtained also by temperature shock (Holt and Berendes, DIS 40) suggests that temperature treatment may act via the respiratory system (cf. Ritossa 1964, Exp. Cell Res. 35).

Meyer, Helen U. and Rayla G. Temin. University of Wisconsin. A recessive suppressor of Curly found in a third chromosome from a natural population of D. melanogaster.

In an experiment in which chromosomes 2 and 3 from nature were made homozygous with the aid of the Cy-Oster and the Me inversions, the classes (Cy/2; Me/3), (Cy/2; 3/3), (2/2; Me/3) and (2/2; 3/3) were expected in a 4:2:2:1 ratio in the absence of any viability mutations. In about 1500 such tests, one case was found in which only the first and last classes were present. The latter appeared to have the normal wild phenotype. This was inconsistent with the presence of a recessive lethal on either or both chromosomes, but suggested instead a translocation or some type of epistasis. Testing for a translocation, by crossing Cy/2; Me/3 with cn bw; e, yielded negative results. Other such testcrosses, using instead the non-Cy, non-Me sibs as parents, unexpectedly gave rise to Cy progeny. Twenty-four such "wild-type" males, bred individually, gave 778 Cy to 771 non-Cy offspring. The 1:1 ratio suggested that the wild-type flies belonged, in fact, in the Cy, non-Me class, their phenotype being suppressed by a recessive gene on the 3rd chromosome. This agreed with the observation of a nearly 2:1, rather than a 4:1, ratio of Cy:non-Cy progeny in repetitions of the original inbreeding cross, Cy;Me x Cy;Me. The actual counts from 9 such tests were 257 Cy;Me to 119 "wild type." When the 3rd chromosome was extracted from the original stock and made homozygous in the presence of Cy-Oster/Pm from another stock, the Cy phenotype was again suppressed. This test showed, furthermore, that the original 2nd chromosome from nature was not required for the effect.

A recessive lethal in chromosome 2 was shown to be responsible for the absence of the non-Cy genotypes in the original culture. Such lethals occur with a frequency of about 25 percent in nature.

Therefore the flies originally classified as wild type were in fact of the composition Cy/lethal 2; suppressor-Cy/suppressor-Cy. They are of good viability and can be kept in stock in the above form.

Lee, William R. The University of Texas.

A modified "Maxy" stock that produces only females of the proper type.

The "Maxy" stock (Muller and Schalet, 1957, DIS 31:144) used for detection of specific-locus mutations in the X-chromosome regularly produces only one type of female and male. In experiments where sperm are treated with a mutagenic agent, males of the F_1 generation are useless except to detect the absence of a marked Y as is possible in a modified "Maxy" stock described by Lee (1963, DIS 38:87-88). Therefore, for some experiments it is desirable to produce only females of the proper type to increase the efficiency of scoring and to provide virgins for the crosses necessary to make a balanced stock for testing for X-chromosome lethals in the F_2 and succeeding generations.

To accomplish this a stock was made with the following composition: sc^8.Y.B^S/1J1 $sc^{J1(+)}$ In49 F1 g B^{M1}, In/ "Multiple from the Maxy Stock of Muller and Schalet (1957)." When virgins from this stock are crossed with males of the composition, IN49 v ptg oc F1 B^{M1}In, the unwanted class of females is killed by F1 being homozygous and all males are killed either by 1J1 or the 1 in "Multiple Maxy Chromosome." It was feared that the use of both 1J1 and F1 would greatly reduce the viability; however, this stock has been found to have good viability in our laboratory and gives a normal ratio of males to females. F1 is known to act as a dominant or semi-dominant in some stocks (Muller and Zimmering, 1960, Genetics, 45:1001-1002); however, in the stocks reported here (as a result of selection by the author when these stock were initiated) it acts as a recessive.

Non-disjunction produces sterile vermilion males and garnet females. The latter can readily be distinguished from vermilion garnet females produced by a garnet mutation in the treated chromosome. The v in the treated chromosome also makes the scoring for other eye colors more objective. The possibility of fertile males in the F_1 generation being produced by non-disjunction from a female that has a Y is eliminated by using sc^8.Y.B^S in the parental stock and discarding any B^S females when collecting virgins.

The modified "Maxy" stock described in this report has also been useful in our laboratory in producing automatic virgins for the scheme described by Lee (1963) of detecting loss of either the X or Y chromosome in combination with detecting mutations at specific loci.

In both the breeding scheme reported here and the one previously reported (Lee, 1963), mutations are induced in the In49 v ptg oc F1 B^{M1} chromosome. The spontaneous rate of recessive lethals for this chromosome has been found to be 0.15% (9,155 chromosomes tested) and only 2 visibles have been found in 20,900 F_1 females.

This investigation was supported by Public Health Service Research Grant GM11449-02 from the National Institute of General Medical Sciences).

Scharloo, W. and W. Vreezen. Genetisch Laboratorium der Rijksuniversiteit, Leiden, The Netherlands. Selection for increased 4th vein interruption in Hairless.

A gap of the 4th vein is one of the features of H. This interruption responds easily to a temperature change when the vein is almost complete, but with larger interruptions changing the expression by temperature becomes increasingly difficult. The same character in ci^D is very difficult to change when the 4th vein is nearly complete, but can easily be altered at more extreme expression (Scharloo, 1962). In preliminary experiments it was found that selection for more extreme 4th vein interruption in H leads to accumulation of modifiers which cause a 4th veing interruption even in the absence of H ("assimilation" of the mutant character). Assimilation never occurred in long term selection for ci^D fourth vein expression (Scharloo, 1962 and DIS 38). With the purpose of testing whether the assimilation of the H 4th vein interruption is a property associated with the H mutant, 4 selection experiments were done. H and the new mutant H^{57c}(obtained from H. Gloor) were introduced in the backgrounds of the Pacific and Kaduna cage populations. All lines responded rapidly to selection for larger 4th vein interruption. The first assimilated flies appeared in all lines between generations 4-6, but the speed of further increase in frequency and expression differed widely. Introduction of chromosomes with dominant markers showed that factors on both large autosomes are involved. The frequency distributions of H expression show large variability in the first generations of the experiments when expression still overlaps with wild type, and very low variability when more extreme expression is reached. This confirms the conclusion of the temperature experiments that change is relatively easy in the neighbourhood of wild type. The introduction of chromosomes with dominant markers revealed that differences in modifiers also have larger effects when the 4th vein is almost complete.

Watson, J. E., E. Scheinberg, and L. A. Dittmar. Purdue University. Effects of ether on fitness traits.

We were interested in assessing the effects of etherization on the fitness of very young D. melanogaster imagos, so the following experiment was run: Two wild-type strains of D. melanogaster were chosen; (1) W101 is a laboratory wild stock which had been subjected to etherization periodically since 1955 and (2) WSB is a strain derived in 1963 from a collection in the wild and had never been exposed to ether, having been maintained by mass adult transfer. The ether treatment consisted of a 15-20 second uniform exposure to males and females at ages of 3-4 hours. Four types of matings were made: 1-♀ and ♂ non-etherized (N.E.), 2-♀ and♂ etherized (E.), 3-♀ (N.E.) and ♂ (E.), 4-♀ (E.) and ♂ (N.E.). Twenty single pair matings for each strain x mating were made. The females laid eggs on caps spread with a charcoal media while in inverted 1/2 pint milk bottles. Each 48 hours the caps were replaced until 4 successive broods had been isolated from each parental pair. The experiment was thus analyzable as a 2 x 4 x 4 factorial experiment with 20 observations per cell.

A cursory look at the analysis of variance yields the following information - Eggs laid: There is no significant difference between (or among) strains or matings in the total number of eggs laid. The brood fraction of the variance is highly significant which is what one expects due to the known maternal-age-effect on number of eggs laid. It appears that etherization has a stimulatory effect on the onset of egg lay for although the differences are not significant (at α=.05) in the crosses where one or both of the parents were etherized the mean number of eggs laid is 2 to 3 times that of the control value. This holds true, however, only for the first 48 hours, after which an adjustment in the respective means occurs such that the 8 day totals for each strain x mating are essentially equal.

Egg hatchability: A significant difference in percent hatch was obtained between W101 and WSB. W101 had an overall hatch percent of 90.1% while the non-previously etherized strain yielded 85.2%. That this difference was not due to undetected and unanalyzed lethals present in this "natural" wild strain can be supported by the observation that the unetherized control matings of each strain yielded essentially the same hatch percentages. The significant difference arose because in the Number 4 matings (only ♂ etherized) of the WSB strain, the hatch percentages were noticeably lower than those obtained for W101. This holds across all four broods. This result is of interest and will be studied further. Also, the fourth brood percentages of the WSB strain are lower than those for W101, in both etherized and control matings. This could be due to the non-acclimatization of this strain to laboratory conditions. Sex Ratio: In a sub-analysis of this experiment no significant differences were detected in the sex ratios of the progeny obtained.

A more detailed analysis of this data is in progress. (We wish to acknowledge the use of the facilities of Dr. A. E. Bell and the Population Genetics Institute).

Barigozzi, C. University of Milan, Italy. New Data of the transmission of Freckled.

New data have been collected proving that Frd behaves erratically as a non entirely mendelizing unit.

New investigations have been brought about to know more precisely its localization, and, provisionally, it can be taken that its locus may be between brown (104.5) and speck (107.0), thus near the right tip of the 2nd chromosome. The localization, on the other hand, in some crosses is not possible, owing to unexpected facts.

Besides several cases of backcrosses of the type: $\frac{+\ +\ Frd}{Cy\ L\ +} \times \frac{+\ +}{+\ +}$, where there is a low recombination between the markers and Frd in the female, and no recombination in the male, several others have been found, which can be classified as follows: a) considerable or very high frequency of recombination in males, even in proportions of 50 and 100%, giving rise to symmetrical complementary classes; b) recombinations in the male in presence of inversions on the homologous chromosome (Cy L and Pm), sometimes with statistically significant differences between the complementary classes; c) complete lack of one of the complementary classes, which often denotes lethality of the chromosomes originally carrying Frd; d) loss of Frd in offspring of crosses, where its presence was expected in the 50% of the individuals; e) sudden changes in expressivity and penetrance of Frd, followed by loss of the unit. All these phenomena have been found in both sexes. The findings point to a peculiar behaviour of Frd, interpretable as a changing relationship between Frd and the carrier chromosome.

Gugler, H. D., W. D. Kaplan, and K. Kidd.
City of Hope Medical Center, California.

The displacement of first-mating by second-mating sperm in the storage organs of the female.

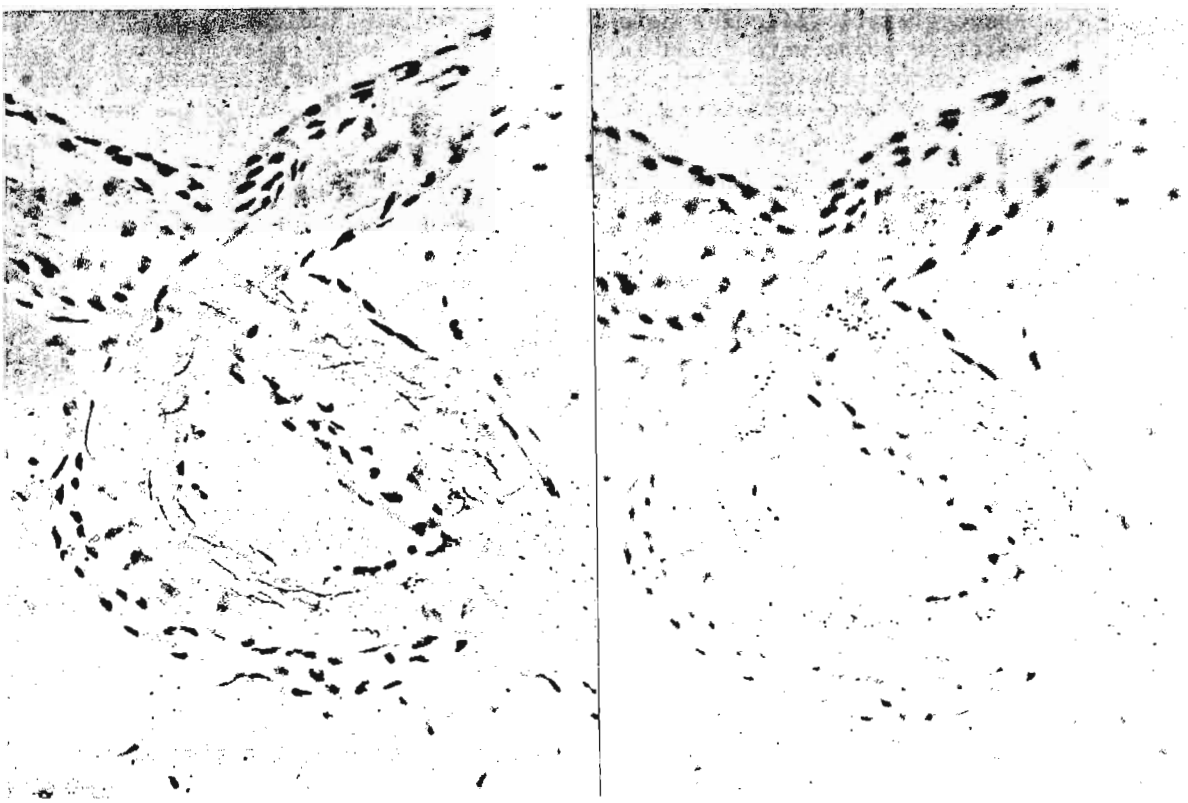
The sperm storage organs of D. melanogaster females are non-elastic and non-contractible, providing room for only one-fifth of the volume of sperm initially deposited in the uterus by the male. Lefevre and Jonsson (1962) showed that one mating normally fills these organs to capacity, leaving no space for the storage of sperm from a second mating. Although the fecundity of twice-mated females did not significantly exceed that of once-mated females, the majority of their progeny derived from sperm of the second mating.

By means of females mated first to one-day old untreated Canton-S males and secondly to Canton males whose sperm had been labeled with tritiated deoxycytidine we were able to observe directly the displacement of the unlabeled by the labeled sperm in the spermathecae and ventral receptacles of the females. The sperm were labeled by placing 16-hour old larvae upon food containing a total activity of 200 µc from the presence of tritiated deoxycytidine. The sperm utilized by these males for their first matings contained about 90% labeled sperm bundles.

Virgin females, 24-hours old, were mated with 24-hours old non-labeled virgin males, the time of mating was observed and immediately thereafter, without etherization, the female was removed to another vial and presented with a virgin labeled male. The interval between the first and second mating was noted. After observing the second mating the females were taken and squash preparations were prepared of the spermathecae and ventral receptacles. Radioautographs were prepared from these squashes from which the presence of labeled and non-labeled sperm could be noted.

Precise timing of first and second matings was obtained in three cases of a total of 30 females with which the study was started. In all those cases more than half of the sperm of the spermathecae and ventral receptacles appeared to be from the second mating. The shortest interval between the second mating and fixation of the female reproductive tract was 20 minutes. It, therefore, may be concluded that within 20 minutes sperm from the second mating can replace first-mating sperm in both these storage organs.

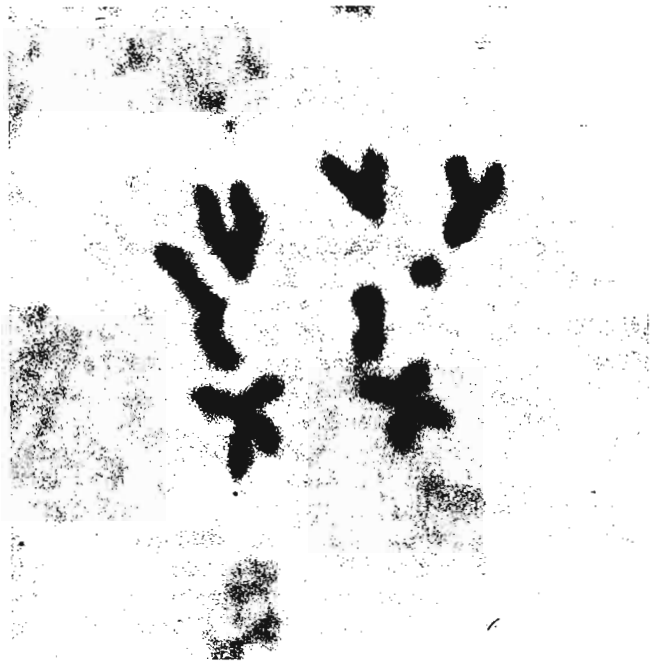
The accompanying figure shows the ventral receptacle of the female killed 20 minutes after the second mating.



Legend:

Ventral receptacle from female inseminated first with unlabeled and secondly with labeled sperm (a, tissue level; b, level of photographic film).

Mather, Wharton B. University of Queensland, Australia. The metaphase chromosomes of D. rubida.



In DIS 34, E. B. Lewis and Linda Smith Riles described and illustrated a technique, modified from mammalian chromosome methods, for preparing D. melanogaster metaphase plates from the larval brain. The great advantage of this technique is that preparations with many metaphase plates are obtained and the position of the centromere in each chromosome is pinpointed because the sister chromatids tend to separate. Thus, V chromosomes appear as X-shaped bodies and rod chromosomes as V-shaped bodies. With this technique metaphase plates have been obtained which clearly show that the chromosome number of the tropical Australasian species D. rubida of the immigrans group consists of 1 pair of V's, 2 pairs of rods and 1 pair of dots. In the photograph only one of the dots is split into two chromatids.

Barker, J. S. F. University of Sydney, Australia. A new parasite of laboratory Drosophila.

In April, 1963, some small Hymenoptera were observed in our laboratory. The numbers rapidly increased, and they were then found to be present in a number of population cages. Examination of empty pupal cases from these cages showed a proportion to have a small circular hole in the operculum, indicating that they had been parasitized. Parasitized pupae were found in cages containing D. melanogaster, D. simulans, D. pseudoobscura, and D. nebulosa. In an attempt to control the infestation, adult Drosophila of stock populations were transferred to clean population cages. While removing the media jars during cleaning of the old cages, individual Hymenoptera females were observed apparently ovipositing in newly formed pupae. Three such pupae were taken after the female moved away, and were placed individually in small vials at 25°C, 65-70% relative humidity. 29 days later, one adult Humenoptera emerged from each of two of these pupae. There was no emergence from the third.

G. E. J. Nixon, Commonwealth Institute of Entomology, London, kindly identified the parasites as Hymenoptera; Proctoterupoidea - Spilomicrus spp. Species of this genus have not, I believe, previously been recorded as parasites of Drosophila. Adult Spilomicrus showed strong positive phototaxis, and the infestation was readily controlled by the use of light traps. It is assumed that the parasites entered the laboratory from a natural population, but there has been no re-infestation this year.

Fujii, S., T. Kanehisa, and M. Ohinishi. Kobe University, Japan. On protein analysis in a tumor strain.

Eggs or pre-third instar larvae were analyzed for soluble protein by mean of electrophoresis and D.E.A.E. column-chromatography. A remarkable increase of Albumen fraction was found in the tumor strain (tu st). This fraction from tumor individual, comparing to a control (Oregon R), has a unique part eluted by NaCl 0.1M. Further, a remarkable decrease of a fraction which has a maximum adsorption at 260 mμ was clarified in the alkalien-soluble fraction of PH 4.5 precipitates from the Albumen fraction above mentioned which showed a qualitative difference by Tiselius-electrophoresis.

Hochman, Benjamin. The University of Tennessee. A note on the salivary chromosome 4 in D. melanogaster.*

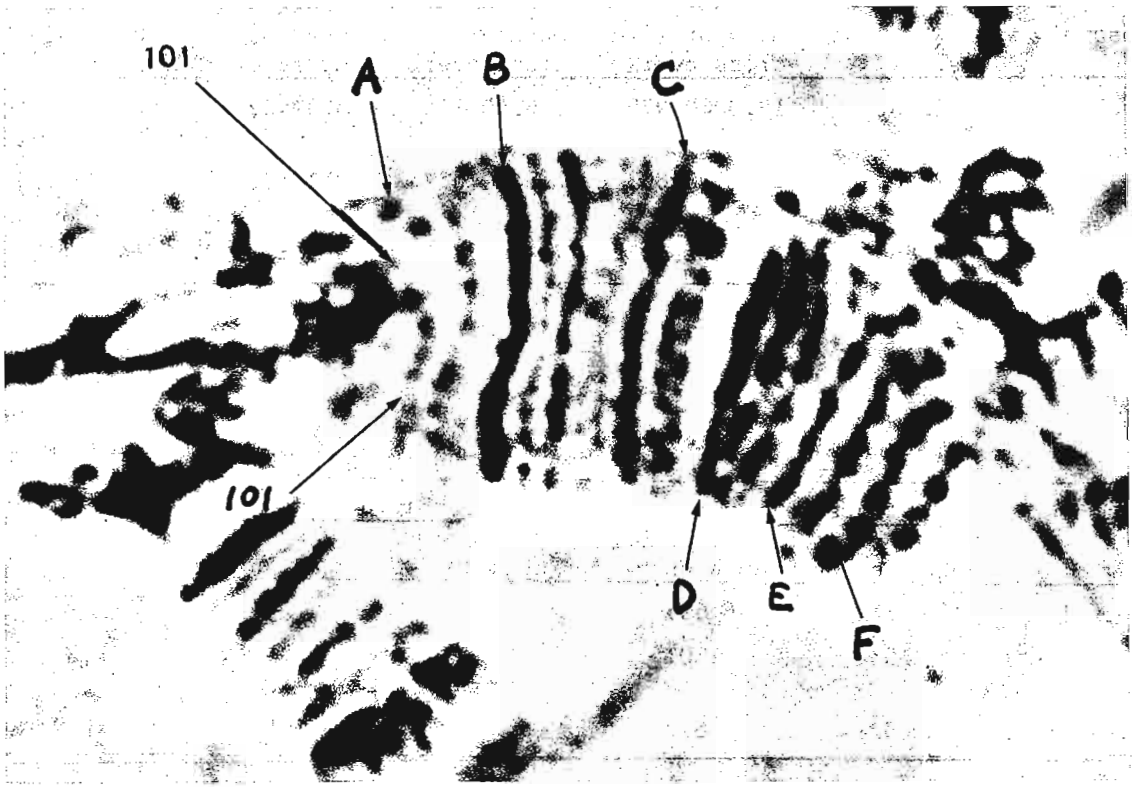
I am presently occupied with the salivary chromosome analysis of a substantial number of spontaneous and induced lethals situated on the fourth chromosome of D. melanogaster. Genetic evidence indicates that several of these lethals are deficiencies and, as expected, the salivary chromosomes of some of them lack one or more of the bands found on a normal fourth chromosome. Through the use of these aberrations it is hoped to eventually map most or all of the lethal loci, as well as the visibles, that have been detected on chromosome 4.

While the literature contains a few drawings of the fourth chromosome as it appears in salivary gland cells (e.g. Bridges, 1935 and Slizynski, 1944), I have been unable to locate a published photomicrograph of 4 that bears sufficient resemblance either to the aforementioned drawings or to what can be observed with the microscope. Utilizing (with some modifications) the method of Dr. J. Schultz as described by Nicoletti (DIS 33:181-182), I have obtained preparations that exhibit the banding details shown on the accompanying photograph.

If this picture is compared with the commonly reproduced Bridges' drawing (J. Heredity 26: 60-64) a close agreement in banding pattern is readily discerned. Most of the darker lines in Slizynski's drawing (J. Heredity 35:322-324) of a greatly stretched fourth chromosome can also be matched to those appearing in the photograph.

In the illustration shown nearly all of the right arm of 4 can be seen extending from the chromocenter (at the left). (The proximal region of another chromosome is also in view). Additional chromocentric material may be observed adhering to the distal part of 4R (right, top). Unless the squash pressure is adequate both ends of 4R may remain embedded in the chromocenter giving the chromosome a C - shape and making cytological work virtually impossible. Although the left arm of 4 is not visible in this cell, it is seen frequently in our preparations. Its general appearance is much like that noted by Slizynski.

The arrows (A-F) superimposed on the photograph indicate the doublet bands that mark the beginning of each of the six subdivisions of the 102 division namely 102A1,2, B1,2...F1,2. The two arrows labeled 101 point to the last dark band in division 101 (F1,2 ?). A closer inspection of the figure reveals that one of the homologues lacks 102A1,2. The genotype of the dissected larva was $M(4)^{63a}$/ ci^{D}. This Minute is viable over ci^{D} and it also uncovers ci.



Legend: The moderately stretched right arm of salivary chromosome 4 from a larva with the genotype $M(4)^{63a}/ci^{D}$.

One may tentatively conclude that the Minute and cubitus interruptus (recessive) loci are in or near band 102A1, 2. Dr. M. J. Fahmy, in whose laboratory $M(4)^{63a}$ was discovered, reports that the chromosome in question is deficient for bands 101 F6,7 and 102A1,2 (pers. comm.).

*work supported by U.S. Public Health Service Research Grant GM 11627-02

Sharma, R. P. and A. T. Natarajan. Indian Agricultural Research Institute, India.

Studies on the effect of X-Chromosome inversion on crossing-over in 2nd chromosome of Drosophila melanogaster.

The present investigation was carried out to get information about the mode of action of hetero- and homozygous X-Chromosome inversion on crossing-over when both the homologues of the second chromosome are normal and when they are heterozygous for inversion. A complex X-Chromosome inversion (sc^{S1} In-S sc^{8}) obtained from 0_1 stock, having a recessive yellow body, has been used throughout the study. Two types of experiments were conducted. The females were with either hetero- or homozygous X-Chromosome (0_1) inversion, whereas for the second chromosome the genetic constitution of the females was + + + L^2 +/ dp b cn + bw in the first experiment, Cy + + + +/dp b cn bw in the second experiment respectively. Cy, a wing marker gene is associated with two long inversions, i.e., Cy Ins (2L+2R). These females were mated separately with the dp b cn bw/dp b cn bw males.

It is evident from table 1 which includes data from the first experiment that there is a significant increase over control in crossing-over in the presence of hetero- as well as homozygous inversion, but inspite of involving the whole or arm of the chromosome, it is restricted only in certain regions, mainly region b-cn, centeromeric region. The difference between the cross-over value of hetero- and homozygous inversions is not statistically significant. In the second experiment when 2nd chromosome was having Cy Ins (2L+2R), no cross-over in the case of control (X-Chromosome without inversion) has been observed, but it is clear that the centromeric region viz. b-cn, shows an increase in cross-over value in the presence of both hetero- and homozygous 0_1 inversion (table 2).

The results obtained (a) cross-over increase in only certain regions of the chromosome (b) enhancement in cross-over frequency in the presence of homozygous inversion, argues against the "mechanical hypothesis" proposed for interchromosomal effect.

Table 1. Analysis of the single cross-over in the cross + + + L^2 +/dp b cn bw ♀ X dp b cn bw/dp b cn bw ♂

Sl. No.	Treatment (X-Chromosome)	Total No. scored	dp-b Mean of cross over%	b-cn Mean of cross over%	cn-L^2 Mean of cross over%	L^2-bw Mean of cross over%
1.	+/+ (control)	2226	18.00±0.975	3.65±0.314	6.39±0.573	15.33±1.408
2.	y sc^{S1} In-S sc^{8}/ + + + +	2310	17.39±0.863	7.84±0.657	10.19±0.665	14.06±1.569
3.	y sc^{S1} In-S sc^{8}/ y sc^{S1} In-S sc^{8}	1084	16.31±1.407	7.15±0.721	9.94±1.528	14.16±1.582

Table 2. Analysis of crossing-over in the cross Cy/dp b cn bw ♀ X dp b cn bw/dp b cn bw ♂

S.No.	Treatments	Parental types		Recombined types		Cross-over%
		Cy ++++/ dp b cn bw	dp b cn bw/ dp b cn bw	Cy ++ cn bw/ dp b cn bw	dp b ++/ dp b cn bw	
1.	+/+ (Control)	712	655	-	-	-
2.	y sc^{S1} In-S sc^{8}/++	1538	1201	22	20	1.53
3.	y sc^{S1} In-S sc^{8}/ y sc^{S1} In-S sc^{8}	821	612	10	10	1.39

Patton, J. L. and W. B. Heed. University of Arizona. Elevational differences in gene arrangements of D. pseudoobscura in the Santa Catalina Mountains, Tucson.

On April 12 and 26, 1964, D. pseudoobscura males were collected from four different elevations and habitats in the Santa Catalina Mountains near Tucson and crossed to laboratory virgin females containing the Standard (ST) gene arrangement kindly supplied by Dr. Th. Dobzhansky. A total of 100 pair matings were made from each locality and an attempt was made to analyze both homologues of chromosome III by scoring at most six larvae from each pair. Thus, the ideal number of chromosomes from each locality is 200; the table shows that all pair matings were not successful. Soldiers Trail is at 3000' in the Lower Sonoran Desert of paloverde and sahuaro. Molino Basin is at 4500' in the Upper Sonoran Chaparral of juniper, piñon pine and scrub oak. Windy Point is at 6000' in the Upper Sonoran Oak-Pine Woodland. Organization Ridge is at 7200' in the Transition Zone of almost pure Ponderosa pine. April 12 at Windy Point and Organization Ridge was the earliest possible collecting date for 1964. There is no significant difference of inversion frequencies between dates within any locality and there is no significant difference between adjacent elevations. However, the inversion frequencies in every other elevation are significantly different at the 5% level using a 4 x 2 contingency test. The Arrowhead (AR) and Chiricahua (CH) inversions both steadily increase in frequency with increase in elevation and at 7200 feet they do so at the expense of Pikes Peak (PP) which was also increasing in the same direction. The elevations of greatest diversity of genotypes are also the areas probably most ecologically diverse for pseudoobscura, Molino Basin and Windy Point, the Pikes Peak gene arrangement being the indicator.

	N	AR	ST	CH	PP
Soldiers Trail					
April 12	106	77.35	16.04	4.72	1.99
April 26	92	80.44	11.94	5.44	2.18
Total	198	78.79	14.14	5.05	1.01
Molino Basin					
April 12	142	82.41	8.44	6.33	2.82
April 26	56	83.93	7.14	5.36	3.57
Total	198	82.82	8.08	6.06	3.03
Windy Point					
April 12	64	85.96	1.56	7.80	4.68
April 26	116	85.34	2.58	7.76	4.32
Total	180	85.56	2.22	7.78	4.44
Organization Ridge					
April 12	44	86.34	2.28	11.38	-
April 26	130	89.23	2.31	8.46	-
Total	174	88.51	2.30	9.19	-

Kim, K. W. Chunnam National University, Korea. Chromosomal studies of Korean Drosophila species.

In order to examine the karyotype and its intraspecific variation, a number of strains of several different species of Korean Drosophila were investigated.

Larval ganglion smear method were used for the determination of the metaphase chromosome configuration.

5 species were checked, among which D. immigrans Sturtevant (Kwangju strain) is quite similar to type 1 reported by Clayton and Ward (1954), but differs from it in having J-shaped chromosome instead of V-shaped chromosome. The karyotype of D. brachynephros Okada (Chiri strain) consists of 5 pairs of rod-chromosomes and a pair of dot-chromosomes, and this is the same configuration of D. transversa Fallen. The remaining 3 species, D. melanogaster Meigen, D. auraria Peng, D. virilis Sturtevant were identical with the karyotypes reported by previous workers.

Mukherjee, A. S. Max Planck Institut für Biologie, Germany. Cytological localization of the white locus in D. hydei.

Analysis of the salivary gland chromosomes of white-mottled-1 (w^{m-1}), white-mottled-2 (w^{m-2}) and white-mottled-3 (w^{m-3}) shows that although the three mutants differ phenotypically from each other in their extent of mottling, the distal break is, in every case, between 17A3 and 17A4 (numbering of bands and segments is according to Berendes, Chromosoma, 14, 1963). This breakpoint is close to a heavy doublet, the 17A(1-2).

While the w^{m-1} and w^{m-3} are cases of transposition of the heterochromatic arm of the X chromosome and nucleolar organizer into the region of the distal break (17A3-17A4), the w^{m-2} involves a compound double inversion. Other breakpoints are given below:

w^{m-1} and w^{m-3}: Proximal break is in the heterochromatin most likely at the end of the euchromatic arm between the centromere and the band segment 1A (In hydei one arm of the X is wholly heterochromatic: Figure 1). The difference between the two mutants may be the consequence of differential transposition of the heterochromatin.

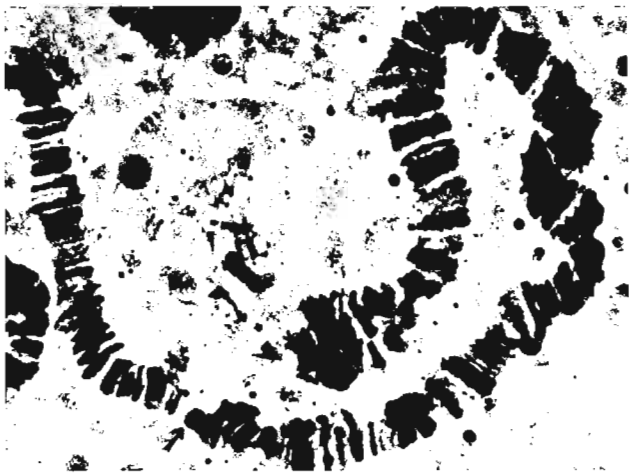


Figure 1: X-chromosomes of w/w^{m-1}. Arrow indicates the suggested white locus.

w^{m-2}: Set I: Distal break same as above; proximal break is between 1A and the centromere; Set II: Break 1= between 9A4 and 9B1; Break 2= in the heterochromatic arm; this last break shifts the centromere to the region 9A4-9B1 and perhaps splits the nucleolar organizer (Figure 2).

Frequent heterochromatinization is observed extending from the break to about 16D4 on the one hand, and 17A6 on the other. Whenever heterochromatinization is found the 17A (1-2) is always involved.

These results, together with the finding that white-deficiency (w^{df-686}) shows a deletion for the bands from 16D3 to 17A(1-2), strongly suggest that 17A(1-2) is the white locus. The doublet nature of the white locus and the Notch phenotype of the deficiency (w^{df-686}, male lethal), suggests a homology between this region in this species and that in D. melanogaster.

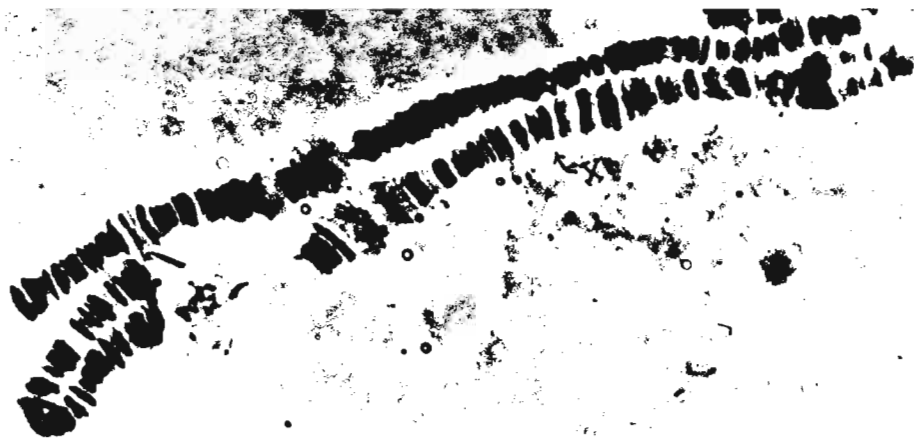


Figure 2: X-chromosomes of w^{m-2}/w^{m-2}.

Brosseau, G. E., Jr. University of Iowa.
Somatic pairing in structural heterozygotes.

Somatic pairing and crossing-over in Drosophila have been known and studied for a long time. However, little or no information is available concerning somatic synapsis in structural heterozygotes. The nature of pairing of structurally dissimilar homologues should give additional insight into the mechanism of somatic pairing and permit comparisons with meiotic pairing. Neuroblast squash preparations from larvae bearing chromosomal aberrations were examined to determine how well these chromosomes would pair somatically. No counts were made and relatively few (15-30) figures of each type were studied. Three different heterozygotes were examined: Ins(2L+2R)Cy/Bl, In(3LR) Ubx_{130}/Tp(3:3) Vno and T(2:3)Xa/In(3LR)Ubx_{130}. In general, somatic synapsis was fairly regular in all of the structural heterozygotes when compared to the control (Oregon R), although some incomplete synapsis and even asynapsis was observed. Incomplete synapsis was never seen in the Oregon R preparations. Pairing was least complete in T(2:3)Xa/Ubx, although it was fairly good here also. The chromosomes always paired as "bivalents," never as a multivalent. A bivalent with one arm paired and the other unpaired was often seen; pairing was usually complete in the other bivalent in these cells. Complete asynapsis of one bivalent was occasionally seen. It is, of course, impossible to identify chromosome arms in these preparations and thus to determine which arms are paired with each other; however, it does seem apparent that a good deal of nonhomologous pairing must be occurring.

Pairing in the inversion heterozygotes appeared to be quite good, however, without any indication of inversion loops as would have been found in the salivary gland chromosomes. Thus nonhomologous pairing is the rule with inversion heterozygotes. As in the case of the translocation, some incomplete synapsis and asynapsis was observed. Although specific chromosomes are difficult to identify, in the Ubx/Vno heterozygote a shift in the position of heterochromatin has occurred and thus it was possible to see in a few favorable prophase figures that when incomplete synapsis occurred, it is the normal 2nd chromosomes which are paired and the aberrant 3rd chromosomes which are experiencing difficulty in synapsis. When the inversions pair, they show the typical somatic "chiasmata." No indication was found that somatic synapsis occurs best or preferentially in heterochromatin.

While these observations do not comprise a thorough analysis of the somatic pairing of structurally dissimilar chromosomes, they should be of interest to workers interested in somatic crossing over and related phenomena. It seems clear that somatic synapsis in neuroblast cells (and presumably other mitotically active somatic cells) is very different from meiotic synapsis or even pairing in salivary gland chromosomes. Extrapolations from knowledge concerning pairing in these latter cells to account for various events occurring in mitotic cells should be made with caution. (Supported by USPHS Grant RG 06508-05).

Brosseau, G. E., Jr. University of Iowa.
The sequence of loci on $B^{S}Yy^{+}$ and $y^{+}Y$.

$B^{S}Yy^{+}$ was obtained by crossing over between $B^{S}Y$ and $bw^{+}Yy^{+}$, which in turn is derived from $y^{+}Y$ by the transfer of y^{+} from Y^{L} to Y^{S}. $B^{S}Y$ was originally derived from an induced detachment of an attached-X, followed by the introduction of $T(1:4)B^{S}$ by crossing over and subsequently deleting the euchromatin between B^{S} and the proximal heterochromatin. Thus this very useful marked Y chromosome has a history of several induced and spontaneous alterations. It would be useful to know, therefore, whether the sequence of the known Y-linked loci (the fertility factors and bb^{+}) has been altered by any of these changes. While the exact sequence of these loci has never been determined for a normal Y chromosome, it is known for $y^{+}Y$. The sequence of Y chromosome loci can be ascertained by analyzing exchange products between the X and Y chromosomes recovered as detachments of the attached-X. If the sample of detachments includes an array of exchange points along the length of the Y, an unequivocal ordering of the loci can be made by means of standard tester stocks. A number of detachments (115) involving exchange with $B^{S}Yy^{+}$ was available from another study. Analysis of these detachments reveals the following sequence: B^{S}, kl-5, kl-4, kl-3, kl-2, kl-1, bb^{+}, ks-1, ks-2, y^{+}. The position of the centromere cannot be determined by this technique; however, it is assumed to lie between bb^{+} and Kl-1. This sequence is identical to that of $y^{+}Y$ except that in $y^{+}Y$, y^{+} is distal to kl-5 and Y^{S} has no X derived terminal marker. Therefore, it seems likely that the various alterations which occurred in going from $y^{+}Y$ to $B^{S}Yy^{+}$ were restricted to the regions of the Y distal to the fertility gene complexes. Supported by USPHS Grant RG 06508-05.

Hiraizumi, Yuichiro and Kyoko Nakazima. National Institute of Genetics, Japan.

SD in a natural population of D. melanogaster in Japan.

From the beginning of July, 1963, wild flies of D. melanogaster from a natural population in Ohdate City were collected periodically till the end of November, 1963, when fly collection became impossible because of the cold temperature. The City of Ohdate, Akita Prefecture, is located in the northern part of the Mainland of Japan (Honshu), about 400 miles to the north of the Genetics Institute in Mishima. A person residing in Ohdate City had been engaged in fly collections since July, 1963, but the earliest time when D. melanogaster appeared in that area was the end of August. Males collected from the Ohdate population (constitution: $+_1/+_2$ for the second chromosome) were individually mated with virgin cn bw females. From the F_1 progeny of each cross, five males were collected and mated individually to cn bw females. In the F_2 generation we expect the ratio of phenotypically cn bw to wild flies to be 1 : 1 if the $+_1$ and $+_2$ chromosomes behave normally. In fact, this was so for about 600 tested males. However, there were 8 exceptional cases. In these exceptional males some of the wild second chromosomes behaved as if they carried the segregation-distorter (SD) locus, i.e., they revealed extreme excess of + flies in the F_2 generation (practically 100% instead of its expected value of 50%). The number of tested males, SD-carrying males and the time of fly collections are presented in Table 1. As can be

Table 1: The number of tested and SD-carrying males, and the time of fly collections in 1963.

No. of tested males	No. of SD males	Time of Collection
12	0	Aug. 23 - Aug. 28
120	2	Sept. 3 - Sept. 7
63	1	Sept. 12 - Sept. 15
230	2	Sept. 23 - Sept. 27
73	1	Oct. 4 - Oct. 10
57	1	Oct. 17 - Oct. 20
48	1	Oct. 30 - Nov. 3
0*	0	Middle of Nov.
1	0	End of Nov.
Total 604	8	

*fly collections were continued but no melanogaster was captured

seen, SD-carrying males were found throughout the season. Some of the SD^+ chromosomes in the Ohdate population were insensitive to the action of SD, or there were systems suppressing SD-action. Salivary gland chromosome examination revealed that all of the 8 SD-bearing chromosomes carry two overlapping inversions, one involving the sections from 52A to 56F and the other from 55E to 60E. (These are approximate; exact determinations have not yet been made.) The former inversion is identical to that found in SD-72 and SD-5. This inversion is also fairly commonly found in natural populations of D. melanogaster and is called Nova Scotia (In (NS)). One hundred and ninety-three wild, SD^+, second chromosomes from the Ohdate population were examined as to whether they carried any inversions. Only 11 were found to carry In (NS), and the remaining 182 were free of any inversions. Many genetic tests are in progress in the author's laboratory, and it is hoped that the present investigation will give a hint on the origin and evolutionary history of SD, as well as on the mechanism of SD action.

Kikkawa, H. Osaka University, Japan.

An electrophoretic analysis of amylase gene in D. simulans.

By using an agar-gel electrophoresis which was improved by Ogita (DIS 37:142), there were found two types of amylase isozymes, viz., Amy^F and Amy^S among eight strains of D. simulans. A genetic analysis showed that these amylase isozymes are controlled by allelic and co-dominant genes located near plum gene on the second chromosome of this species.

Malich, C. W. and R. Binnard. NASA Ames Research Center, California. Observations on Drosophila given a high dose of protons.

Inseminated females of a strain of D. melanogaster homozygous for dumpy were exposed in three sets of 100 each to 30,000 rads of 50 Mev protons, a week after eclosion and three days following mating. Each set was transferred to a separate bottle immediately following treatment, and two hours later all the flies were parceled out into nine vials. These P_1 were transferred to fresh food about every three days. Some were remated after three days to compare the fertility of females having fresh sperm to those having only the original irradiated sperm.

The flies were very lethargic after the irradiation but recovered in a few hours. While many eggs were laid in the early broods, only four hatched. Neither the flies with fresh sperm nor those with treated sperm produced any larvae after the third day, indicating heavy damage to the oocytes and oogonia. Three larvae from the second brood of one set pupated, but these died before eclosion. One larva from the first (two hour) brood of another set produced a phenotypically normal female which eclosed much later than normal. This single F_1 offspring when mated to untreated males gave 49 F_2 males and 50 F_2 females. All four expected classes were present in the second generation in roughly equal numbers, and no evidence for radiation induced mutation was found.

It is extremely unlikely that the irradiation was so non-uniform that any of the flies excaped the high dose of 30,000 rads. The proton beam was scattered by 1/8" of carbon to produce a nearly constant flux over a circle of diameter 1 1/2", and only the central 1" disk was used. In addition, the beam was oscillated rapidly (several hundred cps) to smooth out any minute irregularities. The dose delivered corresponds to approximately 1260 protons per square micron, so that each chromosome should have received multiple hits.

The median life of the flies after this dose of protons was shorter than normal, half dying in 18 days compared to the shortest period of 21 days observed for virgins under similar crowded conditions. Fourteen days after eclosion 222 of the original 300 were alive, 172 lived 17 days, 121 lived 20 days and 3 were alive at an age of 32 days. The food was rather damp and since this dumpy strain gets stuck easily, the environmental conditions may have affected the lifetime more than the radiation. Consequently we conclude from this series only that the sterilizing effect was virtually complete.

Degenerative effects associated with high radiation doses were evident in five flies sacrificed for histological studies. Two flies dissected 17 days after treatment had degenerate ovaries and their oogonia were vacuolated and necrotic. Live motile sperm were seen in the seminal receptacle of the fly taken from a vial with mates, while no sperm were found in one which had been without mates for a week. The three flies which lived 32 days were dissected, and showed more advanced degeneration of the ovaries and the oogonia than the younger pair. No sperm were seen in these flies, which had been without mates for 10 days. Further work with lower doses has suggested other non-reparable damage produced by protons, and some biochemical effects on the brain of adults are being investigated.

Scharloo, W. Genetisch Laboratorium der Rijksuniversiteit, The Netherlands. Temperature sensitive periods in ci^D.

Scharloo and Nieuwenhuis (DIS 37 and 1964) reported that the temperature sensitive period (T.S.P.) of ci^D lies after puparium formation, and they found no evidence for an influence of the genetic background. However, new experiments with two long inbred ci^D lines having oppositely directed temperature reactions revealed that the genetic background can affect the localization of the T.S.P. in development. Unpigmented prepupae were transferred from 27.5° to 17.5°. In both lines the T.S.P. for the 4th vein interruption ended before puparium formation. A further experiment on an inbred line related to the H-stock of our earlier experiments showed again a T.S.P. starting several hours after puparium formation. The T.S.P. for the 5th vein interruption was invariably located after puparium formation in all lines.

It is perhaps relevant that the 5th vein had an interruption of about the same size at 27.5°, and was complete at 17.5°, in all lines. Further, both strains with a 4th vein T.S.P. before puparium formation, show a change of expression in that part of the scale where expression and temperature have a linear relation. In both stocks with the T.S.P. after puparium formation, the expression is less extreme and is changed through the expression range where a marked facilitation of change occurs (see Scharloo 1962).

de Mazar Barnett, Beatriz K. Comisión Nacional de Energía Atomica, Argentina. Recessive lethals induced in sperm by X rays, alkylating agents and combinations of both.

A comparison was made of the frequencies of sex-linked recessive lethal mutations induced in mature sperm by (a) X rays, (b) two alkylating agents and (c) a post-irradiation 30 minutes after administration of the chemicals. The alkylating agents used, a nitrogen mustard (NITROMIN) and a polyethylene-imine (THIO TEPA) were injected intra-abdominally in a 0.4% saline solution and the irradiation dose was 800r in all cases.

For the experiments, one day old Oregon-R males were treated and 24 hours later mated to "Basc" females, left for one day and then discarded. The females were allowed to oviposit for two additional days.

Standard recessive lethal tests were made with the F_1 females. The results obtained in the F_2 are shown in the table. Taking into consideration these results, as well as others from combined treatments carried out at different intervals, the effect of combined treatments with alkylating agents and X-rays is at least additive with the nitrogen mustard but not with the polyethylene-imine, in which case no combined treatment has ever induced a recessive lethal frequency as high as that induced by the chemical alone.

	Alkylating agent		X rays		Alk. a. X rays 30 m interval	
	No. chrom.	%let.	No. chrom.	%let.	No. chrom.	%let.
NITROMIN (7×10^{-2}M)	788	1.14	1084	1.92	951	4.94
THIO TEPA (3×10^{-2}M)	983	12.00	1084	1.92	693	6.63

de Mazar Barnett, Beatriz K. Comisión Nacional de Energia Atomica, Argentina. Extreme variability in oviposition rate.

An attempt is being made to establish the frequency of dominant lethals induced in mature or nearly mature oocytes of females treated with various combinations of X-rays and chemical mutagens, in the hope of elucidating previous data on recessive lethals induced by these agents. Individual females were mated in vials containing a special medium for egg counts (see note in this issue DIS) and were allowed to lay eggs for 36 hours (this time had to be used since it was the period used previously for the recessive lethal tests). It was found that a variable proportion of the females did not lay eggs at all during the 36 hour period (though most of them did finally lay eggs later). This was true of all groups including the controls.

In addition, there was an extreme variation in the number of eggs laid by the individual females, again in all groups. In some treatments, the range was from 1 to 100, in others 1 to 50, etc. and the distribution of egg number was rarely a normal one.

Schneider, Imogene. Yale University. Inadvisability of using the raft technique for Drosophila organ culture.

In a recent review, the observation was made that although insect organs have been cultured in hanging drops and on solid media for in vitro differentiation studies, there has been no report in the literature of using the rayon raft technique for this purpose, the latter being a very common practice in vertebrate organ culture (M. Martignoni in Insect Physiology, Oregon State Univ. Press, 1963).

This technique was attempted a number of times in culturing D. melanogaster organs (cephalic ganglia with attached eye-antennal discs, salivary glands, testes and ovaries) using not only rayon rafts but also rafts of nylon monofilament cloth, perforated cellophane and millipore filters. Regardless of the material which served as the raft, the end result was unsatisfactory. Except for testes and ovaries from young third instar larvae, all the above-mentioned

organs, whether from third instar larvae, prepupae, or pupae, immediately flattened out when placed on such rafts. After 24 hours in culture most organs were so distorted that it was difficult to visualize their original outlines. None of the organs survived as long as their controls in hanging drops, nor was there, with few exceptions, any significant differentiation. Millipore filter rafts had one further disadvantage in that it was extremely difficult to see the explants against the solid white background. Placement of Drosophila organs at an air interface within the culture chamber should therefore be avoided since such an arrangement promotes neither long survival nor extensive differentiation and as such is decidedly inferior to complete submersion of explants in a liquid medium.

Ayala, Francisco J. Columbia University.
Development of incipient sexual isolation between laboratory populations.

Two strains of D. birchii from Cairns, Australia, and from Popondetta, New Guinea (Ayala, 1965a), were crossed in both directions. Two populations were started with 150 pairs of F_1 hybrids each, one being maintained at 25°C and the second at 19°C. The technique of maintaining the populations has been described elsewhere (Ayala, 1965b). After 48 weeks of "natural" selection (about 16 generations at 25° and 10 generations at 19°C) samples were taken from both populations, and crossed to each of the parental strains in the two possible directions. The test was made by placing 10♀♀ and 10♂♂ together in a 1/2 pint bottle for 10 days, and then the females were dissected and examined for presence of sperm. The results are given in table 1. (The control crosses give 90% or more insemination). The two hybrid

Table 1: Sexual preferences between two hybrid experimental populations and their parental strains.

Cross	♀♀ tested	♀♀ inseminated	Per cent inseminated
Hybrid population 25° x Cairns	64	14	22
Hybrid population 25° x Popondetta	57	26	46
Hybrid population 19° x Cairns	55	40	73
Hybrid population 19° x Popondetta	60	31	52

populations show a similar moderate degree of sexual isolation with the Popondetta parental strain. However their behavior with the Cairns parental strain is strikingly different, the 25° population showing fairly high isolation and the 19° very little. The difference in isolation of the 25° and 19° populations with Cairns is highly significant (χ^2=30, P<<.001), and the difference between the behavior with Cairns and with Popondetta is significant at P<.01 for the 25° hybrid population and at P<.05 for the 19° population. The two populations have, then, evolved different degrees of sexual isolation with respect to the parental strains. Since the populations were kept completely separated, sexual isolation has evidently arisen as a by-product of genetic divergence.

Literature: Ayala, F. J. 1965a. Sibling species of the Drosophila serrata group. Evolution, in press. Ayala, F. J. 1965b. Relative fitness of populations of Drosophila serrata and Drosophila birchii. Genetics, in press.

Ayala, Francisco J. Columbia University.
Improvement of fitness in experimental populations.

Two experimental populations, one maintained at 25°C and the second at 19°C, were started with 150 pairs of founders taken from mass culture stocks of the Popondetta strain of D. serrata. The technique of maintaining the populations has been described elsewhere (Ayala 1965), the relevant factor being that strong competition exists

both between the adults and during the immature stages of development. The populations reached equilibrium between the 8th and the 15th week, and thereafter oscillated around the same level. After 52 weeks samples were taken from both populations and from the original mass culture stocks, and a new generation was produced at 25°C under uncrowded conditions. From this progeny different amounts of flies were placed in 1/2-pint culture bottles, and transferred regularly to fresh bottles (3 times a week at 25°C, twice at 19°C). A total of 10 transfers were made at 25° for each level of parental density, and 6 transfers at 19°C. The mean number of flies produced per bottle are presented in Table 1. In all the six cases studied the flies

Table 1: Mean number of flies produced per food unit by two experimental populations and their parental stocks.

Number of Parents	Number of flies produced per bottle					
	25°C			19°C		
	Experimental Population	Control	Difference	Experimental population	Control	Difference
100	396	363	33	233	208	25
300	423	381	42			
1000	513	495	18	477	379	98
1500	543	497	46			

from the experimental populations produced more progeny than the controls. It seems likely that, under the action of strong natural selection, new genotypes have been selected in the experimental populations which improved their fitness, as measured by the number of progeny produced per food unit.

Reference: Ayala, F. J. 1965. Relative fitness of populations of Drosophila serrata and Drosophila birchii. Genetics, in press.

Thompson, Peter E. Iowa State University.

The killing and resorption of eggs after injection of Drosophila females with actinomycin-D.

In the course of tests of the effect of actinomycin-D on crossing-over, females were injected with about 0.25 microliter of dissolved material at concentrations of 10 and 50 microgram/milliliter. Injection was via the thorax. Neither concentration had a marked effect on survival under sterile conditions. While the 10-gamma solution did not reduce fertility, the 50-gamma solution invariably led to a permanent or temporary cessation of egg laying.

In the 50-gamma series, females were inseminated during their first imaginal day and injected at 1 1/2 days. Each female laid 2-5 eggs soon after treatment, after which oviposition ceased for at least 6-7 days. Following this lapse, roughly one-third (37/124) of the females showed a recovery of fertility. The pattern of crossover effect after recovery was comparable in its time scale to effects of the 10-gamma treatment, heat treatments, etc., as if no marked retardation of survivng oocytes were involved. This included the observation of multiple rare exchanges, presumably gonial in origin, in individual females from the tenth day on.

Females examined early in the sterile period show degeneration of all advanced egg stages, and the ovary is distended with decomposition products. At day 6 (about 4 1/2 days after injection) most of this debris has been resorbed and oocytes up to about stage 3 or 4 can be seen. Again, the rate of development of these surviving oocytes must be nearly normal, for their maturation is complete in some females within another two days, at the return of fertility. A more thorough treatment of this aspect is projected, hopefully to establish a time scale of oogenesis similar to that found by Welshons and Russell (PNAS 1957) in their irradiation of Drosophila males. (Research supported by U.S.P.H.S. Grant GM 08912).

Strömnaes, Öistein and Ingerid Kvelland
University of Oslo, Norway. Radiosensitivity of sperm ejaculated within 12 hours after irradiation.

Unmated D. melanogaster males from an Oslo wild-type stock were irradiated with 3000 r of X-rays when the males were either 12, 24 or 72 hours old, and then mated individually to virgin M5 (Muller-5) females. All matings were observed and recorded. Copulating females were immediately isolated and after impregnation transferred to egg laying tubes. Thus, the frequency can be calculated of sex-linked recessive lethals in individual sperm ejaculates.

Analysis of the data (Table 1) indicate that the frequency of sex-linked lethals increase with age of males at time of irradiation. This is in agreement with previous findings.

Table 1: The frequency of sex-linked recessive mutations in successive ejaculates after irradiation.

	Ejaculate sequence							
	First		Second		Third		Total	
Series	1/N	% 1	1/N	% 1	1/N	% 1	1/N	% 1
12 A	665/9137	7.28	70/1168	5.99	11/138	7.97	755/10575	7.14
24 A	1356/16563	8.19	422/6795	6.21	117/2277	5.14	1918/26067	7.36
72 A	795/9259	8.58	286/3635	7.86	61/603	10.11	1145/13523	8.47

It can also be seen from the table that all age groups of males have a lower frequency of lethals in their second ejaculate than in the first ejaculate. The data are not consistent in the third mating for the three age groups of males. The 24 hour old males (Series 24 A) exhibit a further lowering in the frequency of lethals from the two previous matings; while the younger as well as the older males (Series 12 A and 72 A) have a higher frequency of lethals in their third ejaculate than in their second ejaculate. The data for 12 and 24 hour old males (Series 12 A and 24 A) are in agreement with previous findings, while we have no data directly compatible with those obtained in Series 72 A.

The data have not been analyzed in detail yet, but a preliminary examination of the data suggests that for the youngest age group of males (Series 12 A) the frequency of lethals in the first ejaculate depends on the time after irradiation. Thus, first ejaculates available for fertilization the first eight hours after irradiation exhibit a higher frequency of lethals than do first ejaculates available for fertilization eight to twelve hours after irradiation.

Gfeller, S. M. David. University of Oregon. The action of an autosomal modifier on the pteridine of the mutant, white-blood (w^{bl}).

The action of an autosomal recessive factor (Clancy, 1964, DIS 39:65) which affects the production of the pteridine eye pigments (Clancy, 1962, Gen. 47:948-949) in Drosophila is being studied with respect to the mutant, white-blood (w^{bl}), a temperature sensitive allele of the white series. Preliminary experiments involving comparative measurements of the fluorescing pigments resolved by paper chromatography (Hadorn & Mitchell, 1951) reveal that at both 25°C and 18°C all of the pteridines are significantly reduced in w^{bl} when homozygous for the modifier. A similar result is obtained with a stock homozygous for the modifier, but otherwise wild type in appearance and constitution.

Comparative measurements have also been made at both temperatures on the compounds of w^{bl} with vermilion (v) and scarlet (st), genetic blocks to ommochrome formation. The pattern of action of the modifier is not the same with these two compounds, nor is it the same as that found with w^{bl} alone. The above patterns are quite consistent in flies from five to sixteen days of age.

All measurements were made on head pigments of male flies derived from stocks in which the female parents were attached-X and wild type with respect to w^{bl}. Preliminary experiments involving males derived from stocks in which the mothers were also w^{bl} have given results which indicate the possibility of a maternal effect with respect to the interaction of the pteridine modifier and the eye color mutant. (Work supported by training grant No. 5T1-GM-373 and research grant GM09802, USPHS).

Bateman, A. J. Christie Hospital, Manchester, U. K. An attempt to induce ND of chromosome 2 by X-raying ♂♂.

dp b cn bw/b pr vg ♂♂ were irradiated with 1000 and 1500 rad and mated to 2L dp.2R px ♀♀, daily for 12 days. The matings were very infertile: 4000 mated ♀♀ produced only 68 progeny. Expected ND phenotypes in the progeny were dp px (from nullo-2 sperm) and b $+^{px}$ (from diplo-2 sperm). Only 4 such flies were found among a total of 68. It would seem that nullo-2 and diplo-2 sperm is non-functional. Moreover, no progeny have been produced from irradiated sperm with the normal complement from which chromosome 2 has then been lost at first cleavage. Perhaps the 4 expected gametic classes are not formed at meiosis in 2L.2R eggs, or are formed in unequal proportions so that nearly all haploid egg nuclei are 2L or 2R. This would explain the moderate yield of progeny (17 proven) with one paternal iso-2, all from X-raying pre-meiotic stages.

Of the remaining 47 progeny, 7 were proved to contain a (spontaneously reconstituted) dp px chromosome. Of the remainder the majority were proven triploids, and were produced with equal readiness from pre- or post-meiotic irradiation of sperm. It seems an open question whether these are due to spontaneously produced diploid eggs or to dispermy in a 2L.2R egg with loss of one paternal chromosome 2.

Watson, J. E. and Allan B. Burdick. Purdue University. The effects of selected background rearrangements on the v to g region in D. melanogaster.

In studies dealing with the nature of crossing-over in the miniature-dusky region on the X-chromosome the data presented in table 1, showing the interchromosomal effects of the listed rearrangements, was derived. These data show the alteration effect on the recombination distances between v, dy^{60k} and g and are presented here for the information of others working in this and other adjacent regions of the X-chromosome. Of interest also is the effect the several rearrangements have on the mean number of offspring produced by a single female.

The detailed description of this study may be found in: Watson, J. E., 1963, "A Comparative Study of the Inter-locus and Intra-locus Response to Chromosomal Rearrangements in D. melanogaster;" M. S. Thesis, Purdue University Libraries, Lafayette, Indiana.

Table 1. Recombination data derived from v dy^{60k} g/+ ++ females

Rearrangement	Progeny	v-dy^{60k}	dy^{60k}-g	Coef. of coincidence	Mean no. of progeny per female
Control	14,199	3.08 ± .15	6.59 ± .21	.034	237
In(2LR)Rvd	5,029	3.09 ± .25	6.16 ± .34	.209	93
In(2R)Mo^{k}	3,206	3.34 ± .32	6.36 ± .34	.103	146
In(2LR)dp	3,584	2.93 ± .28	6.86 ± .43	.139	63
In(2L)NS	12,599	4.17 ± .18	6.83 ± .23	.084	238
In(2LR)Gla	5,706	4.45 ± .28	7.12 ± .34	.387	114
Ins(2L+2R)NS	5,885	4.23 ± .26	7.34 ± .34	.164	101
SM5	7,061	4.32 ± .24	8.10 ± .33	.000	126
In(3L)D	5,451	3.17 ± .24	6.13 ± .33	.094	99
In(3R)Hu	11,766	3.66 ± .17	6.37 ± .23	.269	294
In(3LR)Cx	6,471	3.54 ± .22	7.48 ± .33	.058	175
In(3LR)Ubx^{101}	7,301	3.48 ± .22	7.96 ± .32	.495	183
In(3L)Me'	5,475	4.24 ± .27	7.32 ± .36	.235	140
TM3	10,956	4.58 ± .20	7.67 ± .26	.078	288
Ins(3L+3R)P	4,660	4.06 ± .29	8.37 ± .40	.063	83
(2L+2R)NS-TM3	4,383	4.02 ± .33	9.54 ± .43	.049	115
SM5-Ubx^{101}	1,727	4.17 ± .48	10.42 ± .74	.000	69
SM5-Cx	1,375	5.45 ± .61	11.05 ± .85	.000	65
SM5-TM3	1,706	5.80 ± .57	10.79 ± .77	.094	11
SM5-P	3,873	5.55 ± .37	12.14 ± .52	.077	76
(2L+2R)NS-P	8,961	5.65 ± .24	12.39 ± .35	.096	172

The underlined values differ significantly from the control at $\alpha = .05$.

Bateman, A. J. Christie Hospital, Manchester, U. K. X-irradiation of heterozygous attached-X.

The attached-X used had arisen in progeny from sc ec cv/ct v g ♀♀ which had been irradiated. It was homozygous for sc ec and cv, and heterozygous for ct v and g. ♀♀ were irradiated with 4000 rad or kept as unirradiated controls. The progeny of eggs laid on days 7-11 after irradiation were scored for homozygosis for ct v and g, with the following results:

		Homozygous for			
	Total ♀♀	ct	v	g	Detachments
Control	1167	81	80	39	None
as %		6.9	6.9	3.3	
4000 rad	534	38	81	56	10
as %		7.1	15.2	10.5	
Increase over Control		0.2	8.3	7.2	

It is seen that 4000 rad produces a 3-fold increase in c-o between g and the centromere. The increase in homozygosis for v is the same as for g, meaning that there has been no change in c-o between v and g. The absence of any change in homozygosis for ct means that c-o between ct and v must have decreased by an amount equal to the increase proximal to g. These results confirm those presented in DIS 37:68 in respect of segments ct - v - g and add the additional information that X-rays enhance c-o near the centromere (to the right of g). The controls show relatively low homozygosis for ct. This is in all probability due to some parental attached-X ♀♀ being homozygous $+^{ct}$.

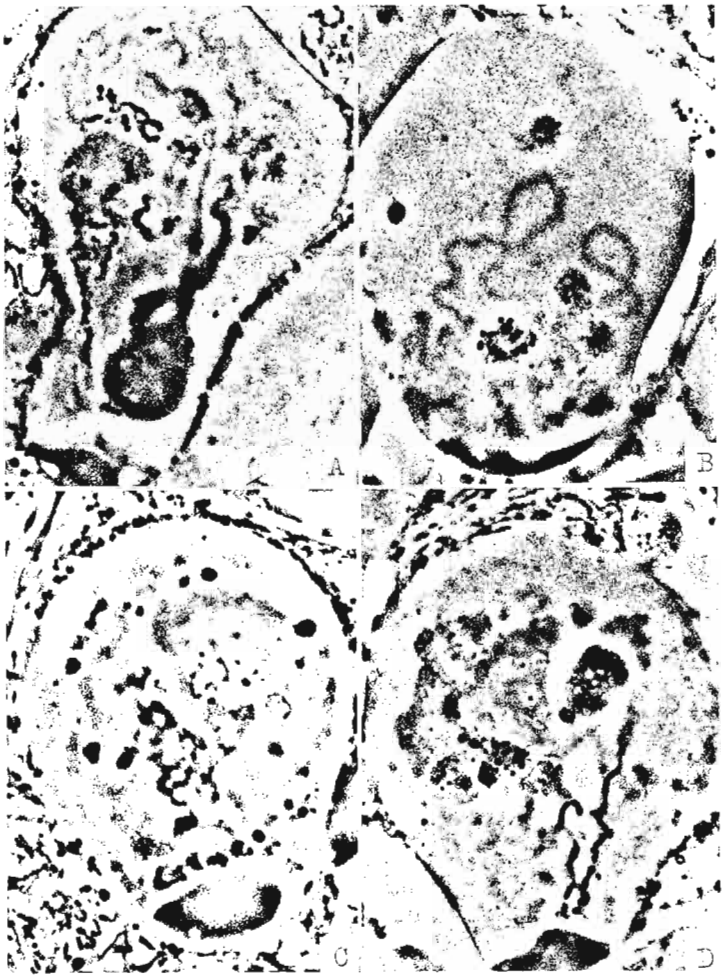
Bateman, A. J. Christie Hospital, Manchester, U. K. ND of chromosome 2, production of iso's, and reconstitution of normals from iso's by X-irradiation of ♀♀.

For these experiments we used an iso-chromosome stock (2L dp. 2R px) supplied by the Institute of Genetics at Pavia. ♂♂ of the stock were mated to irradiated ♀♀ of constitution dp b cn bw/b pr vg. The progeny obtained were dp px (from nullo-2 eggs) b + ^{px}(from diplo-2 eggs) dp + ^{px} (egg with 2R iso) and b px (egg with 2L iso). The greatest yields were obtained over the first 8 days of laying after doses of 2-6 K rad. Nullo-2 eggs accounted for 66% of all progeny and diplo-2 eggs for 9%. This is the ratio found for X-ray induced ND of the X chromosome. We were surprised to find 25% of all progeny were from iso-2 eggs (50/50 2L and 2R). Triploid progeny were very rare. These induced iso's are homologous to attached-X, which however are rarely produced by irradiation. Of the diplo-2 eggs, only 5% were homozygous at the centromere. By contrast, 25% of the iso's were homozygous at the centromere. It is concluded that 25% of the iso's arise by misdivision of the centromere (sensu lato) and 75% by centric fusion (sensu lato).

Reciprocal matings, with irradiation of 2L dp.2R px ♀♀, freely produced flies of constitution dp px/b. Thus a normal second chromosome has been reconstituted from two iso's. The reconstituted chromosome does not appear to be deleterious. The heterozygotes are fertile and the homozygotes viable and fertile. The ease of reconstitution is suggestive of a close association between the centromeres of the iso's (which are of course homologous) with X-ray induced c-o within the centromeric zone.

Meyer, G. F. Max-Planck Institut für Biologie, Germany. A reliable cytological method for classification of Drosophila species.

The recently demonstrated lampbrush-like modifications of the Y chromosome within the genus Drosophila are both phase and species specific (Hess and Meyer, 1963; Meyer, 1963) and can, therefore be used as a reliable taxonomic criterion. Numerous representatives of several subgroups have been examined and the results demonstrate the strict species specific evolution of the Y heterochromatin in primary spermatocytes. For observation of the functional structures of the Y chromosome in living spermatocytes testes are dissected in Drosophila Ringer (also last instar larvae can be used), transferred into a drop of Drosophila Ringer on a slide and covered with a cover glass. Surplus Ringer is then removed by filter paper until the gonad ruptures and spermatocytes flow out. No strong pressure should be applied to the cover glass in order to prevent severe mechanical damage of the cell nuclei. Observations are made with a phase contrast microscope. It could be demonstrated that even closely related species such as D. melanogaster and D. simulans or D. hydei and D. neohydei can be easily distinguished by the cytological method described above. The figures demonstrate some species specific functional structures of the Y chromosome in the hydei subgroup and the usefulness of this "cytotaxonomical" method even for routine work.



References:

Hess, O. und G. F. Meyer: Portug. Acta Biol. A, VII:29(1963);
Meyer, G. F.: Chromosoma (Berl.) 14:207 (1963)

Legend for figures:

Functional structures of the Y chromosome in primary spermatocytes. A) D. bifurca, B) D. nigrohydei, C) D. neohydei, D) D. hydei

Di Pasquale, A. and L. Zambruni. University of Milan, Italy. Further data on the manifestation of the "brown spots" character of Drosophila melanogaster.

Experiments have been undertaken in order to investigate if the manifestation of the bsp character (caused in the female by copulation) depends on the spermatic fluid. bsp females have been operated inserting into the vagina paragonia homogenate, other inactive substances, or no fluid at all, but the needle only. These females never showed spots, but a similar phenotype consisting of brown masses spread throughout the body. Matings of bsp etherized females produced a significant decrease in the frequency of brown spots. Copulations interrupted before the beginning of ejaculation caused the appearance of brown spots. These observations emphasize the existence of a mechanical stimulus correlated with the copulatory act, independently of the spermatic fluid.

Kurokawa, H. Tokyo Metropolitan University. Variability in the genitalia of Drosophila auraria found in nature.

Extensive Drosophila surveys have been made during last summer in some localities on Hokkaido. Among the flies collected, a few females of D. auraria produced many F_2 offspring showing variable characteristics in male genitalia. Almost all of the F_2 males have many bristles, ranging from 11 to 16 in number, on their 6th sternites. Submedian process with a median notch of the novasternum is apparently different in shape from those of three races, A, B and C. The size of this part is considerably greater than in race A, but smaller than in race B. In many cases it has been found that the flies show asymmetry (or irregularity) in this shape. In some flies, an apical spine is found in either tip of the bifurcated process, and no spine is found in the others. The aedeagus is very similar in shape to that of race A. It is conjectured that the flies first collected in Hokkaido might be interracial hybrid.

Ohba, S. and Y. Kataoka. Tokyo Metropolitan University. The behavior of a recessive gene, cn, in artificial populations of D. virilis.

In four experimental populations of bottle type, the frequencies of a recessive eye color gene, cn, have now been followed for more than 80 weeks. The mutant used occured spontaneously in a wild D. virilis strain (Tokyo) in 1961 and was kept together with its wild allele in the same vial as a mixed stock. In experimental populations all live flies, both old and newly emerged, were etherized and classified for sex and eye color, then transferred to a new bottle twice a week. Two kinds of culture media, 20% and 10% yeast medium, were used for each pair of populations. At the beginning the gene frequency of cn and of its wild allele was equal, 50%. After 80 weeks, the cn frequency exhibits no remarkable decrease or increase. At a glance, it seems to be in equilibrium at the level of 50-60% (20% yeast medium) or 40% or so (10% yeast medium), though some fluctuations occur from time to time. Furthermore, precise examination indicates that regular rhythmic changes may occur in all populations, and that the cycle of these rhythmic changes coincides with generation intervals in each population. Another experiment in which populations have been kept in a plastic cage for more than 40 or 60 weeks gives similar results. Using 10% yeast medium, we can find a slow decrease in the cn frequency (less than 1% per generation) in the cages of high initial frequency (75%), but no significant change if initial frequency is low (10%). In populations with an intermediate initial frequency (50% or 25%) only doubtful changes were observed.

Okada, T. Tokyo Metropolitan University. Negative correlation between the development of hooked scaly bristles and of sex-combs.

As reported in DIS 38:38, the males of the species belonging to the suzukii, takahashii, and ficusphila species subgroup of the melanogaster species group and also the males of Drosophila (Tanigasterella) gracilis Duda, possess a special kind of hooked scaly bristles on the tibia and/or on the metatarsus of the middle leg. Applying R-technique of finding relationship between two characters over all species examined, it was estimated that the development of this special kind of bristles is negatively correlated with that of the sex-combs on male fore legs. The correlation coefficient calculated, classifying each character in three classes, is -0.64 ($\rho \neq 0$), for the species of the melanogaster group and D. (T.) gracilis. Simultaneous occurrence in a species of both characters, which are similar secondary sexual characters, seems to be a homeotic expression of sex-combs on the homonymial organs. Furthermore, it can be assumed that the high development of sex-combs on the fore legs prevents the expression of the homeotic effect upon middle legs to produce scaly bristles through the phenomenon of enantiomorphosis or compensation of body materials.

Tobari, I. National Institute of Radiological Sciences, Japan. Effects of temperature on the pre-adult viability of lethal heterozygotes in D. melanogaster.

With the aim of clarifying the mechanism through which deleterious genes are retained in natural populations, viabilities of heterozygotes for lethal genes were examined under different temperatures.

Three hundred fifty-five second chromosomes were extracted at random from a laboratory population which had been kept in a constant temperature room at 25°C for about one year before the experiment. Cy/Pm viability test showed that of these 355 chromosomes 15.5 percent were lethal, 8.7 percent semi-lethal, 9.9 percent subvital and 65.9 percent normal. The allelic rate of the lethal chromosomes was 8.8 percent and the number of nonhomologous lethal chromosomes was 24.

Pre-adult viabilities of lethal heterozygotes relative to normal heterozygotes were tested at three different temperatures (17°C, 25°C and 29°C) by using the 24 lethals obtained and 31 normal chromosomes taken at random. Every lethal chromosome was tested in combination with 10 different normal chromosomes. The testing scheme employed is as follows:

P	Cy/l_k♀♀	N_i/N_i	Cy/N_j♀♀	N_i/N_i
F_1	Cy/N_i	l_k/N_i	Cy/N_i	N_j/N_i
Frequency	$(1-q)$	q	$(1-q')$	q'

1: lethal chromosome N: normal chromosome
k = 1,2, ···· 24. j = 1,2, ···· 15. i = 1,2, ····10.

The relative viability (v) of a lethal heterozygote is estimated by $q(1-q')/q'(1-q)$ and the selection coefficient for viability by 1-v.

The mean selection coefficients obtained for 17°C, 25°C, and 29°C were 0.0245, 0.0253 and - 0.0148 respectively. These figures indicate that lethal heterozygotes are at a selective disadvantage at 17°C and 25°C, but they are selectively favoured at 29°C on the average.

From the result of analysis of variance for selection coefficient, it can be seen that the effect of temperature is significant at the 5% level. The effect of interaction between lethals and temperatures is also significant. These results show that the degree of dominance of lethal genes are highly dependent on the temperature at which flies are cultured.

Nagle, James J. North Carolina State. Studies on experimental sympatry between two sibling species.

The effects of experimental sympatry involving the sibling species D. arizonensis and D. mojavensis baja were studied. Five population cages were analyzed. Cages 1 through 4 were initiated with equal numbers of males and females of both species. Cage 5 was initiated with male and female interspecific F_1 hybrids. Cages 1, 2, and 5 were analyzed cytologically using salivary gland chromosomes, and Cages 3 and 4 were analyzed by use of mutant eye markers.

In Cages 1 through 4 both species coexisted for the duration of the experiment (approximately 11 generations). Only in Cage 1 did a trend toward replacement seem imminent. The dominant species was baja, which represented 90.8% of the population.

Interspecific F_1 hybrids and recombinant backcross types were produced in low frequencies in the mixed populations, even though a choice of mates was available. However, a hybrid swarm was not produced. This is attributed to a lack of luxuriance in the hybrids. Hence, a non-coadapted hybrid complex was in competition with the coadapted complexes of the parental species. This non-coadapted hybrid complex obviously had a lower adaptive value than either parental complex.

In Cage 5 a "hybrid" population flourished for more than 12 generations. It is hypothesized that heterosis exists in certain recombinant types which, once formed, constitute the initiators of a hybrid swarm such as that demonstrated by Mettler (1957) using different races of the same two species.

Sterility and semi-sterility were found to exist among the males of F_1 hybrids. This reduced reproductive capacity is no doubt a contributing factor to the low adaptive value of the hybrids when they are competing with the parental species.

It is further hypothesized that the heterosis which would lead to the production of a hybrid swarm is functional only in those recombinant types which do not also contain poorly adapted combinations in other parts of the genotype (such as factors affecting fertility). As a result, the probability of obtaining "good" male and female recombinant types in a population consisting predominantly of the parental species is very low. This is thought to be due to (1) the low frequency of interspecific hybrids (2-3% of the population), (2) the low adaptive value bestowed upon these types by their non-coadapted gene complex, and (3) the greatly reduced fertility of hybrid males.

Thus, a reproductive barrier exists between the parental species beyond the one dealing with the initial production of F_1 hybrids. If the second barrier (production of highly fit heterotic recombinants) is hurdled, the production of a hybrid swarm, or more correctly an introgressed population, would result.

Paik, Y. K., and J. S. Geum. University of Texas. Distribution of natural lethal genes on the second chromosome of D. melanogaster.

Twenty-nine lethal genes extracted from Korean natural populations were localized by use of three recessive marker genes. The distance between marker genes and lethal genes was adjusted by Kosambi formula. The results are as follows:

Collection	Non-allelic loci	Distribution		
		Left	Middle	Right
S62	17	4	11	2
K62	12	2	5	5

It can be seen that the lethal genes of S62 population are distributed in the central region (X^2 = 10.0, d.f. = 2, P = 0.01 - 0.001). However, the lethals of K62 population seem to be randomly distributed (X^2 = 1.5, d.f. = 2, P = 0.3 - 0.5).

Browning, L. S., and E. Altenburg. University of St. Thomas, Texas. A comparison of the sterilizing effect of X-rays, quinacrine mustard and azaserine on Drosophila males.

Males of Muller's Maxy stock were treated with X-rays, quinacrine mustard and azaserine and individually mated (in vials) to Maxy females (2 to 3 per male). The males were transferred to new food vials with fresh virgins every third day for several such broodings. The dose of X-rays was 3000r or 5000r and that of the quinacrine sufficient to give a 2 to 3% lethal rate in mature sperm. The azaserine was weakly mutagenic (about 1% lethal inducing). In the present experiments, the X-rays produced a drastic drop in fertility in the third brood (8-10 days after treatment) from which there was a large measure of recovery in the fourth brood. In the case of chemical treatments, there was no such definite brood pattern. The effect of the three agents on the fertility of the Maxy males is shown in the following table:

Agent	Brood (and days)	No. ♂♂	No. Fertile Cultures	Percent
X-rays	1 (1-4)	793	739	93
	2 (5-7)	748	633	85
	3 (8-10)	712	208	29
	4 (11-14)	635	470	74
Quinacrine	1	721	332	46
	2	507	201	40
	3	279	127	46
	4	191	109	57
Azaserine	1	432	241	55
	2	388	237	61
	3	252	202	80
	4	214	74	35

These results might indicate that the reduced fertility of the males after the chemical treatments is due to a toxic effect on their soma (reducing their life span and fitness to mate), rather than a sterilization of the germ cells at a sensitive stage (as in the case of X-rays), since the viability of the cultures undergoes considerable reduction from one brood to the next after the chemical treatments. With the X-rays, there is no such drastic drop in the viability.

Narise, Takashi. University of Chicago.
The migration of Drosophila ananassae under competitive conditions.*

It is probable that the mode of migration of flies in a heterogeneous population is quite different from the mode of migration in homogeneous population, even though the strains in the former population have their own migratory activity genetically as shown by Sakai et al. (1958) and Narise (1962). From this point of view, an experiment was conducted with four strains of D. ananassae in order to find out the mode of migration under competitive conditions.

Two of four strains were so called light ananassae which have yellow body color and collected in Pogo Pogo (L-pp), Tutuila, American Samoa, and Majuro (L-maj), Marshall Islands. The other two strains were so called dark ananassae having black body color, and collected in Pogo Pogo (D-pp) Tutuila, American Samoa, and Rarotonga (D-rar), Cook Islands.

Two kinds of experiments were conducted: 1) migratory activity of light and dark ananassae in a mixed population, and 2) the mode of migration in dark and light ananassae in a mixed population. In the experiments, four migration tubes were connected with each other radially in the former experiment, and eleven radially in the latter experiment. All experiments were conducted in $25^{\circ} \pm 1^{\circ}C$ in a dark room.

From the results in the first experiment, the following conclusions have been drawn: 1) the migratory activity in light ananassae was stimulated by dark ananassae, while the activity in dark ananassae decreased under the mixed condition; 2) in some combinations, for example D-rar and L-pp, the migratory activity of those strains depends on the relative frequency of both strains in the original tube into which the flies were introduced at the beginning of the experiment, but in other combinations the activity has no connection with the relative frequency in the original tube; and 3) the rate of increasing or decreasing of the activity due to mixing in a strain is quite different, depending on the combination of two strains. In this connection it is of interest to find that the migratory activity of D-rar strain is stimulated by mixing with L-pp, while it loses activity when coexisting with L-maj.

In the second experiment, it was found that light ananassae dominated in all tubes in the combinations D-pp and L-pp as well as D-pp and L-maj. However, light ananassae dominated in central tubes, but dark ananassae in the surrounding tubes in the combination D-rar and L-maj as well as between D-rar and L-pp, although the total number of migrant flies in light ananassae is greater than in dark ananassae.

From those two experiments, it is expected that light ananassae should be widely distributed in natural populations and dark ananassae should occupy marginal populations or isolated populations. It is also clear that the migratory activity of a strain is affected by other strains in the population and the mode of migration is determined by what kinds of strains coexist in a heterogeneous population.

Ullman, S. Institute of Animal Genetics. Edinburgh.
Epsilon and polar granules in Drosophila pole cells and oocytes.

An electron microscopic investigation of the pole cells of D. melanogaster, D. virilis and D. willistoni has revealed, beside the polar granules, the presence of other organelles, the epsilon granules. Polar granules are spherical and lack a bounding membrane. Each consists of a granular, electron opaque cortex surrounding a less dense core.

Epsilon granules are ovoid or dumb-bell shaped organelles, bounded by a delicate double membrane. Within a fibrous matrix ill-defined spaces occur. The functions and interrelationships of the polar and epsilon granules is still obscure.

Preliminary observations suggest that the epsilon granules arise late in oogenesis, from convolutions of the oocyte plasmalemma.

Gregg, T. G. and J. Day. Miami University. A further note on ovoviviparity in Drosophila.

It has been observed (King and Sang, DIS 32) that fertilization can, on rare occasion, take place in the ovariole in melanogaster females. It has also been observed (Gregg and Day, DIS 37) that there is considerable variation between strains in the extent to which adult, virgin melanogaster females retain and store mature oocytes.

King (DIS 38) has suggested that egg retention and ovariolar fertilization are two conditions that are necessary for the development of ovoviviparity. He further suggested that the interstrain variation in egg retention in melanogaster virgins gives some indication of the genotypes available in Drosophila for the establishment of high egg retention, and the development of ovoviviparity.

It may be pointed out that, indeed, not only are the genotypes available in Drosophila for the establishment of high egg retention, but that there is at least one species, D. hydei, where a type of egg retention has been established. Furthermore, this retention occurs not only in virgin females aged beyond the normal mating time, or in females under adverse conditions, but in mated, laying females as well. In melanogaster virgins egg retention results because the females abstain from laying while eggs continue to be produced. In hydei, a large species with a long life cycle, a large surplus of eggs is built up prior to the onset of laying while the female is maturing. After the onset of laying the rate of production of new eggs approximately equals the rate of oviposition. Thus, even in the ovaries of laying females there is a large number (100-150) of fully developed eggs. At the rate of oviposition generally encountered under optimum conditions in the laboratory (40-60 eggs/female/day) a newly produced mature oocyte will remain in the ovary, on the average, two to three days before it is laid. Under adverse conditions the retention time would undoubtedly be increased.

Thus far, ovariolar fertilization has not been observed in this species during the course of dissecting approximately 500 females.

The retention of eggs in hydei probably explains the capacity that these females have for laying prodigious numbers of eggs in short bursts, since retention would result in a reserve that could be drawn on in times of peak egg laying. It is easy to imagine that such a capacity would be selected for in a species where females routinely lay eggs in environments that are suboptimal for egg deposition, with occasional opportunities arising for the deposition of eggs in choice environments.

Most of the data on egg retention in hydei have been obtained from a mutant (w lt) lab stock, but a wild-type lab stock has also been checked. One hundred virgins (one day old) from the w lt stock were separated into two groups of fifty each, and individually placed in vials. The females in one group were mated (i.e. placed with males), and the females in the second group were not. Each females was transferred to a fresh vial every day, and the eggs laid on the previous day were counted. Also, each day several females in each group were sacrificed to determine the number of mature oocytes present in the ovaries, the number of ovarioles present, and whether the female had been inseminated. Females in the mated group were mated to males of approximately the same age, and insemination usually occurred on the seventh day after the eclosion of the female, but never sooner. There is some indication from other experiments that the use of older males can speed the process of insemination by a day or two.

An ovariole count of 148 ovaries of various ages showed an average of 21 ovarioles per ovary. There does not appear to be any increase or decrease in the number of ovarioles with age. The average number of mature oocytes present in a three day old female is about twelve. Mature oocytes continue to accumulate until the seventh day when each female contains an average of 125. The average number of eggs laid by twenty-nine females for an eight day period, from day seven to day fifteen, was 58 eggs/female/day. In seven day old females, even if they have been inseminated, most of the ovarioles contain three mature oocytes. However, some of the peripheral ones contain only two, while some of the central ones contain as many as six. The typical ovariole from an inseminated laying female from the ninth to the fifteenth day continues to contain two or three mature oocytes. The appearance of the ovarioles in the virgin females parallels very closely that in the mated females with the exception that since the virgins are laying very few eggs, there is no turnover of eggs in these ovaries

after the initial accumulation. After fifteen days, the number of mature oocytes in the ovaries becomes more variable. The fact that the females were kept in separate vials makes it possible to assert with certainty that females that were laying fertile eggs were also retaining eggs. There appeared to be an inverse correlation between the number of eggs laid in a day and the number of mature oocytes present in the ovaries at the end of the day, but the data were not sufficient to establish this point. It may also be pointed out that the type of egg retention exhibited by hydei is not necessarily characteristic of large flies with long life cycles. In D. virilis which is very similar to hydei in these two respects, the number of mature oocytes in the ovaries of a seven day old virgin is very small, averaging less than one per ovariole.

Average egg retention for several kinds of hydei females are shown in the accompanying table.

Egg Retention in hydei females

	7 day old virgins	18 day old virgins	18 day old mated
w lt	103/female(34♀♀)	87/female(62♀♀)	127/female(62♀♀)
wild Type	124/female(47♀♀)		

Edwards, J. W. and J. R. Simmons. Utah State University. Optic asymmetry and the absence of somatic crossing-over in D. melanogaster.

A developmental problem in such D. melanogaster mutants as eyeless and eyes-reduced is the occurrence of structural optic asymmetry in otherwise bilaterally symmetrical organisms. Intra-organismal nuclear differentiation by means of somatic crossing-over was discovered by Stern in 1936. He found that the frequencies of mosaicism in heterozygotes varied from 0.0 to 6.0 percent in the head-thorax and from 4.6 to 20.0 percent in the abdomen. The presence of minute factors increased the frequencies by 0.0 to 22.3 percent in the head-thorax and by 8.0 to 36.6 percent in the abdomen.

DeMarinis (Genetics 44:1101-1111) reported that, barring the less common events such as somatic mutations and somatic crossing-over, the same genetic constitutions occurred on corresponding sides of bilaterally symmetrical organisms. He concluded that, consequently, asymmetry must have resulted from differences in external and internal environmental factors. Somatic crossing-over is an established phenomenon, however, and genetic factors have been shown to increase its frequency. Also Baron (J. Exp. Zool. 70:461-490) had stated that sets of modifiers were present on all chromosomes, particularly the second and third in flies homozygous for ey^2. Thus, it seemed desirable to test critically for the possible occurrence of the "less common" phenomenon of somatic crossing-over. If this were occurring, structural optic asymmetry would result from nuclear differentiation by means of somatic crossing-over. Further, although all nuclei were homozygous for the eyeless genes, the recombination of genetic modifiers in the second and third chromosomes (and in the X-chromosomes of females) would yield asymmetrical phenotypes.

To test this possibility, the second and third chromosomes were marked with bw and st genes. Six kinds of experimental crosses (all progeny were homozygous for ey^4) and two kinds of control crosses (progeny were $ey^4/+$ or $+/+$) were made. The following genotypes resulted: (1) bw/+;st/st;ey^4/ey^4 (2) bw/bw;st/+;ey^4/ey^4 (3) bw/+;st/+;ey^4/ey^4 (4) bw/+;st/+;$ey^4/+$ (5) bw/+;st/+;+/+. Only white ommatidia were scored.

The experimental crosses produced 2593 flies. Six had mottled pigmentation (white ommatidia) in one eye. Of a total of 1396 flies produced from the control crosses, one female was observed to have white ommatidia in one eye. Thus, the observed frequencies of mosaicism in the experimental and control groups were 0.23 percent and 0.07 percent, respectively. No simple objective method for accurately measuring asymmetry has been devised; however, by using the frequency of flies with ommatidia only on one side of the head, a minimum estimate was possible. Of the 155 progeny so scored, 35 or 22.6 percent had asymmetrical eye structure. Consequently, the conclusion seems justified that somatic crossing-over probably does not play

a role in the production of structural optic asymmetry in eyeless[4] Drosophila. Furthermore, if somatic crossing were an active factor, asymmetry should not occur in an isogenic strain of eyeless flies. Spofford, however, (Genetics 41:938-959) found that the right-left correlation in an isogenic ey^4/ey^4 strain was negligible (coefficients of +0.08 for males and -0.22 for females). Thus, nuclear differentiation by means of somatic crossing-over is not responsible for optic asymmetry in eyeless[4] Drosophila melanogaster.

Smoler, M. University of Wisconsin. The ineffectiveness of anoxia in promoting non-disjunction.

Mottram (1930) found that there was an increase in the rate of non-disjunction when D. melanogaster females were treated with CO_2. The present experiments were an attempt to learn if anoxia was causing the increase in the rate of non-disjunction.

In these experiments yellow apricot virgin females, 12-36 hours old, were exposed to Linde highest purity nitrogen for 1 hour. The treated and control groups of females were mated to yellow; sc^8.Y males. Experiment I was limited to a control group to serve as a pilot experiment. In experiments I and II the females were mated singly in shell glass culture vials and in experiments III and IV fifteen pairs were mated in 1/4 pint milk bottles. In all experiments the flies were mated on cornmeal, molasses and agar food medium. The flies were transferred to fresh culture bottles every 2 days for a ten day period. Only transfers 1, 4 and 5 (days 0-2, 6-8 and 8-10) were examined. The expected classes of offspring from this mating are yellow females and apricot males, whereas the exceptional flies produced by maternal non-disjunction are apricot females and yellow males, and a double non-disjunctional event would be detected by the presence of yellow apricot females or wild-type males.

A total of 32,701 offspring from treated females were compared to 29,488 offspring from control flies, and the results indicate that there is no significant difference in the rate of non-disjunction in the 2 groups, and there was no difference between the tested 2 day broods. The ratio of nullo-X non-disjuncts to double-X is in agreement with that found by other workers.

EXPERIMENT	GROUP	NORMAL ♂		EXCEPTIONS y♂	apr	+♂	y/apr	TOTAL	PERCENTAGE
I	Control	5,234	5,297	2	1		1	4/10,531	.038
II	Control	1,467	1,445					0/2,912	--
	Treated	1,703	1,722	1		2	1	4/3,424	.117
III	Control	4,541	4,473	6			1	7/9,014	.078
	Treated	7,075	7,166	6			3	9/14,241	.063
IV	Control	3,429	3,602	2				2/7,031	.028
	Treated	7,627	7,408	4				4/15,035	.027
Total	Control	14,671	14,817	10	1		2	13/29,488	.044
	Treated	16,405	16,296	11		2	4	17/32,701	.052

Oshima, C. and T. K. Watanabe. National Institute of Genetics, Japan. Location of recessive lethal genes on the second chromosome of D. melanogaster.

A total of one hundred and five recessive lethal chromosomes of various origins (64 chromosomes isolated from Kofu-Katsunuma natural populations, 18 chromosomes isolated from Suyama-Juriki natural populations and 23 chromosomes obtained by Dr. Mukai as spontaneous lethals) was tested to determine the locus of individual lethal genes on the second chromosome by crossing with the balanced dominant marker Sp Bl L/Cy strain.

Five kinds of inversions (In A, B, K on the left arm; In C on the right arm; a pericentric inversion D) were detected in natural lethal chromosomes.

Seventy natural lethals and all spontaneous lethals were found to be single lethal genes on the second chromosome. The remaining 12 lethal chromosomes seemed to have double, multilocus and synthetic lethals.

If the lethal genes from natural populations are grouped according to their location on the chromosome by the three regions (left 0-40; middle 40-70; right 70-108), the distribution found was 18 in the left, 36 in the middle and 16 in the right region. This concentration of lethal genes in the central region is similar to that of recessive visible mutant genes on the genetic map of the second chromosome. On the other hand, spontaneous lethal genes seemed to be distributed randomly; 6 in the left, 7 in the middle and 10 in the right region. This distribution of spontaneous lethal genes may be similar to that reported for natural lethals by Paik (1960) and Seto (1963).

Table 1. Comparison of autosomal inversions and their locations, that have been found in natural populations of Japan and other countries, as well as in laboratory stocks.

Chromosome	Natural populations in Japan	Natural populations in U. S., Mexico and Hawaii (Warters)	Laboratory stocks (Bridges)
2 L	In(2L)A: 26A-33E In(2L)B: 22D-34A In(2L)K: 22D-26B	In(2L): 22D-34A	In(2L)t: 22D-34D
2 R	In(2R)C: 52A-56F	In(2R): 51D-56F	In(2R)NS: 52A-56F
2 LR	In(2LR)D: 36F-49B		
3 L	In(3L)E: 63A-74C In(3L)F: 66C-71B	In(3L)A: 63B-72E In(3L)B: 66C-70B	In(3L)P: 63C-72E
3 R	In(3R)G: 89D-96A In(3R)H: 92D-100F In(3R)I: 93D-98F In(3R)J: 96E-98F	In(3R)B: 89E-96A In(3R)A: 94C-98F In(3R)C: 86B-92F	In(3R)P: 89C-96A In(3R)C: 92E-100F In(3R)Mo: 93E-98F

Table 2. Types of inversions and their frequencies in Japanese natural populations.

Population	Suyama	Katsunuma	Kofu
Inversion & standard		Frequency (%)	
2L (standard)	84.5	68.0	67.0
In(2L) A	0.5	0.0	0.5
In(2L) B	15.0	32.0	32.5
2R (standard)	100.0	79.0	73.0
In(2R) C	0.0	21.0	27.0
3L (standard)	97.5	89.5	92.0
In(3L) E	2.5	8.5	6.5
In(3L) F	0.0	2.0	1.5
3R (standard)	82.0	61.0	63.5
In(3R) G	2.5	18.0	11.0
In(3R) H	15.5	10.0	14.0
In(3R) I	0.0	11.0	11.0
In(3R) J	0.0	0.0	0.5

Halfer, C. University of Milan, Italy. Different degree of compatibility in several stocks of Drosophila melanogaster, tested with ovary grafting.

A different degree of comptatibility between genotypically different stocks of D. melanogaster, tested using the method of ovary grafting, has been already observed in the first series of experiments (DIS 37).

In a later investigation, I took into consideration, as recipients, not only the two wild tumorous stocks: tuA_2 and tuB_3, but also the wild stock Varese, which is completely tumourless and the almost tumourless mutants vermilion and yellow white. In this way I obtained, besides the combinations between the different genotypes, as donors and as recipients, also combinations between donor and recipient of the same genotype. Only the larval survival after grafting and the percentage of graft attaching has been considered.

tuA_2 (28-45%) and vermilion (39-82%) yielded consistently high rates of survival, irrespective of the genotype acting as donor, while the other three stocks: tuB_3 (1-12%), Varese (2.7-9%) and yellow white (0-2.8%) showed a very low survival in almost all cases. From these considerations it is quite clear that this is a case of host specificity, where the host genotype only is important in these grafting experiments, being the genotype of the donor of no influence. The graft attaching was always good, even in the cases of genotype showing a poor compatibility with the graft. Grafting experiments were carried out also with heterozygotes obtained from reciprocal crosses between the stock vermilion (with a high rate of survival) and the three stocks: tuB_3, Varese and yellow white (with a low survival). They showed always a rather good survival rate (about 40%).

The different behaviour of homozygotes and heterozygotes supports the view that the host specificity is genetically controlled.

Peterson, G. V. and E. J. Gardner. Utah State University. Melanotic tumor associated with failure to pupate in the tumorous head stock of D. melanogaster.

Over a period of years it was observed that large abdominal melanotic tumors appeared in mature larvae of one tumorous head stock of D. melanogaster. These tumors were rarely, if ever, seen in the adults. The melanotic tumors first became visible 102-125 hours after hatching. Normal time of puparium formation was found to be approximately 102 hours. Some larvae from the strain carrying melanotic tumors and the lethal factor lived 200 hours or longer but never pupated. About half of all larvae that failed to pupate did not develop visible melanotic tumors.

The lethal effect was observed in about 33 percent of the larvae when the abnormal condition was first observed. It decreased at each generation with the proportion of tumorous larvae remaining between 35 percent and 65 percent of the total number of lethals.

Crosses with a stock bearing inversions on the second and third chromosome have indicated that both chromosomes from the tumorous head stock are associated with lethal or low viability effects. The third chromosome is mainly responsible for the failure of pupation, however. No indication of sex linkage has been found.

Crosses with other tumorous-head stocks and wild stocks indicated that melanotic tumor development was not influenced by either of the major genes (tu-1 on the first chromosome and tu-3 on the third chromosome) associated with tumorous head. However, crosses between parents, both of which expressed the tumorous head phenotype and crosses in which neither parent expressed the phenotype did show a significant difference. When they both expressed the tumorous head phenotype, the progeny showed 12.5 percent lethal and 6.5 percent with melanotic tumors compared to 6.0 percent lethal and 1.9 percent with melanotic tumors for the other parental

group. This may indicate that a modifier gene increased the penetrance of the tumorous head trait as well as influencing or being responsible for the lethal effect. Because only a certain proportion of the lethal larvae developed melanotic tumors, it seems probable that the tumor formation is a secondary characteristic resulting from a ring gland defect.

Sokoloff, A. University of California at Berkeley. A possible maternal effect on quantitative characters in D. pseudoobscura.

In a previous report (DIS 33:162-165) comparison was made between measurements of length and width of wing and length of tibia derived from flies whose progenitors had been reared deliberately under different conditions. The statistical tool was the t-test, taking one character at a time in flies reared, say at 24°C, but whose mothers had been reared at 16° and 24°C and vice versa. It was reported that this test failed to show any maternal effect on the three characters mentioned. Recently, in analyzing some data demonstrating geographic variation in D. pseudoobscura, it became evident that differences between populations derived from different localities (some as close as six miles) cannot be demonstrated if one takes one character at a time but, if one takes two characters, significant differences are easily found when Fisher's Discriminant Function method is applied. In the light of this finding the problem of maternal influence on quantitative characters has been re-examined, applying this, more powerful, statistical method. The technical details can be found in the above reference. A summary of the history of the flies measured is given at the bottom of Table 1, and the results of the statistical analysis can be seen in Table 2. Although not all the possibilities were tested, the conclusions from this analysis are as follows:

(1) Flies reared in bottles differ significantly from those reared in vials at both temperatures (compare A-B and F-G variance ratios), even though attempts were made to avoid crowding.

(2) As expected, a period of starvation in the larval stages results in flies which are smaller than flies well fed for the whole larval stage (comparisons E-F). In turn, their progeny differ, even though they were reared under identical, non-crowded conditions (comparisons C-D).

(3) The temperature prevalent during the development of the progenitors appears to influence the body characters of the next generation when the latter flies are reared at the higher temperature (compare F-G), the bigger mothers producing generally bigger offspring but the reverse situation (B-D) appears to have no effect.

These experiments omitted some combinations, owing to time limitations. However, the results suggest that, in studies involving quantitative characters, the history of the preceding generation of D. pseudoobscura, particularly if the flies are reared at 24°C, must be known and specifically stated.

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Table 1. Means, variances and covariances of wing length (x) and tibia length (y) from D. pseudoobscura derived from Grand Canyon, Arizona. Upper block: flies reared at 16°C.; lower block: flies reared at 24°C. (for each sample N=50).

Sex		$\bar{m}_x$	s^2x	$\bar{m}y$	s^2y	cov_{xy}
	A	65.924	0.7998	23.612	0.2096	1,588.60
Females	B	63.692	1.181	23.100	0.2502	1,501.60
	C	63.308	1.080	22.896	0.1604	1,479.34
	D	63.372	1.022	23.052	0.1351	1,490.85
	A	59.184	0.9108	22.312	0.2010	1,347.54
Males	B	57.324	4.2365	21.808	0.7608	1,277.19
	C	57.208	0.7935	21.872	0.1824	1,276.99
	D	57.680	0.5878	21.988	0.1067	1,294.23

	E	56.984	0.9789	21.660	0.2322	1,259.71
Females	F	57.352	0.9012	21.484	0.1569	1,257.44
	G	57.832	1.398	21.788	0.1280	1,285.94
	H	58.036	0.8633	22.164	0.1629	1,312.74
	E	51.640	1.322	20.560	0.1845	1,083.61
Males	F	51.972	1.089	20.244	0.1849	1,073.74
	G	52.260	0.7465	20.672	0.1253	1,102.48
	H	52.400	1.037	20.764	0.2118	1,110.53

A = F_1 of single females reared in bottles at 16°C.
B = F_2 from A reared in vials at 16°C.
C = F_2 from E reared in vials at 16°C.
D = F_2 from F reared in vials at 16°C.
E = mass F_1 reared at 24°C.
F = F_1 of single females reared in bottles at 24°C.
G = F_2 from F reared in vials at 24°C.
H = F_2 from A reared in vials at 24°C.

Table 2. Results of the analysis of the data.

		A	B	C	D
			66.88	138.36	131.08
Females at 16°C	A	---	51.17	74.39	72.41
				0.126	.0235
	B	62.72	---	2.243	.969
					.107
	C	90.23	2.72	---	2.069
	D	87.80	1.17	2.51	---

		A	B	C	D
			4.96	56.40	26.67
	A	---	14.08	47.50	32.66
Males				.0136	.0146
at 16°C	B	17.07	---	.7378	.7652
					.2765
	C	57.59	.895	---	3.3257
	D	39.62	.928	4.03	---

		E	F	G	H
			.825	.982	7.281
	E	---	5.746	6.267	17.066
				1.159	21.80
Females at 24°C	F	6.97	---	6.809	29.53
					2.831
	G	7.60	8.25	---	10.642
	H	20.69	35.81	12.91	---

		E	F	G	H
			2.34	.361	.652
	E	---	9.66	3.80	5.106
				3.71	5.01
Males at 24°C	F	11.72	---	12.18	14.16
					.0069
	G	4.61	14.77	---	.5253
	H	6.19	17.16	.6369	---

Note: Upper and lower entries in right triangle cells are sums of squares for $\frac{n^2}{2}D$ between and for D within, respectively $X10^3$. The cells in the left triangle show the variance ratios. (For $n_1 = 50$ and $n_2 = 50$ a variance ratio of 1.94 is significant at the 0.01 level, and a ratio of 1.60 is significant at the 0.05 level).

Glassman, E. and E. C. Keller, Jr. University of North Carolina. The maternal effect of ma-1^+: The effect of hypoxanthine; the effect of lxd.

The maroon-like (ma-1) eye color mutant lacks detectable amounts of xanthine dehydrogenase, pyridoxal oxidase (pyridoxal- pyridoxic acid), and the ma-1^+ complementation factor. When ma-1 flies are derived from female parents that carry an ma-1^+ gene, their eye colors are wild-type and xanthine dehydrogenase activity can be detected, especially in early development (Glassman and Mitchell, Genetics 44:547, 1959; Glassman and McLean, Proc. Nat. Acad. Sci. 48: 1712, 1962).

The maternally-affected eye color is observed only in adults that emerge in the early days of hatching. Those that emerge in older bottles have the usual mutant ma-1 eye color. This loss of the maternal effect is not due to the age of the mothers, and must be ascribed to changes in the food (Glassman and Mitchell, loc. cit.). Either a substance is being used up or an inhibitor is accumulating. We believe that the latter explanation is true, and that the purines which accumulate in the food are inhibiting the low amounts of xanthine dehydrogenase in the maternally-affected ma-1 flies so that little pterdine eye pigment is synthesized.

To test this we crossed y f:=; st females to m ma-1; st males and allowed eggs to be laid on three different media: a) the usual Drosophila medium (devised by E. Lewis), b) the usual medium containing 0.1% hypoxanthine, and c) the usual medium containing 0.1% uric acid.

In these experiments the maternal effect did not diminish on the control medium until 10 and 12 days (replicate experiments) had elapsed. On the other media, the maternal effect diminished by 2 and 4 days (replicate hypoxanthine experiments) and by 7 and 9 days (replicate uric acid experiments). Thus, hypoxanthine (and perhaps uric acid) accelerates the disappearance of the maternal effect. Whether these compounds accumulate to a sufficient extent in the food to account for the loss regularly observed, and whether the mechanism is through inhibition of the small amounts of xanthine dehydrogenase remains to be shown.

The mutant, lxd, lacks the ma-1^+ complementation activity as well as the pyridoxal oxidase activity. Since these are also lacking in ma-1, we tested the effect of lxd on the maternal effect as follows:

A. The cross y f:=; ru lxd by females x v f Bx^3 ma-1 males produced v f Bx^3 ma-1; ru lxd by/+ males all of whom (94 in the first three days) were maternally affected. Thus, homozygous lxd in the mother has no influence on the maternal effect in the progeny.

B. The cross, v f Bx^3 ma-1; ru lxd by/TSS x ru lxd by/D, produces three types of males which were classified and counted during the first three days of emergence. These were:

genotype	phenotype (number maternally affected)
v f Bx^3 ma-1; ru lxd by	Only 1 out of 41
v f Bx^3 ma-1; ru lxd by/TSS (or D)	56 out of 56
v f Bx^3 ma-1; TSS/D	22 out of 22

Thus, homozygous lxd in the progeny completely abolishes the maternal effect.

We interpret these results as follows: The maternal substance responsible for the maternal effect is the product only of the ma-1^+ locus. The lxd locus is not involved and thus lxd mothers can have progeny that are maternally affected. This is also true for the ry locus (Glassman and Mitchell, loc. cit.). However, for the maternal effect to be expressed in the progeny, enough product of the lxd^+ locus must be available for production of sufficient xanthine dehydrogenase for eye color synthesis to proceed normally. If lxd is in the progeny, then the maternal effect is abolished.

This interpretation is in accord with the idea that the lxd^+, ry^+, and ma-1^+ loci code for polypeptides (L, R, and M, respectively) that polymerize with each other. In this scheme, the maternal substance is, or is composed of, only the M subunit; the ma-1^+ complementation factor and the pyridoxal oxidase (these may be identical) are composed of M and L subunits;

while xanthine dehydrogenase is composed of M, L, and R subunits. The proof of this theory must await purification of these substances, although finding electrophoretic variants of xanthine dehydrogenase associated with each locus would indicate that all three are structural genes for this enzyme. We are currently analyzing three electrophoretic variants which we have found.

The idea that the ma-1^+ maternal substance is a stable template would also explain the effect of lxd on the maternal effect. This possibility is also being tested by analyzing the amount of the ma-1^+ complementation factor during the development of maternally affected flies.

Mittler, S. Northern Illinois University. AET and radiation induced crossing-over in male D. melanogaster.

A re-examination of the problem (Mittler and Hampel DIS 38) of whether AET has any effect upon radiation induced crossing-over in male D. melanogaster indicates that AET does afford significant protection to 9-12 day brood after irradiation. Adult males 2-16 hr. old heterozygous for ru h th st cu sr e^s ca were injected with 1 X 10^{-7}1. of 30 mg. AET/10ml buffered to pH of 7 and irradiated with 2000r and then back crossed to homozygous "rucuca" females at ratio of 1 male to 3 females. The males were transferred to new group of females every 3 days.

Number Males	9-12 day brood			12-15 day brood		
	Crossover	Non	% Crossover	Crossover	Non	% Crossover
120 Treated	57	8,502	.666	54	11,361	.473
101 Control	49	5,025	.966	29	4,187	.688

Glassman, E. University of North Carolina. Interaction of ma-l and lxd in the synthesis of pyridoxal oxidase and xanthine dehydrogenase.

The maroon-like (ma-l) and rosy (ry) eye color mutants lack detectable amounts of xanthine dehydrogenase. A strain which is mutant at a third locus (lxd) has only 20 to 25% normal amounts of this enzyme. Analysis of lxd flies reveals that the level of CRM (the suspected product of the ry$^+$ locus) is the same in lxd as in wild type, while pyridoxal oxidase (the suspected product of the ma-1^+ locus) is absent in lxd. Complementation tests in vitro are in agreement with these findings; thus there is no ma-1^+ complementation factor in extracts of lxd ry flies while the ry$^+$ complementation factor seems to be present in normal amounts in ma-l; lxd.

It seems evident that lxd is deficient in those molecules which are also specifically deficient in ma-l (such as pyridoxal oxidase and the ma-1^+ complementation factor) while the molecules missing in ry (such as CRM) seem to be present in normal amounts in lxd. Thus, the normal production of the ma-1^+ complementation factor and pyridoxal oxidase must be due to the interactions of the lxd$^+$ and the ma-1^+ loci. The paradox of the presence of substantial, though less than normal, amounts of xanthine dehydrogenase in lxd in spite of the absence of the product of the ma-1^+ locus can be resolved by various hypotheses. One can postulate that the ry$^+$, the ma-1^+, and the lxd$^+$ loci code for three different polypeptide chains, designated R, M, and L, respectively. These polypeptides produce different enzyme activities by polymerizing with each other in different ways. Thus, a polymer of the M and L polypeptides will have pyridoxal oxidase activity, while any polymer containing R polypeptides will have CRM activity. Only a polymer containing R, M, and L polypeptides will have xanthine dehydrogenase activity. On this basis, the in vitro complementation reaction between ma-l and ry extracts would be visualized as an interaction between two large polymers containing ML and R subunits, respectively. The deficiencies observed in lxd flies would be due to a defective L polypeptide which can still react in vivo to yield some xanthine dehydrogenase activity, but which is too defective to yield active ML polymers (pyridoxal oxidase). It is of interest that pyridoxal oxidase has a molecular weight almost as great as that of xanthine dehydrogenase (about 250,000).

Alternatively, one can view the lxd gene as a regulator of the activity of either the $ma\text{-}l^+$ gene or its product. In this case, the pyridoxal oxidase would be a polymer composed of only M polypeptides, CRM would again be any polymer containing R polypeptides, while xanthine dehydrogenase activity would be a polymer of M and R polypeptides. Another possibility is that only the ry^+ locus codes for a subunit of xanthine dehydrogenase, while the products of the $ma\text{-}l^+$ and lxd^+ loci are regulators or activators, not only of the R polypeptide which forms xanthine dehydrogenase, but also of other polypeptides which form pyridoxal oxidase and other unknown enzymes.

Muller, H. J. Indiana University. An improved stock, "vix," for scoring visible mutations that arise at specific loci in the X of the female.

The fertile females of this stock have X-chromosomes of the same composition as those of the "Maxy-v" stock (number f30 of the 1957 Bloomington stock list in DIS 31), except that in this stock the recessive genes ptg and oc and the dominant B are in the X-chromosome having most of the recessive mutants, while the balancing X-chromosome is supplied with lz^s instead of ptg oc. Moreover, the fertile females contain a Y-chromosome, of normal type. The X-chromosome of the fertile males has the composition y oc ptg lz f. As in the stocks "jynd" and "plond" (f72 and f88, respectively, of DIS 31), but even more so because of the additional pressure of the B^{M1} inversion in one X, the combination of heterozygous inversions in the presence of a free Y causes a high frequency of non-disjunction of the X's, with resultant matroclinous daughters, bearing both the mother's X's, that can be scored for visible mutations arising in the originally wild-type loci of the non-multiple-recessive X, while at the same time the only fertile sons (in this case, in fact, the only viable sons) are patroclinous. One kind of disjunctionally produced daughter, of phenotype y oc ptg B f, is sterilized by oc, while the other kind, of phenotype lz B^+, is rendered highly infertile by the lz/lz^s combination and, if it should breed, would produce virtually no crossovers of a kind that would disturb the genetic system.

The reproducing individuals are as follows:

males: Y^+/y oc ptg lz f

females: $\dfrac{Y^+/1J1\ sc^{J1(+)}\ In49\ v\ lz^s\ B^{M1},In}{y\ sc^{S1}\ car\ odsy\ B\ f\ g^2\ dy\ v\ ras^2\ ptg\ oc\ sn^3\ ct^6\ cm\ rb\ ec\ w\ pn\ 1\ sc^8}$

Any mutant or suspected mutant daughter arising non-disjunctionally can be bred with her brothers for verification and for establishing a balanced stock. The phenotypes of the disjunctionally arising daughters present combinations of peculiarities that keep them from being mistaken, even at first sight, from mutant daughters of non-disjunctional origin.

The "vix" stock not only has the advantage over "jynd" and "plond" of giving no disjunctional males. Its normal Y gives rise to more non-disjunction of the X's (more than half of the daughters being non-disjunctional) than does the ring-Y (Y^{LC}) of "jynd". Moreover, the Y^+, unlike the $y^-.Y^L$ of "plond", leaves uncovered the deficiency of the left end of the X that arises when, occasionally, the X's undergo acrocentric attachment with one another, or when one of them exchanges with the Y (see the Valencias and Muller in DIS 23:99-103); it thereby results in the death of these exceptions which when viable are difficult to recognize for what they really are. It is also to be noted that the homozygosity for vermilion of the parental and non-disjunctionally produced females, which was recommended by Altenburg, makes the other eye color mutants here in question more conspicuous. That is, it sensitizes the observer, as it were, to these changes, and although the locus of vermilion is thereby sacrificed so far as the finding of mutations to v is concerned, more is gained than lost by the greater ease and reliability of the scoring for these other loci.

The loci in the non-multiple-recessive X-chromosome of the "vix" stock in which it is practicable to recognize mutants and test them genetically fall into the following phenotypic categories: eye color, 7 (car, g, ras, cm, rb, w, pn); eye form, 3 (sy, B, ec, although B makes sy and ec more difficult); wing conformation, 3 (od, dy, ct); body color, 1 (y, although ptg might perhaps be used also); in addition an extreme "minus" mutation in sc^8 could be recognized, and dominants such as N, Bx, Sc, Minutes, Tu and Tul (see my "New Mutants" report in this issue of DIS) would also be detectable. For comparison with visible mutations arising at specific loci in the X of the male, the above mentioned "Maxy-vermilion" stock provides a close counterpart to "vix".

In some of the work on specific-locus mutations it is desirable to know how many lethals are arising at the same time, so that the results may be calibrated to a given standard of spontaneous or induced mutation frequency, as was done in earlier work by the Valencias and myself and by Schalet. The "vix" stock lends itself to such tests by merely crossing the females (whose mothers should have been derived by random sampling from the same cultures as supplied the flies for the specific locus tests) to males that have a Y of the type $1J1^+$.Y or sc^8.Y, in order to "cover" the 1J1 of the X-chromosome to be tested and thus allow another lethal to be recognizable by the absence of sons having the X under investigation. Of course the mothers of the females thus tested should have been bred individually, so as to prevent pre-existing lethals from being confused with the newly arising ones that are to be scored.

Abrahamson, Seymour and Helen U. Meyer. University of Wisconsin. Use of Minute to facilitate studies of mutations in second chromosomes.

The most laborious step when breeding for autosomal mutations is the collection of virgin females in the F_2 generation to cross with brothers of the same, balanced genotype. A new and rather simple method facilitates this step by having the unwanted type of F_2 heterozygous for Minute while the class needed for inbreeding is non-Minute. Due to the fact that the Minute flies have a considerably longer period of development than the desired type of non-Minute, Curly flies, only the latter are present in the F_2 cultures during the first 2-3 days of hatching. By taking advantage of this period one eliminates the need for getting Curly virgin females. Mrs. Gloria Daniels of our laboratory has recently demonstrated that raising the F_2 cultures at 16-19°C further extends the difference in developmental time between the Minute and the non-Minute flies. The stock used for this purpose ("M") has the composition S M(2)S7 bw^D/ dp^{txI} Cy,Ins0 pr cn^2 sp and is of good viability. It is not necessary to have special second chromosomes in the P_1 generation; wild type or marked chromosomes may be used.

The simplest procedure is to cross the P_1 generation to the M stock, select Curly F_1 males and then cross these again, individually, to 2-3 virgin females from the M stock. Parents should be removed. In F_2 one should use offspring from the first 2-3 days of hatching. These are usually all of the desired, Curly type, although occasionally a Minute, brown fly may already be present. However, their offspring can readily be identified by these markers when scoring for lethals in F_3.

We prefer to use a somewhat more elaborate scheme and combine this method with H. J. Muller's scheme of "criss-crossed" second chromosome lethals, S, Cy, dp^{tx}, and Sp. (Sp is now being incorporated into the "M" stock). The P_1 generation is first crossed to a stock dp^{tx} Sp pr cn bw sp/S^2 ls Cy,InL pr cn bw sp. This has the advantage that both types of F_1 males, Curlies and non-Curlies, may be utilized. Then one proceeds as described above. A few, late hatching homozygous Curly survivors will appear along with the Minute, brown flies in F_2. These can be distinguished from the regular Curlies by also being homozygous for pr and sp.

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Geer, B. W. Knox College. A new synthetic medium for Drosophila.

Sang's (1956) Medium C or modifications of Medium C have been used extensively in nutritional and genetic studies of Drosophila with excellent results. However, it is sometimes necessary or desirable to replace casein with an amino acid mixture. Many amino-acid mixtures have been tested by this laboratory to determine their adequacy for the diet of D. melanogaster and the following medium was found to be the best.

Amino Acids	mg		mg
L-Arginine·HCl	80	RNA	100
L-Cystine	30	Cholesterol	30
L-Glutamic acid	840	Thiamine·HCl	0.2
Glycine	40	Nicotinic acid	1.2
L-Histidine·HCl	100	Riboflavin-5'-phosphate, Na	1.0
DL-Isoleucine	300	Calcium pantothenate	1.6
L-Leucine	200	Pyridoxine·HCl	0.25
L-Lysine·HCl	190	Biotin	0.03
DL-Methionine	80	Folic Acid	1.0
DL-Phenylalanine	130	Choline chloride	8.0
DL-Threonine	200	$FeSO_4$	1.0
L-Tryptophan	50	$CaCl_2$	1.29
L-Tyrosine	80	$MgSO_4 \cdot 7\ H_2O$	24.6
DL-Valine	280	$MnSO_4 \cdot H_2O$	1.29
Other Components		$NaHCO_3$	100
		KH_2PO_4	183
Agar	1500	Na_2HPO_4	189
Sucrose	1000	Water to	100 ml

Flies have been cultured on this medium by serial transfer under germ-free conditions for several generations.

Preparation of the medium is as follows: Dissolve the RNA in an amount of phosphate buffer solution equivalent to 0.4 of the final volume. Mix the buffer-RNA solution and all of the amino acids except cystine and dissolve the amino acids by gentle heating. After neutralization with dilute NaOH, add the B-vitamins, salts, and choline. Add cholesterol as a suspension prepared by dissolving cholesterol in warm 95% ethanol, adding water, and removing the ethanol by autoclaving for ten minutes. Dissolve the cystine in a minimal amount of 1 N HCl and add. After a final neutralization, add sucrose and agar. Adjust the final volume to 100 ml by adding water. Sterilize the medium by autoclaving at 15 pounds of pressure for 15 minutes.

The reports of Hinton, Noyes, and Ellis (1951), Sang (1956), and Salmon (1964) were especially useful during the development of this medium

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Mazar-Barnett, Beatriz K. de. Comisión Nacional de Energía Atomica, Argentina. A dark medium appropriate for egg counts.

When a dark colored background is needed for counting eggs, caramelized dextrose has proved to be very convenient. It is non-toxic, and one spoonful per Kg of standard cornmeal-molasses-agar medium is enough to obtain a dark even color.

Blaylock, B. Gordon.[1] Oak Ridge National Laboratory, Tenn. A method for preparing salivary-gland chromosomes.[2]

A modification of a method used by Keyl and Keyl (Arc. F. Hydrobiol 56:43-57, 1959) for Chironomus salivary chromosomes produces good results with Drosophila. There are several advantages in this method over most standard techniques. The staining time is short; only two minutes are required. Because there is good resolution of the fine bands on a clear background and the chromosomes spread easily, this is a good method for studying specific regions of the chromosomes.

The details of this method follow:

1. Dissect large well-fed larvae in a drop of Shen's solution (9 g of NaCl, 0.42 g of KCl, 0.25 g of $CaCl_2$, 1 l of H_2O) on a siliconed slide.
2. Immediately transfer the glands to a drop of 3 parts 95% ethyl alcohol and 1 part 45% acetic acid for 30 seconds. (A stop watch is used for timing).
3. Transfer the glands to a small drop of acetic carmine and acetic orcein on a siliconed cover glass for two minutes. The staining solution is 1 part of 1% carmine boiled in 45% acetic acid mixed with 4 parts of 1% natural orcein dissolved in 50% acetic acid.
4. Absorb the excess stain from the glands with a small strip of bibulous paper making sure that the paper does not come in contact with the glands by using a dissecting needle to hold the glands in place.
5. Immediately place a small drop of 72% lactic acid over the glands.
6. Invert the cover glass on a clean slide and hold it in place while tapping the cover glass several times directly over the glands with the eraser end of a pencil.
7. Fold a sheet of bibulous paper around the slide and press well with the thumb in a rolling motion being careful not to let the cover glass slip. A little experimenting will help determine the amount of pressure to use to obtain the desired amount of spreading.

The slide can be studied immediately using phase contrast. The preparations can be maintained for several weeks without sealing; however, the slides can be made permanent by the dry ice technique (Baker, W. K. DIS 26:129, 1959).

[1] Radiation Ecology Section, Health Physics Division Oak Ridge National Laboratory, Oak Ridge, Tennessee.

[2] Research sponsored by the U. S. Atomic Energy Commission under contract with the Union Carbide Corporation.

Doane, W. W. Yale University. Use of disc electrophoresis for analysis of amylase isozymes.

A modification of the acrylamide gel disc electrophoresis method of Ornstein and Davis (1961) has been developed in order to analyze amylase isozymes in various Amy strains of D. melanogaster.

The small pore gel, in which separation occurs, consists of equal parts of "Small-Pore Solutions #1 and #2" but altered to include 0.2% Fisher soluble starch (or Connaught hydrolyzed starch). This is done by heating a 0.4% starch solution in a water bath brought to a boil and, when the solution becomes clear, warming it an additional 3 minutes before cooling. The small-pore solution #2 is then prepared by dissolving 0.14 g. ammonium persulfate in 100 ml. of this starch solution so that the final starch concentration of the gel is 0.2%. This concentration proved best for quantitative estimates of enzyme activity and also for photographic purposes.

Following electrophoresis, gels are removed from their tubes and incubated at 37°C. for given time intervals (from 0 to 45 min.) in either M/20 Tris buffer, pH 7.2, or M/15 phosphate buffer, pH 6.8. With the latter buffer, Cl^- ions should be added in order to enhance amylase activity; p-chloromercuribenzoic acid should also be included in order to inhibit glycogen phosphorylase activity. Immediately after incubation, gels are stained with iodine reagent (Smith & Roe, 1949) diluted to half-strength. This not only stops hydrolysis of starch inside the gel but also stains those regions where enzyme activity does not occur. After 5-10 minutes, gels are rinsed and transferred to 7% acetic acid. Amylase isozymes appear as clear bands in the dark blue gels. These should be photographed immediately as artifacts begin to develop within 15 to 20 minutes.

Wheeler, M. R. and F. Clayton. University of Texas; University of Arkansas. A new Drosophila culture technique.

Since attempts to raise endemic Hawaiian spp. on standard banana or cornmeal media were failures, a new medium was devised, using several dried breakfast cereals of the high-protein, high vitamin and mineral types. Other D. spp. do extremely well on it. The basic recipe is as follows:

1000 ml distilled water
13.5 to 14.5 g Bacto agar, fine (amount varies with climate)
50.0 g dried Brewers yeast
1 jar (4.75 oz) Gerber's Strained Banana Baby food
10.0 g Kellogg's Special K cereal
5.0 g Kellogg's Concentrate cereal
15.0 g Gerber's High-Protein cereal
15.0 g Kretschmer's Wheat Germ

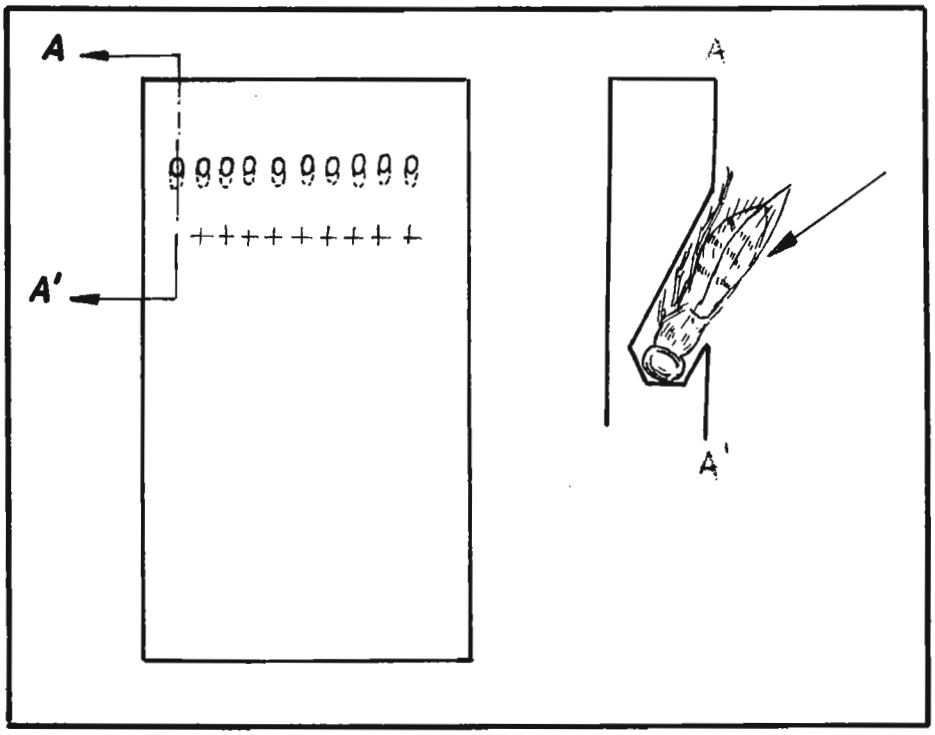
In addition 5 ml of ETOH (95%) was used to wet the dry yeast, and 5 ml of propionic acid was used for mold inhibition.

To secure good pupation and eclosion, mature larvae were transferred to vials half-filled with lightly moistened sand; larvae burrow down (some spp. to 4.0 inches) to pupate. At eclosion adults climb upward through the sand using the ptilinum to force a trail. This technique works best for those spp. whose larvae crawl up into the cotton plug when mature.

Work supported by NSF Grant GB 711.

Mazar-Barnett, Beatriz K. de. Comisión Nacional de Energía Atomica, Argentina. A fly holder for injecting Drosophila.

When injecting Drosophila with mutagenic agents, it is desirable to do it in as short a period of time as possible, especially when the compound injected is chemically unstable. A lucite plate with 20 small cavities, each of a size to accomodate one fly, has proved to be quite useful. The flies are kept in a position suitable for injection, as shown in the drawing, and when ready one or two taps of the plate will remove them easily. It is possible in this way to inject about 150 flies in one hour.



Malich, C. W. NASA Ames Research Center, Moffett Field, Calif. Thin window holder for particle irradiation of Drosophila.

The finite range of energetic charged particles and their variation in linear energy transfer with penetration require special techniques for the treatment of Drosophila. A simple holder has been devised which makes irradiation convenient and uniform for particles having ranges between a few millimeters and a few centimeters. A circular well 1" diam x 0.5 mm deep (for flies averaging 1.0 mm thick) is machined into a lucite disk 1 3/4" diam x 1/2" thick. Eight ventilation grooves 0.015" x 0.015" are milled from this depression to the outside. A cover is made by cementing a thin window over a circular hole 1 1/4" diam in a lucite disk 1 3/4" diam x 1/4" thick. We use Mylar of thickness 0.001" for the window because it is quite strong, handles well and will give a flat surface readily. The window is attached by brushing a small amount of acrylic cement onto the lucite and gently dropping a square of selected Mylar in place. This is trimmed after drying under pressure. John E. Neff of the NASA Ames Research Center suggests stretching 1/2 mil Mylar on an optical flat of glass and cementing this to the plastic ring with Eastman 910 adhesive, to give a very flat, taut window.

In use, flies are lightly etherized and placed dorsally in the depression as close together as desired. The window is placed over the flies carefully and taped to the back piece. The flies are slightly compressed, holding them in place and minimizing the depth of the germ cells and its variation from fly to fly. Air is gently blown through the chamber every few minutes with a hypodermic syringe or rubber bulb until the flies recover from the anesthetic. Ventilation is adequate, since Rosemarie Binnard at the NASA Ames Research Center found no evidence of an oxygen effect in X-ray induced mutations when comparing standard gelatin capsules to these holders. Drosophila will live in them for several hours without forced ventilation, dying on longer containment from apparent dessication. Modification of the holders for continuous flow of moist air (or other gases) is simple. Keying of the two pieces to prevent rotation, as done by Jane Duffy at the Institute for Cancer Research, is recommended to prevent damage to the flies during assembly.

The thick lucite construction is adequate for most particle irradiations. The nuclear properties of H^1 and C^{12} minimize neutron production and bremstralungen, and tests have shown that such secondary reactions contribute less than 1% to the dose in these holders. It is important to have the diameter of the window larger than the beam, so that forward scattering will not contribute to the dose. A double thin window holder has been constructed for comparison, and is preferable for highly penetrating radiations. The double window design is also better for use with X-rays, since the back scatter from thick lucite significantly increases the dose absorbed from the incident radiation and its effective linear energy transfer. We are developing a more sophisticated design which will improve ventilation, increase compression of the flies and permit partial body irradiation. This should be useful with UV as well as charged particles, but the simplicity of the present design makes it preferred for routine work.

Hendrix, Nina, and Elizabeth Ehrlich. University of Oregon. A method for treating bacterial contamination of Drosophila cultures with antibiotics.

It was observed in our laboratory that the medium of some cultures turned a dark, reddish-brown color after several days, while others remained the light, yellow-brown color of the original new food. We had also noticed bacterial contamination in cultures sent to us from various laboratories throughout the country including the two major stock centers. Because in some of the cultures with this discoloration marked decreases in productivity were found, it was decided to try to identify the cause of the change in color.

By allowing flies from the darkly colored bottles to walk on a Petri dish containing new food and then removing them, it was found that the same dark color appeared on this food. From these dark areas sub-cultures were made with the advice of Dr. Priscilla Kilbourn of the University of Oregon, Department of Biology and using standard bacteriological identification methods, we found the organism apparently responsible for the discoloration to be of the Genus Achromobacter. It was a short, small, Gram-negative rod-shaped bacterium. This genus can be found in soil and water.

A sensitivity test was done using discs of broad-spectrum antibiotics and of sulfa drugs. Two drugs - dihydrostreptomycin and tetracycline - were found to have the best inhibitory effects on the bacterial growth.

For purposes of decontaminating the cultures, these two antibiotics were incorporated simultaneously into the regular fly media in the following proportions:

dihydrostreptomycin sulfate (Upjohn Co.)	100 micrograms/ml. food
tetracycline hydrochloride (Tetracyn Phizer)	30 micrograms/ml. food

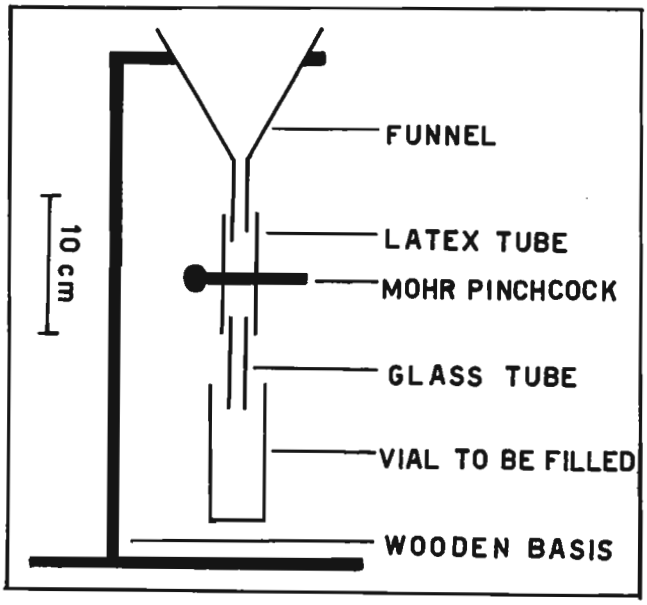
The procedure for decontamination is as follows: contaminated flies are put onto antibiotic food in bottles or vials. After a good number of eggs has been laid, all the parents are removed. The eggs are allowed to mature on the antibiotic food. After hatching, these new flies are transferred again to plain food without antibiotics, preferably without etherizing. If it is necessary to select the flies, care should be taken to clean brushes, etherizers, plates, etc., with 70% ethanol to prevent recontamination. The plain food cultures are then incubated and observed to see if the discoloration appears again.

Because the action of the antibiotics used may have unknown and potentially undesirable genetic effects, the drugs probably should not be used for very special stocks. The treatment should be completed in one generation.

We have found this method very satisfactory for ridding most of our cultures of all contamination. The productivity of the cultures is much improved with this treatment.

Gallo, A. J. Govêrno do Estado de São Paulo, Brazil. An apparatus for filling vials.

We saw this apparatus in a bacgeriological laboratory (Instituto Adolfo Lutz-São José do Rio Prêto) and we are using it with success in our own laboratory. It is specially useful because we can fill all vials with a standard quantity without dirtying the walls of the tubes. The vial is placed under the apparatus and gets filled when one presses the Mohr pinchcock. Only a wooden basis, a funnel, a piece of latex, a piece of glass tube and a Mohr pinchcock are required. According to the picture, it may be made in a proper size.



Lee, William R. The University of Texas. Feeding radioactive isotopes to specific larval stages of Drosophila melanogaster.

In previous work on feeding radioactive isotopes to Drosophila, flies during their entire life cycle have been kept in contact with or close to media containing the isotope. As a result the proportion of mutations induced by beta rays from the media is high in relation to those induced by the incorporation of the isotope into the genetic material. Pulse labeling can be accomplished by injection; however, results reported by Kaplan et al. (Genetics 49: 701-714) (also observed in this laboratory) show poor labeling of sperm from males injected with tritiated thymidine. Kaplan obtained better labeling by feeding larvae. Cytological considerations indicate the desirable time to feed would be just before synthesis of DNA prior to meiosis. Therefore, it is desirable to pulse label larvae during the first 24 hours of the third instar.

To accomplish feeding a radioactive isotope during a specific larval stage, 63 mm diameter and 29 mm deep Stender dishes are filled half full of corn meal Drosophila media. A sheet of black satin through which larvae cannot pass is pressed into contact with the surface of the media and is allowed to extend up on the sides of the Stender dish to prevent larvae from crawling over its edge into the media below. 0.05 ml of a solution containing 50 microcuries of phosphorus-32 as inorganic phosphate is pipetted onto the surface of the satin which initially absorbs most of the P-32. The surface of the satin is then sprayed with yeast and the Stender dish allowed to incubate at 34°C for three days. At the end of the three days the satin is covered with a growth of P-32 labeled yeast.

Larvae from a three day old Drosophila culture are floated out with a concentrated sugar solution and third instar larvae picked out with a brush. For convenience these larvae are stored in petri dishes on moistened filter paper during the collecting process. The larvae are then washed into a funnel screened with a disk of nylon chiffon. This concentrates the larvae into a cluster which is then brushed onto a 4 cm diameter disk of nylon chiffon. This disk is then transferred by 10 inch tongs to a Stender dish containing the labeled yeast.

Larvae are allowed to feed on the labeled yeast for 24 hours; then, the satin containing the larvae on its upper surface is removed with the tongs and placed in a funnel over a jar to collect radioactive materials. The funnel is lined with nylon chiffon which retains the larvae but allows the radioactive yeast and bits of media to be washed through. A fine stream of water from a polyethylene squeeze bottle is directed at the larvae and they are washed until further washing does not reduce the radioactivity. The nylon chiffon containing the washed larvae is then placed into a half-pint bottle with Drosophila media for pupation.

Radioautographs of sperm from females inseminated by males treated in the above manner with either P-32 or tritiated thymidine showed heavy uniform labeling during the first three days of mating yet the male germ cells had been subjected to radiation from radioactive media for only one day and during the relatively insensitive spermatogonial stage.

The 4 mm thick glass of the Stender dishes provides good shielding against the Beta rays of P-32. A wrist film badge worn during these operations has not recorded any significant radiation.

This investigation was supported by Public Health Service Grant GM 11449-02 from the National Institute of General Medical Sciences.

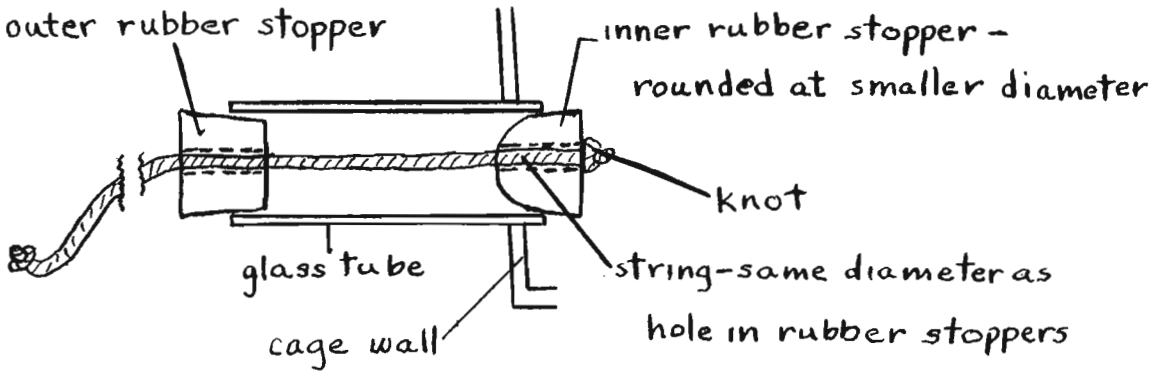
Strickberger, M. W. and L. Harth. St. Louis University. Ready-Made heated dispensers for Drosophila medium.

A simple dispenser for Drosophila medium is an ordinary automatic coffee percolator of the 32 cup variety or larger that is sold quite cheaply in hardware stores as "party" coffeemakers (our's cost $10.00). They are fitted with a spigot that easily controls the amount of medium dispensed and they have a warming element which keeps the medium hot and fluid. To use, remove the inside coffee "basket", add the cooked hot medium, and connect the apparatus to electricity. The hot medium will prevent the high voltage "percolator" element from operating and will restrict heating to the low voltage "warming" element. If desired, the high voltage heating element can be permanently disconnected by reconnecting the wiring so that its circuit is by-passed. In those instances when all of the medium is quickly dispensed through this device, use of the electrical warming element is not necessary.

Wilson, J. University of California at Riverside. A sampler for collecting adults from unetherized population cages.

Sampling adults from Bennett population cages yields the same phenotype frequency as egg samples, is faster, and disturbs the population only to the extent of removing some adults for a few minutes. The simple apparatus and technique described below gives samples of 100-200 flies in less than a minute if the population is moderately dense.

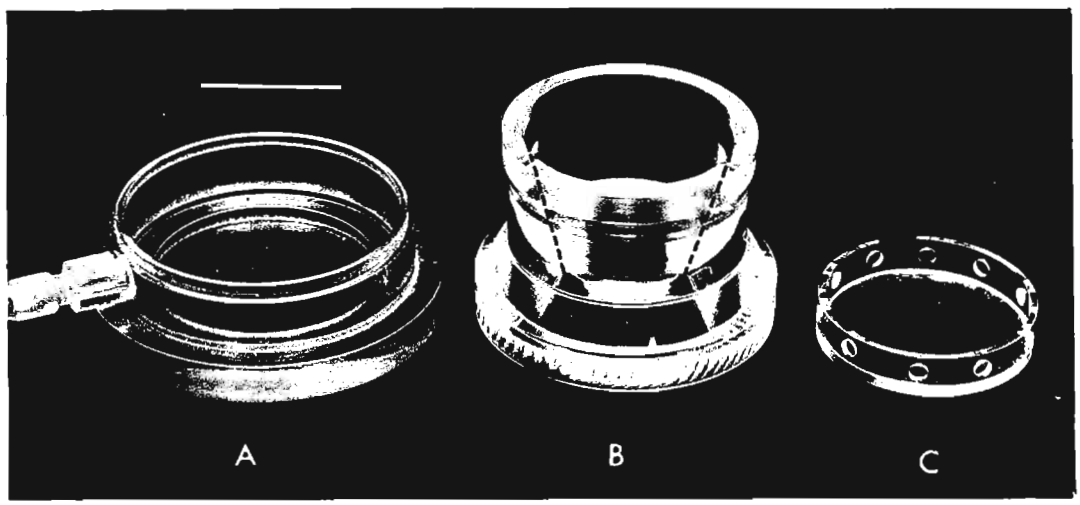
Materials and Assembly: See diagram.



Method: The assembled sampler is inserted in the cage as in the above diagram. The outer rubber stopper is withdrawn ca. two inches. The inner stopper is pushed out of the tube and the outer stopper is immediately replaced. The adults will move into the tube if it is on the lighter side of the cage. The inner stopper is then replaced by pulling the string. The sampling time should be short - 30 to 60 seconds - to obtain maximum density of adults and to avoid larvae in the sample. The tube is then withdrawn. Later, the flies may be replaced in the cage in an empty shell vial. The mortality in the sample is 2-3% due to crushing between the stopper and the wall of the tube.

Brown, E. H. and J. H. Sang. Poultry Research Centre, Edinburgh. A simple CO_2 anaesthetiser.

CO_2 was found to be the best anaesthetic for females being used in egg formation studies. Unlike ether which inhibits egg production, repeated tests showed that CO_2 had little or no detrimental effect even when females were kept under anaesthesia for some minutes. A simple anaesthetiser was turned from perspex in the form illustrated below. CO_2 is regulated through a simple bubble guage into the base (A) which has the perforated dish (C) fitted into it. The cone shaped top (B) is a push fit on to the base, and is made to accept the culture vials from which the adults are tapped. Its inner dimension fits closely onto the perforated dish. (The scale line represents 2 cm.)



Lewontin, R. C. University of Chicago. A cheap, disposable population cage.

Population cages can be made cheaply and quickly enough to be disposed of at the end of experiments or even during experiments by making use of the new polyfoam plastic containers made for picnic coolers, milk boxes, etc. The material is light, waterproof, cuts easily, and is extremely cheap. An example is given below of a cage suitable for most standard population experiments.

Materials

1	Polyfoam porch milk box, with cover, #40HA. Inside dimensions, 8-1/2" wide x 11-3/4" long x 11-1/2" deep. Wall thickness 1". Glo-Brite Products, 6415 N. California Ave., Chicago 15, Illinois.	Cost $2.95
1	Piece of 40 gauge brass mesh 4" x 12"	Cost $ .50
1	Piece of broadcloth or muslin 16" x 20"	Cost $ .10
	Total cost of disposable materials	$3.55

In addition, non disposable materials transferable from cage to cage are:

1	sheet window glass, 13-3/4" x 10-1/2"	Cost $ .75
15	polyethylene cups, 1-1/2" diam. x 2-1/4" high @ $.20 each, #1215: Dynalab Corp., Rochester, N. Y.	Cost $3.00

Make the cage as follows:

1. Cut height of the box down to 8" (outside dimension).
2. Cut rim off the cover so that the remaining piece fits inside the box.
3. Cut 15 holes in the cut-down cover, each hole slightly larger than a food cup.
4. Put cut-down cover with holes on the floor of the box inside to serve as a rack for the food cups.
5. Cut a hole 4-1/4" x 3-1/2" in end of the box, 2" down from the top.
6. Sew muslin into a sleeve or tube shape 16" long and tape into hole at end of box. Use plastic covered storm window tape or other exterior plastic tape.
7. Cut a hole 3" x 10-1/2" in side of the box and tape wire mesh to inside of this hole.
8. Cover the box with the glass plate and tape around the edges with masking tape.

The whole construction takes about one hour, under primitive conditions.

This box is completely fly tight provided the cloth sleeve is carefully doubled and tied after it is used each time. It is much easier to change cups in the cage by reaching through the sleeve than by the usual method of inserting the cups in holes in the cage and the size of the cups in the cage is not critical since they are completely enclosed.

* ANNOUNCEMENT *

SEVENTH DROSOPHILA RESEARCH CONFERENCE: May 8 and 9, 1965

In accordance with the decision reached at the 6th (Madison, Wisconsin) Drosophila Conference, the next meeting will be held in Seattle, Washington. I have chosen May 8th and 9th as reasonable dates. However, there is still time to change, and I invite any suggestions and objections both as to date and as to the format of the meetings.

Any such suggestions may be sent to:

L. Sandler
Department of Genetics
University of Washington
Seattle, Washington

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Edward H. Brown has accepted a position as Assistant Professor in the Department of Zoology at the University of Illinois in Urbana. He will begin this appointment in February, 1965. From January to December, 1964, he worked with Dr. James H. Sang of the Poultry Research Centre, Edinburgh, on a Postdoctoral Fellowship.

John Erickson, formerly of the University of Oregon, has joined the staff at Western Washington State College, Bellingham, Washington, where he is starting a new Drosophila laboratory. Reprints, old and new, would be appreciated.

John W. Gowen. In moving our research from Iowa State University to Colorado State University, a new laboratory of Drosophila genetics has been created. The laboratory is tied in closely with investigations having similar objectives - utilizing large populations of viruses, bacteria, mice and dogs. The inheritance studies cover those events which occur spontaneously, in disease and under irradiation or other environmental stimuli within the species named. All studies are integrated together as well as having the worthwhile cooperation of the disciplines in the College of Veterinary Science and University as a whole. Opportunities for graduate or post-graduate study form a significant part of the program.

T. Kanehisa, University of Kobe (Japan), has returned from the Istituto di Genetica, Universita Di Milano and is continuing work on the tumor-inducing factor of tumor stock Freckled.

Kobe University, Faculty of Science, Biological Laboratory has moved to a new building (Address: 12 Rokkodai-cho, Nadaku, Kobe, Japan).

Ingerid Kvelland is spending two years from October 1, 1964, as a Research Associate at the laboratory of Dr. Doermann, University of Washington, Seattle, Washington.

Ross J. MacIntyre has received the degree of Ph.D. from the Johns Hopkins University, October 1964, and has commenced a two-year postdoctoral fellowship in the Department of Plant Breeding, Cornell University, under Bruce Wallace. The thesis is entitled "Responses of Esterase-6 alleles of Drosophila melanogaster and Drosophila simulans to selection in experimental populations." The alleles are shown to reach stable equilibria when populations are initiated at different allele frequencies, and the particular equilibrium is shown to differ in stocks differently constructed in genotype. Heterosis is concluded to rest upon a polygenic basis, in this case.

George A. Marzluf has received the degree of Ph.D. from the Johns Hopkins University, October, 1964, and has entered upon a postdoctoral fellowship in the Department of Physiological Chemistry, University of Wisconsin Medical School, under Robert L. Metzenberg. The Ph.D. thesis is entitled "Studies of the Mechanism of Action of the Suppressor of Vermilion of Drosophila melanogaster," and compares the properties of the enzymes extracted and partially purified from the wild type and from suppressed vermilion, respectively.

Per Oftedal is spending the academic year at the Genetics Laboratory, City of Hope, Duarte, California.

Frank Seto, formerly of the Radiation Immunology group, Biology Division of the Oak Ridge National Laboratory in Oak Ridge, Tennessee, has joined the faculty of the Zoology Department, University of Oklahoma, Norman, Oklahoma. He will continue research in Developmental Biology.

University of Sussex. A school of Biology will open at this new University in October, 1965. Part of the University College, London, Drosophila group will move there with Professor J. Maynard Smith, and Professor J. H. Sang's Drosophila Unit, now at the Poultry Research Centre, Edinburgh, will also transfer to this school. Accomodation will be available for post-graduate and post-doctoral research workers interested in the areas now being studied by these two groups (see Directory), and in microbial genetics.

This new school will be glad to get reprints of past work in order to supplement its library facilities, and would welcome inclusion in reprint circulation lists.

D. L. Williamson, formerly an NIH Postdoctoral Fellow in Genetics, Department of Biology, Yale University, has joined the Department of Anatomy, Woman's Medical College of Pa., Philadelphia, Pa., as Assistant Professor of Research and is continuing studies on maternally inherited traits in Drosophila.

BIBLIOGRAPHY OF THE BIOCHEMICAL GENETICS OF DROSOPHILA*

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Introduction

No organism is as well characterized genetically nor is more amenable to sophisticated controlled genetic manipulation as is Drosophila. In spite of this Drosophila has contributed little to our knowledge of major genetical concepts at the molecular level. This fact is even more depressing when one realizes that Beadle and Euphrussi's classical studies on the eye pigments in Drosophila served as the forerunner of modern biochemical genetics. Indeed, of the approximately 100 different enzyme or enzyme systems known to be under genetic control in all organisms, Drosophila can only provide 7 or 8 clear cut examples. Furthermore, as of this writing there is not one example in Drosophila of a biochemical control mechanism under genetic influence. This critique could go on and on, but would only serve to belabor the point.

The reasons for this state of affairs is varied and complex, though perhaps the most difficult barrier has been at the technical level. However, two breakthroughs indicate a resurgence of molecular genetic studies in Drosophila. The first of these is the isolation of "biochemical mutants" by Novitski and his group (DIS 37:51) and the second is the successful cultivation of Drosophila tissue in vitro by M. Horikawa and A. S. Fox (Science, in press).

Anticipating this renewed interest created by these two formidable tools a bibliography of biochemical and biochemical genetic studies in Drosophila has been prepared. The source for all titles through 1962 has been the "Bibliography of Drosophila Genetics" first edited by H. J. Muller and then by I. H. Herskowitz. Titles from 1963 through approximately April 1964 were obtained from Current Contents. The classification of the papers should not be taken too seriously but rather only as a rough guide. It is to be noted this is not nor is it intended to be an exhaustive bibliography. Many times when a series of papers by the same author appeared on the same subject only the last paper was cited. In addition there are no doubt many errors of omission including only the most cursory survey of the non-english written journals.

Originally this bibliography was compiled for the private use of members of our laboratory in order to give a birdseye view of what has been done in Drosophila and hopefully to serve as a guide for future studies. It is hoped it will be of some use to other Drosophologists.

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DeMarinis, F. Cleveland, Ohio
Dempsey, T. P. Philadelphia, Pennsylvania
Dempster, E. R. Berkeley, California
Denniston, C. L. Madison, Wisconsin
Dewees, A. A. Lafayette, Indiana
Dharmarajan, M. India, Madras
Dickerman, R. C. Emporia, Kansas
Díez, J. Argentina, Buenos Aires
Dines, J. Australia, Brisbane
Di Pasquale, A. Italy, Milano
Dittrich, W. Germany, Münster
Doane, W. W. New Haven, Connecticut
Dobzhansky, T. New York, New York
Dolfini, S. Italy, Milano
Downie, J. Great Britain, Sussex
Drewes, K. DeKalb, Illinois
Druger, M. Syracuse, New York
Dyson, G. Raleigh, North Carolina
Early, R. C. Oak Ridge, Tennessee
Ebersole, L. A. Columbus, Ohio
Ebitani, N. Japan, Tokyo
Edington, C. W. Washington, D. C.
Edwards, J. W. Logan, Utah
Ehrlich, E. Eugene, Oregon
Ehrman, L. New York, New York
Eiche, A. Sweden, Stockholm
Elequin, F. Austin, Texas
El-Helw, M. R. U.A.R., Alexandria
Ellgaard, E. Iowa City, Iowa
El-Wakil, H. H. U.A.R. , Alexandria
Ely, S. H. Logan, Utah
El-Zoukka, T. A. U.A.R., Alexandria
Engelking, A. B. Austin, Texas
Englert, D. C. Carbondale, Illinois
English, D. S. Ames, Iowa
Epler, J. L. Oak Ridge, Tennessee
Erk, F. C. Stony Brook, New York
Erway, L. Davis, California
Eschenberg, K. New Haven, Connecticut
Estevez, M. Argentina, Buenos Aires
Faberge, A. C. Austin, Texas
Fahmy, M. J. Great Britain, Buckinghamshire
Fahmy, O. G. Great Britain, Buckinghamshire
Falk, C. T. Pittsburgh, Pennsylvania
Falk, R. Israel, Jerusalem
Farqué, C. Eugene, Oregon
Farhang, M. Eugene, Oregon

Farley, B. Bar Harbor, Maine
Farnsworth, M. W. Buffalo, New York
Farrow, M. G. Pittsburgh, Pennsylvania
Faulhaber, I. Baltimore, Maryland
Fedoroff, N. Syracuse, New York
Felsenstein, J. Chicago, Illinois
Fenner, H. Chile, Santiago
Fernández, R. Chile, Santiago
Fielding, C. Great Britain, London
Finlay, D. Australia, Sydney
Finnerty, V. G. Storrs, Connecticut
Folkers, W. Netherlands, Haren
Forbes, C. Moscow, Idaho
Forrest, H. S. Austin, Texas
Fortner, J. M. Athens, Georgia
Forward, K. Ann Arbor, Michigan
Fox, A. S. Madison, Wisconsin
Frahm, R. Raleigh, North Carolina
Frankel, A. Iowa City, Iowa
Frankham, R. Australia, Sydney
Fraser, A. S. Davis, California
Fraser, T. Philadelphia, Pennsylvania
Freeborn, J. Ridgefield, Connecticut
Friedberg, E. Israel, Jerusalem
Friedman, L. D. Hiram, Ohio
Fristrom, J. Pasadena, California
Frydenberg, O. Denmark, Copenhagen
Frye, S. H. Austin, Texas
Fuchs, M. S. Madison, Wisconsin
Fujii, S. Japan, Kobe
Fukui, K. Japan, Hiroshima
Fuller, C. W. Salt Lake City, Utah
Fuscaldo, K. E. Philadelphia, Pennsylvania
Fusté, M. Spain, Barcelona
Futch, D. Pasadena, California
Gall, G.A.E. Lafayette, Indiana
Gallo, A. J. Brazil, São Paulo
Gallucci, E. Italy, Milano
Garcia-Bellido, A. Switzerland, Zürich
Gardner, E. J. Logan, Utah
Gay, H. Ann Arbor, Michigan
Gay, P. France, Gif-sur-Yvette
Gehring, W. Switzerland, Zürich
Geiger, H. R. Pasadena, California
German, E. H. Colombia, Bogota
Gersh, E. S. Philadelphia, Pennsylvania
Gerstenberg, V. L. Austin, Texas
Geer, B. W. Galesburg, Illinois
Gfeller, Sr. M. D. Eugene,Oregon
Giavelli, S. Italy, Milano
Giesel, B. J. Eugene, Oregon
Gifford, M. J. East Lansing, Michigan
Gill, K. S. New Haven, Connecticut
Gilmore, G. Iowa City, Iowa
Glanges, E. Austin, Texas
Glass, H. B. Baltimore, Maryland
Glass, S. S. Baltimore, Maryland
Glassman, E. Chapel Hill, North Carolina
Gleditsch, A. Norway, Blindern
Gloor, H. J. Netherlands, Leiden
Goedhart, A. Netherlands, Leiden
Goetz, W. Switzerland, Zürich
Golden, H. Buffalo, New York
Goldschmidt, E. Israel, Jerusalem
Golley, R. Iowa City, Iowa
Gonzalez, F. W. Madison, Wisconsin
Gorovsky, M. Chicago, Illinois
Gottlieb, F. J. Pittsburgh, Pennsylvania
Gowen, J. W. Fort Collins, Colorado
Grabicki, E. New Haven, Connecticut
Graf, P. Tucson, Arizona
Granobles, L. A. Colombia, Bogota
Grant, B. Raleigh, North Carolina
Grant, R. New Haven, Connecticut
Grassmann, A. Switzerland, Zürich
Green, M. M. Davis, California
Greer, W. Eugene, Oregon
Gregg, T. G. Oxford, Ohio
Grell, E. H. Oak Ridge, Tennessee
Grell, R. F. Oak Ridge, Tennessee
Griffen, A. B. Bar Harbor, Maine
Groot, K. I. de Netherlands, Leiden
Grossfield, J. Austin, Texas
Grüneberg, H. Great Britain, London
Guest, W. C. Fayetteville, Arkansas
Guillaumin, M. France, Paris
Gurysh, R. A. Philadelphia, Pennsylvania
Gustafsson, K. Sweden, Stockholm
Haar, H. C. ter Netherlands, Haren
Hadorn, E. Switzerland, Zurich
Hakani, A. S. U.A.R., Alexandria
Hale, G. New York, New York
Halfer, C. Italy, Milano
Haller, H. J. Germany, Berlin
Hall, B. Eugene, Oregon
Hamouda, S. U.A.R., Alexandria
Hanimann, F. Switzerland, Zürich
Hanks, G. D. Salt Lake City, Utah
Hanly, E. W. Salt Lake City, Utah
Hannah, Alava A. Finland, Turku
Harrison, B. J. Great Britain, Hertford
Harth, L. St. Louis, Missouri
Hartman, A. Chicago, Illinois
Hartmann-Goldstein, I. J. S. A.,Johannesburg
Hartshorne, J. N. Great Britain, Manchester
Hastings, M. Great Britain, Sussex
Havin, E. Norway, Oslo
Hawkes, N. R. Logan, Utah
Hayashi, S. Canada, Vancouver
Hayman, D. L. Australia, Adelaide
Hebb, S. Emporia, Kansas
Heed, W. Tucson, Arizona
Heerema, N. Iowa City, Iowa
Hegab, H. S. U.A.R., Alexandria
Heinonen, P. Finland, Turku
Helton, N. Salt Lake City, Utah
Henderson, A. S. Chapel Hill, N. C.
Hennig, W. Germany, Tübingen
Henslee, E. St. Louis, Missouri
Herforth, R. S. Lincoln, Nebraska
Hernández, E. Spain, Barcelona
Hess, O. Germany, Tübingen
Heuts, M. J. Belgium, Louvain
Hexter, W. M. Amherst, Massachusetts
Hihara, F. Japan, Tokyo
Hillman, N. W. Philadelphia, Pennsylvania

Hillman, R. Philadelphia, Pennsylvania
Himoe, E. Madison, Wisconsin
Hinton, C. W. Athens, Georgia
Hiraizumi, Y. Japan, Misima
Hirsch, M. L. France, Gif-sur-Yvette
Hochman, B. Knoxville, Tennessee
Hodge, L. D. Chapel Hill, North Carolina
Hoenigsberg, H. F. Colombia, Bogota
Hoffer, H. C. Austin, Texas
Hollander, W. F. Ames, Iowa
Hollingsworth, M. J. Great Britain, London
Holm, D. G. Storrs, Connecticut
Holt, M. Eugene, Oregon
Holt, T. K. H. Netherlands, Leiden
Holzman, H. E. Chambersburg, Pennsylvania
Hoogmoed, M. S. Netherlands, Leiden
Horikawa, M. Madison, Wisconsin
Horwood, P. J. Great Britain, Buckinghamshire
Hotz, G. Germany, Karlsruhe
House, V. L. Columbus, Ohio
Hubby, J. L. Chicago, Illinois
Hunt, C. L. Johnson City, Tennessee
Hunt, D. M. Great Britain, Sheffield
Huston, L. Pittsburgh, Pennsylvania
Hyde, T. New Haven, Connecticut
Ichida, H. Japan, Tokyo
Ikeda, H. Japan, Tokyo
Imaizumi, Y. Japan, Chiba-City
Inagaki, E. Japan, Chiba-City
Inouye, I. Japan, Kobe, Okamoto
Ishikawa, M. Japan, Hiroshima
Iturra, P. Chile, Santiago
Ives, P. T. Amherst, Massachusetts
Iyama, S. Japan, Misima
Jackson, W. D. Australia, Hobart
Jeavons, B. New Haven, Connecticut
Jenkins, J. Logan, Utah
Jensen, B. A. Carbondale, Illinois
Jensen, R. Tucson, Arizona
Jha, A. P. India, Varanasi
John, F. Canada, Vancouver
Johnson, F. M. Madison, Wisconsin
Johnson, G. R. Berkeley, California
Johnston, F. Storrs, Connecticut
Jokela, T. A. Pullman, Washington
Jones, J. K. Great Britain, Reading
Jones, L. P. Australia, Sydney
Jost, P. Eugene, Oregon
Jozwiak, J. Buffalo, New York
Judd, B. H. Austin, Texas
Judd, S. E. Chapel Hill, North Carolina
Kadel, B. Baltimore, Maryland
Kaji, S. Japan, Kobe, Okamoto
Kale, P. G. India, Varanasi
Kambysellis, M. New Haven, Connecticut
Kan, J. Madison, Wisconsin
Kanehisa, T. Japan, Kobe
Kaneko, A. Japan, Sapporo
Kanellis, A. Greece, Thessaloniki
Kanfer, P. S. Pittsburgh, Pennsylvania
Kang, S. H. Madison, Wisconsin
Kang, Y. S. Korea, Seoul
Kantor, P. Syracuse, New York
Kaplan, M. New York, New York
Kaplan, W. D. Duarte, California
Karam, J. D. Chapel Hill, North Carolina
Karlik, A. Austria, Wien
Kasnic, G. Philadelphia, Pennsylvania
Kastritsis, C. Austin, Texas
Kataoka, Y. Japan, Tokyo
Kaufmann, B. P. Ann Arbor, Michigan
Kawabe, M. Japan, Kobe
Kawanishi, M. Japan, Misima
Kearsy, M. Raleigh, North Carolina
Keith, A. Eugene, Oregon
Kelly, A. Syracuse, New York
Kenyon, A. Athens, Georgia
Kernaghan, R. P. Stony Brook, New York
Kessler, S. New York, New York
Khan, F. M. Australia, Brisbane
Kim, K. W. Korea, Kwangju, Chunnam
Kim, Y. J. Korea, Seoul
Kimura, M. Japan, Misima
Kindred, B. M. Australia, Sydney
King, R. C. Evanston, Illinois
Kircher, H. Tucson, Arizona
Kiriazis, W. Hiram, Ohio
Kirschbaum, W. F. Argentina, Buenos Aires
Kitagawa, O. Japan, Tokyo
Kitazume, Y. Japan, Kobe
Kläden, B. Germany, Berlin
Klepper, C. Netherlands, Leiden
Klerk, T. H. de Netherlands, Leiden
Klingele, Sr. M. B. Notre Dame, Indiana
Knapp, T. Syracuse, New York
Knight, G. R. Great Britain, Edinburgh
Kobayashi, S. Japan, Misima
Koch, E. A. Evanston, Illinois
Koch, R. Switzerland, Zürich
Kojima, K. Raleigh, North Carolina
Konarski, M. Philadelphia, Pennsylvania
Koref-Santibañez, S. Chile, Santiago
Koske-Westphal, T. Germany, Hamburg
Kosswig, C. Germany, Hamburg
Kosuda, K. Japan, Tokyo
Kramer, W. N. Netherlands, Haren
Krause, E. Lafayette, Indiana
Krauss, M. I. Storrs, Connecticut
Krimbas, C. B. Greece, Athens
Krivshenko, E. D. Rochester, New York
Krivshenko, J. D. Rochester, New York
Kroeger, H. Switzerland, Zürich
Kuhn, D. T. Salt Lake City, Utah
Kuhn, J. Davis, California
Kühner, A. Switzerland, Zürich
Kuich, L. L. Austin, Texas
Kuile, A. E. ter Netherlands, Leiden
Kung, H. H. Chicago, Illinois
Kunze-Mühl, F. Austria, Wien
Kurokawa, H. Japan, Tokyo
Kusyk, C. Chapel Hill, North Carolina
Kuyen, C. J. van Netherlands, Leiden
Kvelland, I. Norway, Blindern
Kyle, W. H. Lafayette, Indiana
Lagowski, J. M. Austin, Texas
Lakovaara, S. Finland, Helsinki

Lamborot, M. Belgium, Louvain
Lamprecht, J. Switzerland, Zürich
Lange, E. L. Lafayette, Indiana
Langer, B. Pittsburgh, Pennsylvania
Lansky, L. Philadelphia, Pennsylvania
Laufer, H. Baltimore, Maryland
Laverde, A. Colombia, Bogotá
Lawson, B. K. Johnson City, Tennessee
Lee, C. C. Korea, Seoul
Lee, C. S. Korea, Seoul
Lee, B. W. Korea, Seoul
Lee, T. J. Korea, Seoul
Lee, W. R. Austin, Texas
Lefevre, G. Cambridge, Massachusetts
LeFever, H. Austin, Texas
Leigh, B. Great Britain, Edinburgh
Leon, W. N. Argentina, Buenos Aires
Leone, J. G. Baltimore, Maryland
Levene, H. New York, New York
Leventhal, E. New Haven, Connecticut
Levin, S. Stony Brook, New York
Levine, L. New York, New York
Levine, R. P. Cambridge, Massachusetts
Levitan, M. Philadelphia, Pennsylvania
Levy, A. Australia, Adelaide
Levy, J. H. Philadelphia, Pennsylvania
Lewenhaupt, M. Sweden, Stockholm
Lewis, E. B. Pasadena, California
Lewis, H. W. Washington, D. C.
Lewontin, R. C. Chicago, Illinois
Lezzi, M. Switzerland, Zürich
L'Heritier, P. France, Gif-sur-Yvette
Lindsley, D. L. Oak Ridge, Tennessee
Ling, L. L. Madison, Wisconsin
Lints, C. V. Belgium, Louvain
Lints, F. A. Belgium, Louvain
Locatelli, F. Italy, Milano
Lommerse, M. A. H. Netherlands, Leiden
Louw, W. Great Britain, Edinburgh
Lovell, R. H. Notre Dame, Indiana
Lucchesi, J. Eugene, Oregon
Luchowski, E. Buffalo, New York
Lüers, H. Germany, Berlin
Lüers, T. Germany, Berlin
Lundin, D. J. Ridgefield, Connecticut
Lüning, K. G. Sweden, Stockholm
Machida, I. Japan, Chiba-City
Macias Neto, V. Brazil, São Paulo
Maeda, Y. Japan, Kobe
Magaribuchi, K. Japan, Kobe
Mainx, F. Austria, Wien
Makino, S. Japan, Sapporo
Malagolowkin-Cohen, C. New York, New York
Mallah, G. S. U.A.R., Alexandria
Manna, G. K. India, West Bengal
Marien, D. New York, New York
Markowitz, E. Madison, Wisconsin
Maruyama, T. Madison, Wisconsin
Masri, A. M. U.A.R., Alexandria
Massasso, J. Great Britain, Buckinghamshire
Massimillo, L. Philadelphia, Pennsylvania
Masuda, H. Japan, Misima
Mather, W. B. Australia, Brisbane
Matthew, C. Great Britain, Edinburgh
Mattson, T. L. Madison, Wisconsin
Mayeda, K. Detroit, Michigan
Mayo, M. J. Australia, Adelaide
Mazar-Barnett, B. Argentina, Buenos Aires
McCarron, Sr. M. J. Philadelphia, Pa.
McCarty, J. Emporia, Kansas
McCloskey, J. Seattle, Washington
McCrady, W. B. Arlington, Texas
McCune, T. B. Knoxville, Tennessee
McDonald, M. M. Pullman, Washington
McFarlane, J. L. Riverside, California
McIntire, S. Oxford, Ohio
McKinley, K. Austin, Texas
McNew, R. W. Lafayette, Indiana
Mead, C. Oak Ridge, Tennessee
Medina, L. Colombia, Bogotá
Megna, F. Italy, Napoli
Meiller, C. R. Knoxville, Tennessee
Mensua, J. L. Spain, Barcelona
Merckx, F. Belgium, Louvain
Merrell, D. J. Minneapolis, Minnesota
Merriam, J. R. Seattle, Washington
Mettler, L. E. Raleigh, North Carolina
Meyer, G. F. Germany, Tübingen
Meyer, H. U. Madison, Wisconsin
Micheli, A. Italy, Roma
Mickey, G. H. Ridgefield, Connecticut
Milkman, R. D. Syracuse, New York
Miller, D. D. Lincoln, Nebraska
Milner, P. N. Austin, Texas
Minamori, S. Japan, Hiroshima
Mindek, G. Switzerland, Zürich
Mitchell, A. Pasadena, California
Mitchell, H. K. Pasadena, California
Mittler, J. E. DeKalb, Illinois
Mittler, S. DeKalb, Illinois
Mojica, T. Colombia, Bogotá
Momma, E. Japan, Sapporo
Monclús, M. Spain, Barcelona
Montalenti, G. Italy, Roma
Montelius, I. Sweden, Stockholm
Moon, H. M. Chapel Hill, North Carolina
Moray, N. Great Britain, Sheffield
Moree, R. Pullman, Washington
Morihira, K. Japan, Hiroshima
Moriwaki, D. Japan, Tokyo
Mortenson, L. R. Storrs, Connecticut
Moskwinski, T. Notre Dame, Indiana
Mossige, J. C. Norway, Oslo
Mourad, A. M. U.A.R., Alexandria
Mourad, E. U.A.R., Alexandria
Mourão, C. A. Brazil, São Paulo
Moyer, S. Raleigh, North Carolina
Muckenthaler, F. A. Baltimore, Maryland
Mukai, T. Japan, Misima
Mukherjee, A. Germany, Tübingen
Mukherjee, R. Canada, Vancouver
Mukherjee, S. Germany, Tübingen
Mukherjee, U. Canada, Vancouver
Mulkay, L. Logan, Utah

Müller, A. Germany, Karlsruhe
Muller, H. J. Duarte, California
Mullins, J. Ames, Iowa
Muñoz, E. R. Argentina, Buenos Aires
Murphy, J. Austin, Texas
Myren, J. Tucson, Arizona
Myszewski, M. E. East Lansing, Michigan
Nadal, A. Spain, Barcelona
Nagle, J. Raleigh, North Carolina
Nair, P. St. Louis, Missouri
Nakai, S. Japan, Chiba-City
Nakajima, K. Japan, Misima
Nakanishi, H. Y. Japan, Chiba-City
Nakao, Y. Japan, Chiba-City
Narayana Rao, N. India, Madras
Narda, R. D. India, Punjab
Nash, D. Madison, Wisconsin
Nassar, M. A. U.A.R., Alexandria
Natarajan, A. T. India, Delhi
Navas, Y. G. Colombia, Bogotá
Nawa, S. Japan, Misima
Negrotti, T. C. de Argentina, Buenos Aires
Nei, M. Japan, Chiba-City
Newby, C. M. Johnson City, Tennessee
Nicoletti, B. Italy, Roma
Niederhuber, S. A. Columbus, Ohio
Nienhaus, A. J. Netherlands, Groningen
Nilsson, M. B. Cambridge, Massachusetts
Nolte, D. J. South Africa, Johannesburg
Nonami, K. Japan, Tokyo
Norton, I. L. Oak Ridge, Tennessee
Nöthel, H. Germany, Berlin
Nöthiger, R. Switzerland, Zürich
Novitski, E. Eugene, Oregon
Obe, G. Germany, Berlin
Oden, C. E. Austin, Texas
Oftedal, P. Norway, Oslo
Ohanessian-Guillemain, A. France, Gif-sur-Yvette
Ohba, S. Japan, Tokyo
Ohnishi, E. Japan, Tokyo
Ohnishi, Y. Japan, Kobe
Ohta, T. Raleigh, North Carolina
Oishi, K. Japan, Misima
Okada, T. Japan, Tokyo
Oksala, T. A. Finland, Turku
Olberding, N. Ames, Iowa
Oliver, C. P. Austin, Texas
Olivieri, A. Netherlands, Leiden
Olivieri, G. Italy, Roma
Olivieri Mancini, A. Italy, Roma
Oostingh, H. T. Netherlands, Haren
Orem, J. Seattle, Washington
Ortiz, M. L. Mexico, Chapingo
Oshima, C. Japan, Misima
Oskamp, A. Netherlands, Utrecht
Oster, I. I. Philadelphia, Pennsylvania
Oster, P. Philadelphia, Pennsylvania
Overbeck, H. Germany, Berlin
Oxley, E. W. Duarte, California

Pack, W. Philadelphia, Pennsylvania
Paik, J. K. Austin, Texas
Paika, I. India, Punjab
Pakonen, C. Z. Pullman, Washington
Palomino, H. Chile, Santiago
Panciera, M. Tucson, Arizona
Parato, T. St. Louis, Missouri
Park, M. S. Korea, Kwangju
Parker, D. Cambridge, Massachusetts
Parker, D. R. Riverside, California
Parrish, J. L. Chapel Hill, North Carolina
Parry, D. Canada, Vancouver
Parshad, R. India, Punjab
Parzen, S. D. Madison, Wisconsin
Pasztor, L. Eugene, Oregon
Pavlovsky, O. New York, New York
Paz, C. Argentina, Buenos Aires
Peabody, A. Cambridge, Massachusetts
Peacock, J. Eugene, Oregon
Pearson, M. Great Britain, Sheffield
Peeples, E. E. Austin, Texas
Pelecanos, M. Greece, Thessaloniki
Pentzos-Daponte, A. Greece, Thessaloniki
Pereyra, E. Argentina, Buenos Aires
Perreault, W. Ann Arbor, Michigan
Perry, M. Great Britain, Edinburgh
Petermann, U. Switzerland, Zürich
Petersen, G. Logan, Utah
Peterson, J. B. Madison, Wisconsin
Petit, C. France, Paris
Petri, G. F. Logan, Utah
Peveling, E. Ann Arbor, Michigan
Phelan, J. Storrs, Connecticut
Phillips, B. J. Chapel Hill, North Carolina
Phillips, D. Syracuse, New York
Phillips, J. P. Logan, Utah
Pieters, J. Netherlands, Haren
Pipkin, S. B. Baltimore, Maryland
Piternick, L. K. Seattle, Washington
Plaine, H. L. Columbus, Ohio
Plaut, W. S. Madison, Wisconsin
Plotkin, H. Madison, Wisconsin
Plough, H. H. Amherst, Massachusetts
Plus, N. France, Gif-sur-Yvette
Podger, R. N. Australia, Sydney
Pontecorvo, G. Great Britain, Glasgow
Portin, P. Finland, Turku
Potthoff, R. F. Chapel Hill, North Carolina
Poulson, D. F. New Haven, Connecticut
Powers, L. M. Baltimore, Maryland
Prakash, S. St. Louis, Missouri
Preuss, B. Denmark, Copenhagen
Prevosti, A. Spain, Barcelona
Price, J. M. E. Great Britain, Buckinghamshir
Printz, P. France, Gif-sur-Yvette
Pritchard, R. H. Great Britain, Leicester
Proust, J. France, Orsay
Prout, T. Riverside, California
Prudhommeau, C. France, Orsay
Pui, M. L. L. du Netherlands, Haren
Pulitzer, J. F. Italy, Naples
Puro, J. Finland, Turku
Rakha, F. A. U.A.R., Alexandria
Ramanamurthy, C. V. Notre Dame, Indiana
Ramel, C. Sweden, Stockholm

Rath, L. Riverside, California
Rathie, K. A. Australia, Sydney
Ratty, F. J. San Diego, California
Rau, C. G. St. Louis, Missouri
Ray-Chaudhuri, S. P. India, Varanasi
Reddig, T. Emporia, Kansas
Reinhardt, K. DeKalb, Illinois
Remensberger, P. Switzerland, Zürich
Remondini, D. Logan, Utah
Rendel, J. M. Australia, Sydney
Resch, K. Austin, Texas
Rezzonico Raimondi G. Italy, Milano
Rhodes, C. D. Chapel Hill, North Carolina
Ribó, G. Spain, Barcelona
Richardson, R. Austin, Texas
Richmond, J. Philadelphia, Pennsylvania
Rinehart, R. R. San Diego, California
Ringelé, M. M. Belgium, Louvain
Ritterhoff, R. K. Baltimore, Maryland
Rizki, R. M. Ann Arbor, Michigan
Rizki, T. M. Ann Arbor, Michigan
Roberts, P. A. Oak Ridge, Tennessee
Robertson, A. Great Britain, Edinburgh
Robertson, F. W. Great Britain, Edinburgh
Roe, L. Eugene, Oregon
Roeder, C. Switzerland, Zürich
Rogers, B. L. Knoxville, Tennessee
Rojas, E. Chile, Santiago
Romero, I. F. Colombia, Bogotá
Ronen, A. Israel, Jerusalem
Roper, J. A. Great Britain, Sheffield
Rosenfeld, A. Seattle, Washington
Rosin, S. Switzerland, Bern
Roushdy, M. S. U.A.R., Alexandria
Rowan, Sr. M. J. Rochester, New York
Rubio, J. Great Britain, London
Ruderer, E. Austria, Wien
Rüegg, M. Switzerland, Zürich
Rümke, C. L. Netherlands, Utrecht
Rundell, C. Davis, California
Russell, D. Emporia, Kansas
Russell, J. Tucson, Arizona
Russell, P. Amherst, Massachusetts
Ryan, C. DeKalb, Illinois
Saeki, T. Japan, Chiba-City
Sakaguchi, B. Japan, Misima
Sakai, K. I. Japan, Misima
Saliternik, R. Israel, Jerusalem
Salverson, H. Madison, Wisconsin
Sandler, L. Seattle, Washington
Sang, J. H. Great Britain, Sussex
Sankaranarayanan, K. New York, New York
Sano, K. Japan, Misima
Sari Gorla M. Italy, Milano
Sarkar, D. N. India, Varanasi
Sasaki, F. Japan, Tokyo
Saul, G. Pasadena, California
Savolainen, S. Finland, Turku
Scandlyn, B. J. Madison, Wisconsin
Schalet, A. Storrs, Connecticut
Scharloo, W. Netherlands, Leiden
Scheidt, G. East Lansing, Michigan
Scheinberg, E. Lafayette, Indiana
Schewe, M. J. Chapel Hill, North Carolina
Schifflers, E. Belgium, Louvain
Schlentz, M. Tucson, Arizona
Schmidt, M. Austin, Texas
Scholefield, J. Canada, Vancouver
Schouten, S. C. M. Netherlands, Utrecht
Schneider, I. New Haven, Connecticut
Schneider-Minder, A. Switzerland, Zürich
Schubiger, G. Switzerland, Zürich
Schwarz, R. Philadelphia, Pennsylvania
Schwinck, I. Storrs, Connecticut
Scott, D. South Africa, Johannesburg
Scowcroft, W. R. Davis, California
Seecof, R. Duarte, California
Seiger, M. B. Rochester, New York
Sen, B. K. Great Britain, Edinburgh
Sendi, K. Eugene, Oregon
Sessi, J. Pittsburgh, Pennsylvania
Shamay, E. Israel, Jerusalem
Shantha, A. P. Syracuse, New York
Sharma, G. P. India, Punjab
Sharma, R. P. India, Delhi
Shaw, B. A. Philadelphia, Pennsylvania
Sheldon, B. L. Australia, Sydney
Shelton, E. E. Logan Utah
Shen, M. W. Austin, Texas
Sherif, M. I. U.A.R., Alexandria
Sherwin, R. M. Pittsburgh, Pennsylvania
Shideler, D. Lafayette, Indiana
Shima, T. Japan, Sapporo
Shiomi, T. Japan, Chiba-City
Shoeb, Y. Z. U.A.R., Alexandria
Shoham, Y. Israel, Jerusalem
Shoup, J. Chicago, Illinois
Sick, K. Denmark, Copenhagen
Sigot, A. France, Strasbourg
Silva, E. Brazil, São Paulo
Simmons, J. R. Logan, Utah
Simpson, M. L. Austin, Texas
Sims, M. Chicago, Illinois
Singeisen, C. Switzerland, Zürich
Singer, K. Storrs, Connecticut
Singh, A. India, Punjab
Singh, M. Chicago, Illinois
Sinnock, P. Raleigh, North Carolina
Sironi, G. P. Italy, Milano
Slatis, H. M. East Lansing, Michigan
Slizynska, H. Great Britain, Edinburgh
Slizynski, B. M. Great Britain, Edinburgh
Smit, A. Pasadena, California
Smith, D. Raleigh, North Carolina
Smith, K. P. Philadelphia, Pennsylvania
Smith, P. A. Evanston, Illinois
Smoler, M. H. Madison, Wisconsin
Sobels, F. H. Netherlands, Leiden
Sokoloff, A. Berkeley, California
Soliman, A. M. Ann Arbor, Michigan
Soliman, M. H. U.A.R., Alexandria
Solima-Simmons, A. New York, New York
Somerville, D. Baltimore, Maryland
Sparvoli, E. Ann Arbor, Michigan

Spassky, B. New York, New York
Sperlich, D. Austria, Wien
Spieler, R. A. Chicago, Illinois
Spiess, E. B. Pittsburgh, Pennsylvania
Spiess, L. D. Pittsburgh, Pennsylvania
Spieth, H. T. Austin, Texas
Spofford, J. B. Chicago, Illinois
Spring, H. Switzerland, Zürich
Springer, R. Austria, Wien
Spurway, R. Australia, Brisbane
Stadler, J. Fort Collins, Colorado
Stalker, H. D. St. Louis, Missouri
Stalker, S. E. Lexington, Kentucky
Stallard, M. A. Galesburg, Illinois
Staub, M. Switzerland, Zürich
Stavrou, H. Riverside, California
Steensma, K. T. Netherlands, Groningen
Steinberg, A. G. Cleveland, Ohio
Steiner, C. St. Louis, Missouri
Stevenson, R. Johnson City, Tennessee
Stewart, G. Storrs, Connecticut
Stewart, M. C. Oak Ridge, Tennessee
Stiers, R. O. Belgium, Louvain
Still, G. Great Britain, Buckinghamshire
Stone, W. S. Austin, Texas
Strachan, K. Great Britain, Edinburgh
Strickberger, M. W. St. Louis, Missouri
Strickland, B. C. Knoxville, Tennessee
Strömnaes, Ö. Norway, Blindern
Struck, E. Germany, Berlin
Sturtevant, A. H. Pasadena, California
Suárez, M. Spain, Barcelona
Suber, T. Philadelphia, Pennsylvania
Sugisaki, R. Japan, Misima
Suguna, S. G. India, Madras
Sullivan, A. Eugene, Oregon
Suomalainen, E. Finland, Helsinki
Suzuki, D. Canada, Vancouver
Swaminathan, M. S. India, Delhi
Swatek, J. Chicago, Illinois
Swift, H. Chicago, Illinois
Szekessy, M. M. B. Colombia, Bogotá
Taira, T. Japan, Misima
Takahashi, A. Japan, Misima
Takaya, H. Japan, Kobe
Talmage, S. H. Lexington, Kentucky
Tantawy, A. O. U.A.R., Alexandria
Tartof, K. Ann Arbor, Michigan
Tasca, R. Philadelphia, Pennsylvania
Tates, A. D. Netherlands, Leiden
Taylor, A. Cambridge, Massachusetts
Taylor, E. Emporia, Kansas
Taylor, R. K. Knoxville, Tennessee
Taylor, W. Chicago, Illinois
Teissier, G. France, Paris
Temin, R. G. Madison, Wisconsin
Terry, A. Chapel Hill, North Carolina
Thomas-Orillard, M. France, Paris
Thompson, C. F. Jr. Pittsburgh, Pennsylvania
Thompson, P. E. Ames, Iowa
Throckmorton, L. H. Chicago, Illinois
Tiffany, B. Amherst, Massachusetts
Tiivola, A. Finland, Helsinki
Tillinghast, B. Storrs, Connecticut
Tobari, I. Japan, Chiba-City
Tobari, Y. N. Japan, Tokyo
Tobler, H. Switzerland, Zürich
Tokuda, T. Japan, Misima
Tokumitsu, T. Japan, Sapporo
Tonzetich, J. Durham, North Carolina
Torres, I. P. Brazil, São Paulo
Torroja, E. Austin, Texas
Torroja, E. New York, New York
Towne, L. Emporia, Kansas
Traut, A. Germany, Münster
Traut, H. Germany, Münster
Travis, D. L. Pittsburgh, Pennsylvania
Trigg, C. E. Great Britain, Buckinghamshire
Trosko, J. E. Oak Ridge, Tennessee
Tsacas, S. Greece, Athens
Tschumi, P. Switzerland, Bern
Tuinstra, E. J. Netherlands, Utrecht
U, R. DeKalb, Illinois
Ufholz, I. Germany, Karlsruhe
Ulrich, H. Switzerland, Zürich
Ursprung, H. Baltimore, Maryland
Valencia, J. I. Argentina, Buenos Aires
Valencia, R. M. Argentina, Buenos Aires
Vandling, W. E. Eugene, Oregon
Van Valen, L. New York, New York
Vasistha, H. C. India, Varanasi
Vigier, P. France, Orsay
Vlist, J. v. d. Netherlands, Utrecht
Vloeberghs, J. V. Belgium, Louvain
Volkers, W. Netherlands, Leiden
von Borstel, R. C. Oak Ridge, Tennessee
Von Halle, E. S. Oak Ridge, Tennessee
Vovis, G. F. Galesburg, Illinois
Vreezen, W. Netherlands, Leiden
Waddington, C. H. Great Britain, Edinburgh
Wagner, R. P. Austin, Texas
Wahrman, J. Israel, Jerusalem
Waldner, R. Switzerland, Zürich
Walen, K. H. Berkeley, California
Walker, L. R. Athens, Georgia
Wallbrunn, H. M. Gainesville, Florida
Wallis, B. Madison, Wisconsin
Walsh, M. DeKalb, Illinois
Walth, G. D. Pullman, Washington
Ward, C. L. Durham, North Carolina
Wassenaar, M. C. Netherlands, Leiden
Wasserman, M. New York, New York
Watanabe, T. Japan, Kobe
Watanabe, I. Japan, Chiba-City
Watanabe, T. K. Japan, Misima
Watson, J. E. Lafayette, Indiana
Watson, W. A. F. Great Britain, Edinburgh
Wattiaux, J. M. Belgium, Louvain
Webb, H. S. Salt Lake City, Utah
Weber, U. Pasadena, California
Weisbrot, D. R. Medford, Massachusetts
Welshons, W. J. Oak Ridge, Tennessee
Westmoreland, D. Johnson City, Tennessee
Westphal, N. J. Lincoln, Nebraska

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