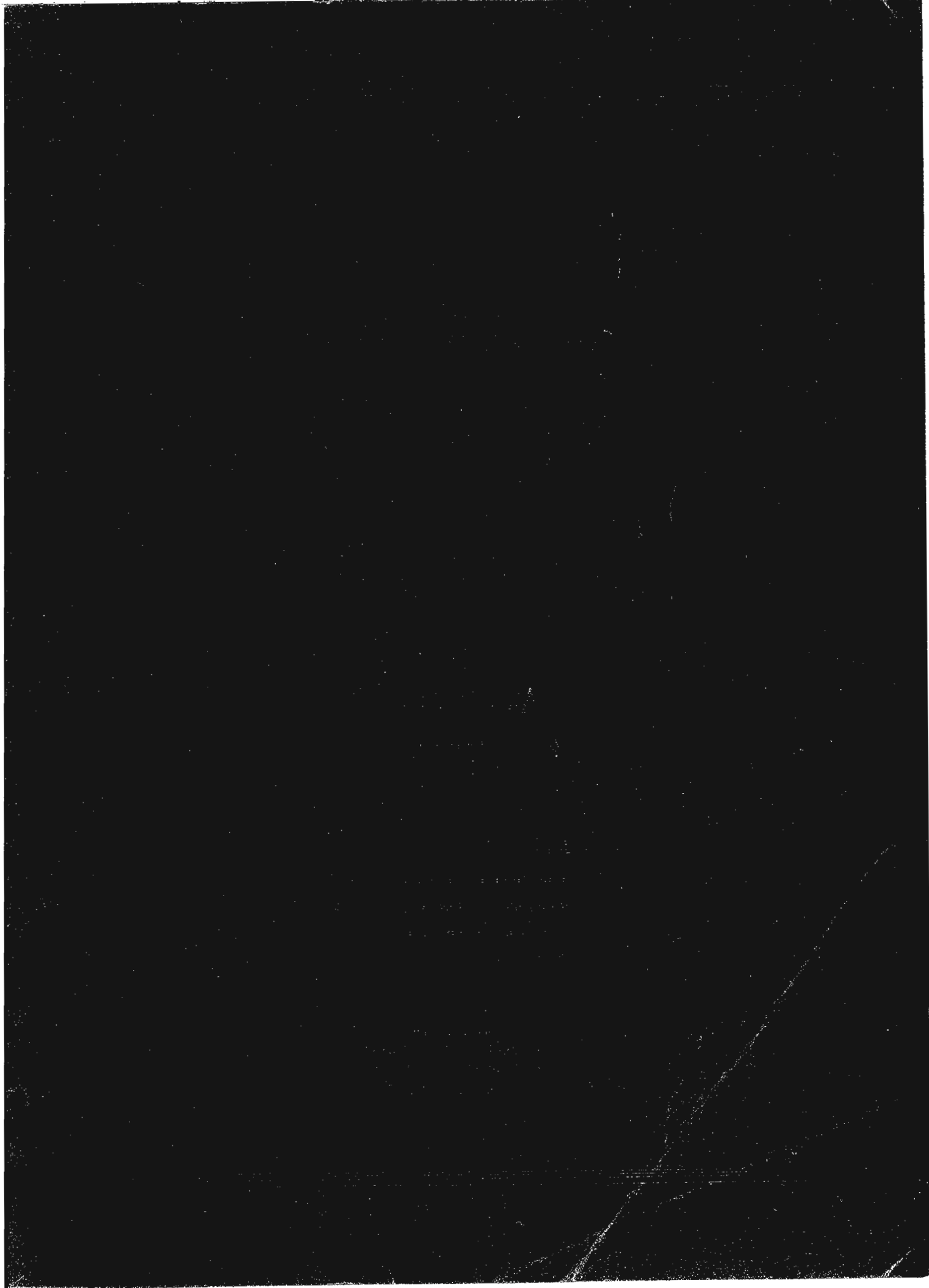

# DROSOPHILA INFORMATION SERVICE

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Technical Notes

Late Directory Additions (continued from page 243)

Schmidt, Maureen (Miss) Technical Assistant. Radiation effects, population studies.
Shannon, Mary Parker (Mrs.) B.A. N.I.H. Training Grant Predoctoral Fellow.
Shen, Margaret W. (Mrs.) M.S. Research Assistant. Recombination, cytology.
Stone, Wilson S. Ph.D. Professor. Evolution, gene action, radiation genetics.
Torroja, Eduardo Ph.D. Research Associate. Radiation genetics.
Wagner, Robert P. Ph.D. Professor. Gene action, biochemical genetics.
Wheeler, Marshall R. Ph.D. Professor. Taxonomy, evolution.
Wilson, Florence D. (Mrs.) B.A. Research Assistant. Radiation effects, population studies.
Yang, Hei L. (Mrs.) B.A. Graduate student.
Yoon, Jong Sik Ph.D. Research Associate. Population genetics, radiation genetics.
Yoon, Kyung (Mrs.) B.A. Research Assistant. Evolution, Hawaiian Drosophila.

## EIGHTH ANNUAL

## DROSOPHILA RESEARCH CONFERENCE

The Eighth Annual Drosophila Research Conference will be held at the University of Chicago on May 27-29, 1966.

Arrangements have been made to hold the conference in the Center for Continuing Education on the University of Chicago campus. This will allow all participants to sleep, eat, drink, and have their sessions under one roof.

Since transportation to and from Chicago is rapid from any part of the country (in fact, it takes less time to go from Chicago to Los Angeles than from O'Hare field to the campus), it is planned to have the opening general session on Friday night, May 27. The final session on Sunday afternoon, May 29, will end at approximately 3:30 to allow participants to catch evening flights from O'Hare field, or they can stay over and enjoy Memorial Day in the big city.

More details of the Conference and registration blanks will be sent about the first of the year. If you do not receive these, get in touch with William K. Baker, Department of Zoology, University of Chicago, Chicago, Illinois 60637.

## PASADENA, CALIFORNIA: CALIFORNIA INSTITUTE OF TECHNOLOGY

Note: For a description of new balancer designations appearing in this stock list, see DIS 27:57-58 for FM1, SM1 and TM1; DIS 28:77 for FM3 and FM4; DIS 29:75 for SM5; DIS 30:71 for FM6; DIS 32:81 for FMA3 and DIS 34:51 for TM3. The symbol, *, is used for cross indexing and signifies that the mutant is carried in a stock whose number is shown in parenthesis.

### Wild Stocks

1 Canton-S
* Florida. . . . . 784
2 Lausanne-S
3 Oregon-R-C
4 Swedish-c
5 Urbana-S

### Chromosome 1

* ac . . . . . 167
6 amx/FM3, $y^{31d}$ $sc^{8}$ dm B 1
7 amx $lz^{g}$ v/y f:=
8 $amx^{55}$
* apr. . . . . (see $w^{a}$)
9 Ax
* bb . . . . . 24, 56, etc.
* $bb^{1}$. . . . . 696, 736, etc.
* $bb^{N}$. . . . . 162
* $bb^{poi}$. . . . . 127
* $bb^{sc5}$. . . . . 102
10 B
* $B^{3}$ . . . . . 39
* BB. . . . . 37
* $BB^{36b}$. . . . . 38
* $B^{i}B^{i}$ . . . . . 40
11 B $Bx^{r}$ car/y f:=
12 Bg B/In(1)AM
13 bi $ct^{6}$ $g^{2}$
14 bo
15 br
16 br $w^{e}$ ec rb $t^{4}$/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B
* $br^{3}$ . . . . . 638
17 Bx
18 $Bx^{2}$
19 $Bx^{3}$
20 $Bx^{J}$
* $Bx^{r}$. . . . . 11
* $Bx^{r49k}$ . . . . . 138
21 car
* cho . . . . . 91
* $cho^{2}$ . . . . . 178
22 cm
23 cm $ct^{6}$
* Co . . . . . 175
24 $cs^{53}$/y w bb
* $ct^{6}$ . . . . . 13, 23, etc.
* $ct^{K}$ . . . . . 168
25 $ct^{n}$ oc/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B
* cu-X . . . . . 742
* cv . . . . . 92, 93, etc.
26 cx
27 $cx^{tg}$ t/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B
28 dm/y f:=
29 dow/FM6, $y^{31d}$ $sc^{8}$ dm B
* $dvr^{2}$. . . . . 180
* dwx . . . . . 92
* dx . . . . . 33, 123
* $dx^{st}$ . . . . . 638, 639
30 dy
31 ec
32 ec $ct^{6}$ s car/FM6, $y^{31d}$ $sc^{8}$ dm B
33 ec dx
* en-bx . . . . . 743
* $en^{2}$-bx . . . . . 647
* $en^{X}$-S . . . . . 640
* eq . . . . . 93, 695, 792
34 Ext/FM6, $y^{31d}$ $sc^{8}$ dm B
35 f
36 f B/y f:=
37 f BB/y f:=
38 f $BB^{36b}$/y f:=
39 f $B^{3}$/y f:=
40 f $B^{i}B^{i}$/y f:=
41 f fu/y f:=
* $f^{3}$. . . . . 162
42 $f^{36a}$
* $f^{5}$. . . . . 152, 677
* $f^{B15}$. . . . . 733
* $f^{B27}$. . . . . 734
43 fa
* $fa^{n}$ . . . . . 738
44 flp
45 fo
* fu . . . . . 41
* fw. . . . . 141, 164
* $fw^{34e}$ . . . . . 186
46 $g^{2}$
47 $g^{2}$ pl/FM3, $y^{31d}$ $sc^{8}$ dm B 1
48 $g^{2}$ ty/y f:=
* $g^{4}$. . . . . 641, 677, etc.
49 $gg^{2}$/FM6, $y^{31d}$ $sc^{8}$ dm B
50 gt $w^{a}$
* Hw. . . . . 677, 739, 752
51 $Hw^{49c}$/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B
52 $if^{3}$
53 kz
54 l(1)7/FM6, $y^{31d}$ $sc^{8}$ dm B (nub$^{2}$/+)
55 l(1)$sc^{J1}$ $sc^{J1}$/l(1)J1 $sc^{J1}$/Del(1)24
56 lh B car bb/y f:=
57 lz/FM3, $y^{31d}$ $sc^{8}$ dm B 1
58 $lz^{3}$/y f:=
59 $lz^{34k}$/y f:=
60 $lz^{36}$/y f:=
61 $lz^{37h}$

62 $lz^{48f}$/y f:=
63 $lz^{BS}$ $lz^{46g}$ $ras^4$ v/y f:=
* $lz^g$ . . . . . 7, 172
* $lz^s$ . . . . . 16, 25, etc.
* $lz^{y4}$. . . . . 111
64 m
* $m^2$. . . . . 641, 677, etc.
65 $m^D$/FM3, $y^{31d}$ $sc^8$ dm B 1
66 M(1)n/FM6, $y^{31d}$ $sc^8$ dm B
67 M(1)o f/In(1)AM
68 M(1)Sp/In(1)AM
69 ma-1/y f:=
70 na/y f:=
71 ny f/FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B (ri)
72 oc $ptg^3$/C1B
73 od
74 pa/FM4, $y^{31d}$ $sc^8$ dm B
75 peb v
* pl. . . . . 47
* pn. . . . . 99, 169, etc.
76 $pn^2$
* $pn^3$ . . . . . 96
* ptg. . . . . 648
77 $ptg^2$
* $ptg^3$. . . . . 72
* $ptg^4$. . . . . 135
78 $r^9$/y f:=
* $r^{12}$. . . . . 140
79 $r^{39k}$ f B/In(1)AM
80 $ras_{dy}$
81 $ras^2$
82 $ras^3$ m
* $ras^4$. . . . . 63
* $ras^V$. . . . . 798
83 rb
84 rb cx
85 rg
86 $rst^2$/FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
87 $rux^2$
88 s
89 sbr
90 sc
91 sc cho
92 sc cv v dwx/FM6, $y^{31d}$ $sc^8$ dm B
93 sc cv v eq
94 sc cv v f
95 sc $ec_{cv}$ $ct^6$ $v_{g^2}$ f/FM3, $y^{31d}$ $sc^8$ dm B 1
96 sc $pn^3$ $g^{2rv}$ $Bx^2$ . . . . . ($g^2$ reverted)
97 sc z ec $ct^6$
98 sc z $w^{17G2}$ ec $ct^6$
99 $sc^2$ pn/y f:=
100 $sc^{3B}$
101 $sc^{3-1}$ w/y f:=
* $sc^4$ . . . . . 748
102 $sc^5$ $bb^{sc5}$
103 $sc^6$ $w^a$
* $sc^7$ . . . . . 682, 750, etc.
* $sc^8$ . . . . . 740, 755, etc.
* $sc^9$ . . . . . 759
104 $sc^{10}$ $w^a$
105 $sc^{10-1}$/y Hw
* $sc^{19}$ . . . . . 792
* $sc^{D2}$ . . . . . 174
* $sc^{J1}$ . . . . . 55
* $sc^{J4}$ . . . . . 726
* $sc^{S1}$ . . . . . 170, 724, etc.
* $sc^{S2}$ . . . . . 791
* $sc^{260-14}$ . . . . . 760
* $sc^{260-15}$ . . . . . 799
* $sc^{260-22}$ . . . . . 761
106 scp t
107 sd
108 $Sh^2$/FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
109 $shf^2$
* sl. . . . . (in C1b, $C1B^{36d}$)
* $sl^2$ . . . . . 683
* sn . . . . . 674, 801
* $sn^2$ . . . . . 156
110 $sn^3$
111 $sn^3$ $lz^{y4}$ v/y f:=
112 $sn^4$
113 $sn^{34e}$
114 $sn^{36a}$/y f:=
115 sp-w
116 spl
117 sta/FM3, $y^{31d}$ $sc^8$ dm B 1
118 sta/y f:=
* su-Cbx . . . . . 646
* su-Hw. . . . . 739
119 $su^2$-s v (bw)
120 $su^2$-s $w^a$ cv t
121 $su^3$-s cv v f (bw)
122 $su^{S2}$-v-pr v (bw)
* su-$w^a$. . . . . 669, 671, 672
* $su^w$-f. . . . . 139
123 $Su^X$-dx dx
124 svr
125 svr $w^a$
126 $svr^{poi}$
127 $svr^{poi\text{-}dish}$ $bb^{poi}$
128 sw
129 sx $vb^2$ sy/FM6, $y^{31d}$ $sc^8$ dm B
130 sy
131 t
132 $t^2$ v f
133 $t^3$
* $t^4$ . . . . . 16
134 tw/FM1,$y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
* ty . . . . . 48
* ty-1 . . . . . 735, 736
135 un $Bx^2$/In(1)AM, $ptg^4$
136 $un^4$
137 v
138 v f $Bx^{r49k}$ car/y f:=
139 v f $su^w$-f
140 v $r^{12}$
141 $v^2$ fw
142 $v^{36f}$
* $v^{0f}$. . . . . 737
143 vb
* $vb^2$. . . . . 129

144 vs
145 w
146 w m f
147 w $sn^3$ m
* $w^{11E4}$ . . . . . 191
* $w^{17G2}$ . . . . . 98
148 $w^a$
149 $w^{a2}$
150 $w^{a3}$
151 $w^{a4}$
152 $w^{bf}$ $f^5$
153 $w^{bf2}$
* $w^{bf3}$ . . . . . 666
154 $w^{Bwx}$
155 $w^{ch}$ $wy_2$
156 $w^{co}$ $sn^2$
157 $w^{col}$
158 $w^e$
159 $w^{e2}$
160 $w^{ec3}$
161 $w^h$
162 $w^i$ $f^3$ $bb^N$
163 $w^{sat}$
164 $w^t$ fw
165 $wy_2$
* $wy^2$ . . . . . 184
166 y
167 y $ac_v$
168 y $ct^K$
169 y pn
170 y pn w cm $ct^6$ $sn^3$ oc $ras^2$ v dy $g^2$ f od car sw/Ins(1)$sc^{S1}$, dl-49, y v B
171 y sc
172 y sc $lz^g$ v f/y f:=
173 y $sc^5$
174 y $sc^{D2}$
175 y w Co/y f:=
176 $y_2$w spl
177 $y^2$
178 $y^2$ $cho^2$
179 $y^2$ cv v f
180 $y^2$ $dvr^2$ v
181 $y^2$ sc $w^a$ ec
182 $y^2$ $w^a$
183 $y^2$ $w^a$ w
184 $y^2$ $wy^2$ $g^2$ ($g^2$ partly reverted?)
185 $y^{2S}$
186 $y^{2S}$ $fw^{34e}$
187 $y^{3d}$/y f:=
* $y^{3P}$ . . . . . 765
* $y^4$ . . . . . 767, 801
* $y^{31d}$ . . . . . 6, 16, etc.
188 $y^{34c}$
* $y^{59b}$ . . . . . 672
189 $y^{td}$
190 $y^{v2}$
191 z $w^{11E4}$

Chromosome 2

192 a px or
193 a px sp
194 ab
195 $ab^2$/T(Y;2)E
196 $ab^2$ $ix^2$ bw $sp^2$/Cy, $dp^{Th}$ Bl $L^4$ $sp^2$
* abb. . . . . 389
197 abr/Cy, $hk^2$
198 abr/SM5, $al^2$ Cy $lt^v$ $sp^2$
199 ad
200 al
201 al b c $sp^2$
202 al dp b bw 1(2)ax/SM5, $al^2$ Cy $lt^v$ $sp^2$
203 al dp b pr blt bw/SM5, $al^2$ Cy $lt^v$ $sp^2$
204 al dp b pr Bl c px sp/SM1, $al^2$ Cy $sp^2$
205 al dp b pr c px $sp^2$
206 al S ast ho/SM1, $al^2$ Cy $sp^2$
207 alpha-1 $p^p$
208 Alu
209 an/SM5, $al^2$ Cy $lt^v$ $sp^2$
210 $an^2$/SM1, $al^2$ Cy $sp^2$
211 ang
212 ant (ro)
213 $ap^4$/SM5, $al^2$ Cy $lt^v$ $sp^2$
214 arch ch1/SM5, $al^2$ Cy $lt^v$ $sp^2$
215 $ast_3$ho
* $ast^3$ . . . . . 770
216 $ast^4$ dp cl
* $ast^X$ . . . . . 288
* At . . . . . 818
217 b
218 b alpha-1
219 b cn beta
220 b el $rd^s$ pr cn
221 b Go/Gla
222 b Go/SM5, $al^2$ Cy $lt^v$ $sp^2$
223 b gp
224 b j
225 b l(2)Bld pr c px sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
226 b lt wxt bw
227 b nub pr
228 b pr tk/T(Y;2)G
229 b sf
230 b vg
* ba . . . . . 240
231 Bl/Cy, $bw^{45a}$ $sp^2$ $or^{45a}$
232 Bl/In(2LR)dp
233 Bl $L^2$/SM5, $al^2$ Cy $lt^v$ $sp^2$
234 Bl $stw^{48}$ blt tuf/SM5, $al^2$ Cy $lt^v$ $sp^2$
235 Bla/SM5, $al^2$ Cy $lt^v$ $sp^2$
236 blo
237 blt
238 $bs^2$
* $bs^3$ . . . . . 316
239 bw

240 bw ba
241 bw tu
242 $bw^{2b}$
243 $bw^{4}$
* $bw^{45a}$ . . . . . 231
244 $bw^{D}$
* $bw^{V34}$ . . . . . 337
245 c
246 c wt px
247 cg c/U
248 ch
249 ch1
250 ch1 en/SM5, $al^2$ Cy $lt^v$ $sp^2$
251 ch1 l(2)bw $bw^{2b}$ $mr^2$/SM5, $al^2$ Cy $lt^v$ $sp^2$
252 chy
253 ck/SM5, $al^2$ Cy $lt^v$ $sp^2$
254 cl
255 $cl^2$/T(Y;2)E
256 cn
* $cn^2$ .(in all stocks containing In(2R)Cy)
257 cn bw
258 cn en/SM5, $al^2$ Cy $lt^v$ $sp^2$
259 $cn^3$/T(Y;2)C
260 $cn^{35k}$
261 cq
262 cr-u/Cy;($w^e$)
263 d/SM5, $al^2$ Cy $lt^v$ $sp^2$
264 d b/SM5, $al^2$ Cy $lt^v$ $sp^2$
265 da/SM1, $al^2$ Cy $sp^2$
266 $dil^2$ hv bw sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
267 dke c
268 dp
269 dp cn bw
* $dp^2$ . . . . . 280, 281, etc.
270 $dp^{Nov}$
271 $dp^o$
272 $dp^{o2}$
273 $dp^{Rf}$/SM5, $al^2$ Cy $lt^v$ $sp^2$
* $dp^{Th}$ . . . . . 196
274 $dp^{tx}$ b/SM5, $al^2$ Cy $lt^v$ $sp^2$
* $dp^v$ . . . . . 658
275 $dp^{v2}$
276 $dp^{v1}$/SM5, $al^2$ Cy $lt^v$ $sp^2$
277 ds dp
278 $ds^{rv}$ ft $dp^{v2}$ l(2)M b pr/SM5, $al^2$ Cy $lt^v$ $sp^2$
279 ds S G b pr/Cy, $al^2$ $lt^3$ $L^4$ $sp^2$
280 $ds^W$/In(2L)Cyt, Su-S $dp^2$ pr
* $ds^{33k}$ . . . . . 316, 342
281 $ds^{38k}$/In(2L)Cy, Cy $dp^2$ b pr
282 dsr
283 dw-24F cl/SM5, $al^2$ Cy $lt^v$ $sp^2$
284 dw-24F l(2)cg, cg/SM5, $al^2$ Cy $lt^v$ $sp^2$
285 ed $Su^2$-dx
286 el
* en . . . . . 250, 258, 697
* En-S. . . . . 323, 381, etc.
* esc . . . . . 771
287 ex
288 ex ds $S^X$ $ast^X$/SM1, $al^2$ Cy $sp^2$
289 fes Alu lt/SM5, $al^2$ Cy $lt^v$ $sp^2$
290 fj l(2)Su-H/SM5, $al^2$ Cy $lt^v$ $sp^2$
291 fj wt/SM5, $al^2$ Cy $lt^v$ $sp^2$
292 fr/Cy, $dp^2$
293 $fr^2$ wt/SM5, $al^2$ Cy $lt^v$ $sp^2$
294 Frd/Cy, $sp^2$
295 ft
* G . . . . . 279
296 $G^{rv}$/SM5, $al^2$ Cy $lt^v$ $sp^2$
* Go . . . . . 221, 222
* gp . . . . . 223
* gt-4 . . . . . 402
* Hia . . . . . 423, 424
297 hk
298 hk pr
* $hk^2$ . . . . . 197
299 ho
300 hv/SM5, $al^2$ Cy $lt^v$ $sp^2$
301 Hx
302 hy/SM5, $al^2$ Cy $lt^v$ $sp^2$
303 hy a px sp/SM1, $al^2$ Cy $sp^2$
* ix . . . . . 362
* $ix^2$ . . . . . 196
304 j
305 J B1/In(2L)NS
306 $J^{34e}$
307 kn
308 L
309 $L^2$
310 $L^4$
311 $L^5$
312 $L^G$
313 $L^r$
314 $L^{si}$
* l(2)301 . . . . . 355
315 l(2)39a px slt sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
316 l(2)a $bs^3$, In(2L)t/Pm, $ds^{33k}$
* l(2)ax . . . . . 202
317 l(2)ay b c $sp^2$/SM5, $al^2$ Cy $lt^v$ $sp^2$
* l(2)Bld . . . . . 225
* l(2)bw . . . . . 251
* l(2)C . . . . . 385
* l(2)cg . . . . . 284
318 l(2)gl a px or/SM5, $al^2$ Cy $lt^v$ $sp^2$
319 l(2)H $L^2$/SM5, $al^2$ Cy $lt^v$ $sp^2$
* l(2)M . . . . . 278
320 l(2)mat/SM5, $al^2$ Cy $lt^v$ $sp^2$
321 l(2)me/SM1, $al^2$ Cy $sp^2$
* l(2)$mr^2$. . . . . 696
* l(2)R . . . . . 397
* l(2)Su-H . . . . . 290, 412
* ll . . . . . 351
322 $ll^2$
323 lm/Cy, $S^2$ $dp^2$ En-S
324 lt/T(Y;2)A
325 lt std/SM2, $al^2$ Cy $lt^v$ $sp^2$
326 lt $stw^3$
* $lt^3$ . . . . . 279, 815, 836
* $lt^v$ . . . . . 198, 202, etc.
327 ltd
328 lw
* lys . . . . . 659

329 M(2)173/SM5, $al^2$ Cy $lt^v$ $sp^2$
330 M(2)B/SM5, $al^2$ Cy $lt^v$ $sp^2$
331 M(2)$1^2$/SM1, $al^2$ Cy $sp^2$
332 M(2)S3/SM2, $al^2$ Cy $lt^v$ $sp^2$
333 M(2)S5/SM5, $al^2$ Cy $lt^v$ $sp^2$
334 M(2)S6/SM5, $al^2$ Cy $lt^v$ $sp^2$
335 M(2)S7/SM5, $al^2$ Cy $lt^v$ $sp^2$
336 M(2)S9/SM5, $al^2$ Cy $lt^v$ $sp^2$
337 M(2)S11/Cy, $bw^{V34}$
338 M(2)S11/SM5, $al^2$ Cy $lt^v$ $sp^2$
339 M(2)z/SM5, $al^2$ Cy $lt^v$ $sp^2$
340 M(2)z Sk b/In(2L)Cy, Cy $dp^2$ b pr
* Mal . . . . . 663
341 mi/$Pm^2$
342 mr $bs^2$/Pm, $ds^{33k}$
343 $mr^2$/Bld, In(2R)Cy
344 msf/SM5, $al^2$ Cy $lt^v$ $sp^2$
* N-2 . . . . . 399
345 net
346 net al ex ds S ast shv ho rub/SM1, $al^2$ Cy $sp^2$
347 net ed $Su^2$-dx
* nub . . . . . 227
348 $nub^2$
349 $nw^2$/Cy-RNS
* or . . . . . 192
* $or^{45a}$ . . . . . 231
350 pd
351 pd ll
352 pd $ll^2$ sp
353 Pfd/SM5, $al^2$ Cy $lt^v$ $sp^2$
354 pi/SM5, $al^2$ Cy $lt^v$ $sp^2$
355 pi l(2)30l/SM5, $al^2$ Cy $lt^v$ $sp^2$
* Pin . . . . . 401
356 pk cn
357 pk tuf ($sp^2$/+)
358 $po_2$vg
359 $po^2$
360 pr
361 pr cn/T(Y;2)C
362 pr cn ix/SM5, $al^2$ Cy $lt^v$ $sp^2$
363 $pr^{bw}$
364 pu
365 puf
366 pw-c/SM5, $al^2$ Cy $lt^v$ $sp^2$
367 px
368 px bl (old Berlin Stock of Goldschmidt) bl = bs (?)
369 px bw sp/T(Y;2)J
370 px bw mr sp/Pm, $ds^{33k}$
371 px slt sp
372 pys
373 Q
* rc . . . . . 659
374 rd/SM5, $al^2$ Cy $lt^v$ $sp^2$
* $rd^s$ . . . . . 220
375 rdo
376 $rdo^2$ pr
377 rh
378 rl
* rn . . . . . 830
* Roi. . . . . 425
379 rub
380 Ruf/Pm, $ds^{33k}$
381 S/Cy, En-S
382 S Sp $ab^2$ ltd/SM5, $al^2$ Cy $lt^v$ $sp^2$
* $S^2$ . . . . . 323, 729
383 $S^R$/Pm, $ds^{33k}$
* $S^X$ . . . . . 288
384 sca
385 sca 1(2)C/SM5, $al^2$ Cy $lt^v$ $sp^2$
386 SD-5/SM1, $al^2$ Cy $sp^2$
387 SD-72/SM5, $al^2$ Cy $lt^v$ $sp^2$
* sf . . . . . 229
388 $sf^2$
389 shr $bw^{2b}$ abb sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
390 shv
391 shv ho
* Sk . . . . . 340
* slt . . . . . 315, 371
392 sm px/SM5, $al^2$ Cy $lt^v$ $sp^2$
393 sm px pd/SM5, $al^2$ Cy $lt^v$ $sp^2$
394 so
395 $so^2$ b cn
* sp . . . . . 193, 204, etc.
396 $sp^2$ $bs^2$
397 Sp/In(2L)t, l(2)R
398 Sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
399 Sp Bl N-2/SM5, $al^2$ Cy $lt^v$ $sp^2$
400 Sp J/SM5, $al^2$ Cy $lt^v$ $sp^2$
401 Sp J $L^2$ Pin/SM5, $al^2$ Cy $lt^v$ $sp^2$
402 spd gt-2/SM5, $al^2$ Cy $lt^v$ $sp^2$
403 sple
404 spt
405 std/SM5, $al^2$ Cy $lt^v$ $sp^2$
406 $stw_2$
407 $stw^2$
408 $stw^3$/T(Y;2)B
409 $stw^5$
410 $stw^{48}$ blt tuf $sp^2$
* Su-dx . . . . . 639
* $Su^2$-dx . . . . . 347, 638
* Su-er. . . . . 662
411 Su-H/Cy, pr
412 Su-H whd l(2)Su-H/SM5, $al^2$ Cy $lt^v$ $sp^2$
* Su-S . . . . . 280
* tet . . . . . 644
413 Tft/SM1, $al^2$ Cy $sp^2$
414 tkd/SM5, $al^2$ Cy $lt^v$ $sp^2$
* tk . . . . . 228
415 tkv
416 tri $vg^{No2}$/SM5, $al^2$ Cy $lt^v$ $sp^2$
* tu . . . . . 241
* $tu^{36a}$ . . . . . 580
417 tuf ltd
418 Uf
419 vg
420 $vg^D$/SM5, $al^2$ Cy $lt^v$ $sp^2$
421 $vg^{ni}$
* $vg^{No2}$ . . . . . 416

422 $vg^{np}$
423 $vg^{nw}$ Hia/SM5, $al^2$ Cy $lt^v$ $sp^2$
424 $vg^{nw}$ Hia/T(2;3)$S^M$ Cy
425 $vg^U$/Roi, bw $sp^2$ or
426 vst/SM5, $al^2$ Cy $lt^v$ $sp^2$
427 whd
428 wt
* wxt . . . . . 226

Chromosome 3

429 a-3
430 aa h
431 aa $tu^{36e}$
432 abd
* $Antp^B$ . . . . . 780
433 app
434 $as^{hg}$
435 $as^{hg}$ $e^s$
* At. . . . . 818
436 bar-3
* Bd. . . . . 544
437 $Bd^G$/In(3R)C, l(3)a
438 bf/In(3R)C, el(3)e
* bod . . . . . 541
439 bp/TM1, Me ri $sbd^1$
440 bul
441 bv
* bx . . . . . .570, 584, etc.
* $bx^3$ . . . . . 560
442 $bx^3$ Cbx Ubx bxd pbx/Xa
443 $bx^{34e}$
* $bx^D$ . . . . . (=Ubx). . . .442, 595, 823
* bxd . . . . . 442, 823
* $bxd^{107}$. . . . . 852
* by . . . . . 553
* c3G . . . . . 576
444 ca
445 ca bv
446 ca K-pn
447 $ca^2$
448 Cbx
449 cd
450 cmp ca/In(3R)C, e
451 cp
452 cp in ri $p^p$
453 cu
454 cu kar
455 cur
456 cv-c
457 cv-c $sbd^2$
458 cv-d
* Cyd . . . . . 472
459 D/Gl
460 $D^3$ Sb $ca^2$/Payne
461 det
462 Dfd/In(3LR)Cx
463 $Dfd^r$
464 Dl H $e^s$ cd/In(3R)spr, spr
465 $Dl^3$/In(3R)C, e
466 $Dl^5$/In(3R)C, l(3)a
467 $Dl^7$/In(3LR)$Ubx^{130}$ $e^s$
468 $Dl^9$/In(3R)C, e
469 $Dl^{11}$/Payne, Dfd ca
470 $Dl^{12}$/Payne, Dfd ca
471 $Dl^{13}$/In(3R)C, Sb e l(3)e
472 $Dl^{14}$/In(3R)Cyd, Cyd
* $Dl^B$. . . . . 781
473 $Dl^x$/Payne
* Dr . . . . . 534
474 drb
475 dwh/Payne, Dfd ca
* e . . . . . .649, 650; In(3R)C
476 $e^4$ wo ro
477 $e^{11}$
478 $e^s$
* $e^x$ . . . . . 549
479 eg/In(3LR)Cx
480 $eg^2$/In(3LR)Cx
* er . . . . . 653, 662
481 eyg
* fl . . . . . 521
482 fz
483 gl
484 $gl^2$ $e^4$
485 $gl^3$
486 $gl^{60j}$
487 Gl Sb/LVM
* gm . . . . . 537, 599
488 gs
489 h
490 $h^2$
491 H/In(3R)hp, hp
492 H Pr/In(3R)C, e
493 $H^2$/Xa
494 $H^3$/In(3R)C, Sb e l(3)e
* $H^{57c}$ . . . . . 596
* $Hn^r$ . . . . . 498
495 $Hn^{r3}$ sr
* hp . . . . . 491
* Hu . . . . . 782
496 in
497 jv
498 jv $Hn^r$ h
499 jvl
* k . . . . . 564
* K-pn . . . . . 446
* kar. . . . . 454, 590
500 $kar^2$
501 Ki
502 l(3)36d10/In(3LR)Cx, D
* l(3)a. . . . . .437, 466, etc.
503 l(3)ac $e^s$ M(3)w/LVM
* l(3)e. . . . . .438, 471, etc.
* l(3)PL . . . . . (Payne; Payne, Dfd ca)
* l(3)PR . . . . . " "
504 l(3)tr Sb/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^s$
505 l(3)tr Ubx/TM1, Mé ri $sbd^1$
* l(3)W . . . . . 581, 781
* l(3)Xa . . . . . 834

506 ld
507 Ly/$D^3$
508 Ly Sb/LVM
509 M(3)124/In(3R)C, e l(3)e
510 M(3)36e/In(3R)C, l(3)a
511 M(3)40130/Payne, Dfd ca
512 M(3)B/In(3R)C, e l(3)e
513 M(3)$B^2$/In(3R)C, Sb e l(3)e
514 M(3)S32/T(2;3)Mé
515 M(3)S34/T(2;3)Mé
516 M(3)S36/T(2;3)Mé
517 M(3)S37/Mé
* M(3)w . . . . . 503
518 M(3)w/In(3R)C, e l(3)e
* M(3)x . . . . . 549
519 M(3)y/Mé
520 ma
521 ma fl
522 mah
523 Mc/Xa
524 N-X/Xa
525 obt
526 p
527 $p^p$
528 $p^p$ bx sr $e^s$
529 $p^p$ cu
530 pb/In(3LR)Cx
531 pbx/Xa
532 Pc/TM1, Mé ri $sbd^1$
* Pdr . . . . . 661
533 Pr/In(3R)C, e
534 Pr Dr/TM3, $y^+$ $ac^+$ ri $p^p$ sep $bx^{34e}$ $e^s$
535 Pt/Xa, ca
536 pyd
537 R Ly/In(3L)P, gm
538 ra
539 red
540 ri
541 ri bod $e^s$/Mé, In(3R)C, Sb e l(3)e
542 ri $p^p$/st, T(Y;2;3)F
543 ro
544 ro Bd ca/In(3R)C, l(3)a
545 ro ra ca/T(2;3)Mé
546 $rs^2$
547 rsd
* $rt^2$ . . . . . 563
548 ru
549 ru h th st $p^p$ H $e^s$ ro/Payne, M(3)x $e^x$
550 ru h th st cu sr $e^s$ ca
551 ru h th st cu sr $e^s$ Pr ca/T(2;3)Mé
552 ru h th st $p^p$ cu sr $e^s$
553 $ru^g$ jv se by
554 ry
555 $ry^2$
556 Sb/In(3LR)$Ubx^{101}$, $Ubx^{101}$
557 Sb H/In(3R)C, cd
558 Sb Ubx/Xa
559 $Sb^{Spi}$/In(3LR)Cx
* $Sb^V$ . . . . . 833
* sbd . . . . . 579
560 $sbd^2$ $bx^3$
* $sbd^1$ . . . . . TM1
* $sbd^{104}$ . . . . . 851
* $sbd^{105}$ . . . . . 715
561 se
562 se h
563 se $rt^2$ th/Mé
564 se ss k $e^s$ ro
* sed . . . . . ($=Hn^{r3}$)
* sep . . . . . 778;TM3
565 Ser/In(3R)C, e l(3)e
566 snb
* spr . . . . . 464
567 sr
568 sr gl
569 ss
570 ss bx $Su^2$-ss
571 ss bxd k $e^s$/Xa
572 $ss^a$
573 $ss^{a-B}$
574 $ss^{a-40a}$
575 st
576 st c3G ca/TM1, Mé ri $sbd^1$ ($sp^2$)
577 st in ri $p^p$
578 st Ki $p^p$
579 st sbd $e^s$ ro ca
580 st sr $e^s$ ro ca; $tu^{36a}$
581 st sr $H^2$ ca/In(3R)$P^W$, st l(3)W ca
582 $st^{sp}$
* su-pd. . . . . 648
583 $su^B$-pr/In(3R)C, e;(pr)
584 $su^2$-Hw bx bxd/TM1, Mé ri $sbd^1$($sp^2$)
* $Su^2$-ss . . . . . 570
585 su-t (t)
* su-tu . . . . . 662
586 su-ve ru ve h th
587 th
588 th st cp
589 th st pb $p^p$/In(3LR)Cx
590 th st pb $p^p$ cu kar $su^2$-Hw jvl ss bx sr gl/TM1, Mé ri $sbd^1$
* tra . . . . . 645
591 tt wo
592 Tu
* $tu^{36e}$. . . . . 431
593 tu-h
594 tx
595 Ubx $e^4$/Payne, Dfd ca
596 $Ubx^{61d}$/$H^{57c}$
* $Ubx^{101}$ . . . . . 556
* $Ubx^{130}$ . . . . . 467, 504, etc.
597 ve
598 ve h th
599 ve R/In(3L)P, gm
* vo-3 . . . . . 658
600 W
601 W Sb/In(3LR)Cx
602 wk/Payne, Dfd ca
603 wo

Chromosome 4

604 ar/ey$^{D}$
605 bt
606 br ey$^{R}$ sv$^{n}$
607 bt$^{D}$/ci$^{D}$
608 Ce$^{2}$/spa$^{Cat}$
609 ci ey$^{R}$
610 ci ey$^{R}$ sv$^{n}$
611 ci gvl bt
612 ci gvl ey$^{R}$ sv$^{n}$
613 ci sv$^{n}$
614 ci$^{361}$
615 ci$^{57g}$
616 ci$^{D}$/ey$^{D}$
617 ci$^{W}$
618 ey
619 ey$^{2}$
620 ey$^{4}$
* ey$^{D}$ . . . . . 604, 616, 636
* ey$^{R}$ . . . . . 606, 609, etc.
621 gvl
622 gvl ey$^{R}$
623 gvl ey$^{R}$ sv$^{n}$
624 l(4)AM-1/ci$^{D}$ (Hochman)
625 l(4)PT-1/ci$^{D}$ "
626 l(4)PT-2/ci$^{D}$ "
627 l(4)PT-3/ci$^{D}$ "
628 l(4)SLC-1/ci$^{D}$ "
629 l(4)ST-1/ci$^{D}$ "
630 l(4)ST-2/ci$^{D}$ "
631 l(4)ST-3/ci$^{D}$ "
* Mal . . . . . 663
632 spa
633 spa$^{Cat}$/ci$^{D}$
634 spa$^{pol}$
635 sv$^{35a}$
636 sv$^{de}$/ey$^{D}$
637 sv$^{n}$

Multichromosomal Stocks

638 br$^{3}$dx$^{st}$; ed Su$^{2}$-dx (1;2)
639 dx$^{st}$; Su-dx (1;2)
640 en$^{x}$-S; S/Cy (1;2)
641 lz$^{D}$/dl-49, m$^{2}$g$^{4}$; Cy/Pm (1;2)
642 v; bw (1;2)
643 v; In(2R)bw$^{VDe1}$/SM1, al$^{2}$ Cy sp$^{2}$ (1;2)
644 sy; tet (1;2)
645 w$^{a}$ v/FMA3, y$^{2}$; tra/In(3LR)Ubx$^{130}$ (1;3)
646 y su-Cbx v; Cbx (1;3)
647 y$^{2}$ en$^{2}$-bx w$^{bf}$/FMA3, y$^{2}$; sbd$^{2}$ ss bx$^{34e}$/TM1, Me ri sbd$^{1}$ (1;3)
648 ptg; px pd; su-pd (1;2;3)
649 y f:=; bw; e; ci ey$^{R}$ (1;2;3;4)
650 y f:=; bw; e; spa$^{pol}$ (1;2;3;4)
651 FMA3, y$^{2}$; net; sbd$^{2}$; spa$^{pol}$ (1;2;3;4)
652 al dp b Bl c px sp/Cy; D/Payne (2;3)
653 b(Su-er$^{+}$)bw; st er (2;3)
654 bw; st (2;3)
655 bw$^{4}$; st (2;3)
656 cn; ry$^{2}$ (2;3)
657 Cy/Pm, ds$^{33k}$; H/In(3R)Mo, sr (2;3)
658 dp$^{v}$; vo-3 (2;3)
659 lys rc; ss (2;3)
660 Pm, dp b/Cy, sp$^{2}$;Sb/D, CxF(ru h ca?) (2;3)
661 px pd; Pdr H, Dp(2;3)P/Pdr (2;3)
662 Su-er tu bw; st er su-tu (2;3)
663 pr; Mal (2;4)

Attached-X

664 br ec/y$^{3d}$
665 f B/su$^{S2}$-v-pr v
666 w$^{bf3}$ M(1)36f/w$^{bf3}$/sn$^{36a}$ (M(1)36f lost)
667 y/g$^{2}$ ty
668 y pn/FM6, y$^{31d}$ sc$^{8}$ dm B
* y pn v . . . . . 672
* y v f. . . . . 679
* y v f car. . . . . 736
* y w bb . . . . . 24
* y w f. . . . . 739
* y$^{2}$, FMA3 . . . (=FMA3); 645, 647, 651)
* y$^{2}$ sc w$^{a}$ ec. . . . . 673
669 y$^{2}$ su-w$^{a}$ w$^{a}$ bb/y sc$^{4L}$ sc$^{8R}$

Attached-XY

670 g$^{2}$ B, $\overline{XY'}$; Y"/y; Y"
671 v f B, $\overline{XY}$/y$^{2}$ su-w$^{a}$ w$^{a}$ bb
672 y$^{59b}$ su-w$^{a}$ w$^{a}$, $\overline{XY^{L}}$ . Y$^{S}$/y pn v

Triploid

673 y$^{2}$ sc w$^{a}$ ec/FM4, y$^{31d}$ sc$^{8}$ dm B

Extra Y

674 In(1)w$^{m4L}$ N$^{264-84R}$, y sn/FM3, y$^{31d}$ sc$^{8}$ dm B 1/Y; dm sn ♂ (DIS 28:137)

Closed-X

675 X$^{c}$, y/y f:=
676 X$^{c2}$, cv v f /ClB
677 In(X$^{c2}$)w$^{vc}$/y Hw dl-49 m$^{2}$ g$^{4}$ f$^{5}$ (Catcheside) (unstable ring)

Closed-Y

678 Y$^{c}$, bw/X$^{+}$; bw(fb "MYR")
679 Y$^{Lc}$/y w Y$^{S}$ and y v f

Deficiencies

Deficiencies-X

680 Df(1)259-4c Df(1)259-4c/FM4, y$^{31d}$ sc$^{8}$ dm B

681 Df(1)260-1 Df(1)260-1/FM4, $y^{31d}$ $sc^{8}$ dm B

682 Df(1)$B^{263-20}$ Df(1)$B^{263-20}$/Ins(1)$sc^{7}$, AM, $sc^{7}$ car

683 Df(1)bb In(1)bb-, $y$ $sl^{2}$ bb-/FM4, $y^{31d}$ $sc^{8}$ dm B

684 Df(1)bb In(1)bb-, y v car bb-/ In(1)AM

* Df(1)$bb^{1}$ . . . . . 696

685 Df(1)$ct^{268-42}$ Df(1)$ct^{268-42}$, y/FM4, $y^{31d}$ $sc^{8}$ dm B

686 Df(1)$g^{1}$ Df(1)$g^{1}$, f B/In(1)AM

687 Df(1)$N^{8}$ Df(1)$N^{8}$/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B

688 Df(1)$N^{264-39}$ Df(1)$N^{264-39}$, $w^{ch}$/FM4, $y^{31d}$ $sc^{8}$ dm B

689 Df(1)$N^{264-105}$ Df(1)$N^{265-105}$ (dm)/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B

* Df(1)$rst^{2}$ . . . . . 86

* Df(1)$sc^{4L}$ $sc^{8R}$ . . . . . 749

* Df(1)$sc^{8}$ . . . . . 726

690 Df(1)svr Df(1)svr, Dp(1;f)101 (Dp het. or hom.)

691 Df(1)$w^{258-11}$ Df(1)$w^{258-11}$, y/FM6, $y^{31d}$ $sc^{8}$ dm B

692 Df(1)$w^{258-42}$ Df(1)$w^{258-42}$, y/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B

693 Df(1)$w^{258-45}$ Df(1)$w^{258-45}$, y/FM4, $y^{31d}$ $sc^{8}$ dm B

694 Df(1)$w^{258-48}$ Df(1)$w^{258-48}$, y $sc^{5}$ spl; Dp(1;3)$w^{Vco}$; y f:=

695 Df(Y)$Y^{bb-}$ Df(Y)$Y^{bb-}$, $y^{2}$ eq

696 Df(Y)$Y^{st}$ $w^{e}$ $bb^{1}$/$w^{e}$ $bb^{1}$; $Y^{st}$ and $w^{e}$ $bb^{1}$; $Y^{+}$; NS, px sp/1(2)$mr^{2}$

* Df(Y)Y" . . . . . 670

697 Df(2)42 Df(2)42, en/SM1, $al^{2}$ Cy $sp^{2}$

698 Df(2)al Df(2)al/Cy, En-S

699 Df(2)$bw^{5}$ Df(2)$bw^{5}$, $sp^{2}$/Xa

700 Df(2)$bw^{VDe2L}$ $Cy^{R}$ Df(2)$bw^{VDe2L}$, In(2R)$Cy^{R}$/G1a

701 Df(2)M33a Df(2)M33a/$Pm^{2}$

702 Df(2)MB Df(2)MB/SM1, $al^{2}$ Cy $sp^{2}$

703 Df(2)MS4 Df(2)MS4/SM1, $al^{2}$ Cy $sp^{2}$

704 Df(2)MS8 Df(2)MS8/SM1, $al^{2}$ Cy $sp^{2}$

705 Df(2)MS10 Df(2)MS10/SM1, $al^{2}$ Cy $sp^{2}$

* Df(2)P . . . . . 706

706 Df(2)Px Df(2)Px/Df(2)P; Dp(2;3)P/In(3R)Mo, sr; $w^{e}$

707 Df(2)$Px^{2}$ Df(2)$Px^{2}$, bw sp/SM1, $al^{2}$ Cy $sp^{2}$

708 Df(2)$rl^{10a}$ Df(2)$rl^{10a}$ lt cn/Pm, $ds^{33k}$

709 Df(2)$S^{2}$ Df(2)$S^{2}$/Cy, En-S

710 Df(2)$S^{3}$ Df(2)$S^{3}$/SM1, $al^{2}$ Cy $sp^{2}$

711 Df(2)$vg^{B}$ Df(2)$vg^{B}$/SM5, $al^{2}$ Cy $lt^{v}$ $sp^{2}$

712 Df(2)$vg^{C}$ Df(2)$vg^{C}$/Rvd

713 Df(2)$vg^{C}$ Df(2)$vg^{C}$/SM5, $al^{2}$ Cy $lt^{v}$ $sp^{2}$

* Df(2)$vg^{D}$ . . . .(= $vg^{D}$). . 420

Deficiencies-3

* Df(3)Ly . (= Ly) 507, 508, 531

714 Df(3)MS31 Df(3)MS31/T(2;3)Mé

715 Df(3)$sbd^{105}$ Df(3)$sbd^{105}$/Xa

Deficiencies-4

716 Df(4)M4 Df(4)M4/$ey^{D}$

Duplications

* Dp(1;f)24 .(=Del(1)24). .55, 762

717 Dp(1;f)101 In(1)$sc^{8}$, Df(0+ac). $w^{a}$ $sc.^{8}$; Dp(1;f)101

718 Dp(1;f)107 In(1)$sc^{8}$, Df(0+ac). $w^{a}$ $sc.^{8}$; Dp(1;f)107

719 Dp(1;f)118 In(1)$sc^{8}$, Df(0+ac). $w^{a}$ $sc.^{8}$; Dp(1;f)118

720 Dp(1;f)135 In(1)$sc^{8}$, Df(0+ac). $w^{a}$ $sc.^{8}$; Dp(1;f)135, $y^{2}$

721 Dp(1;f)$X^{c2}$ Dp(1;f)$X^{c2}$/y 1(1)7/y 1 (1)7

722 Dp(1;f)$z^{9}$ Dp(1;f)$z^{9}$, Df(1)$sc^{J4R}$/y f:=

723 Dp(1;1)112 y f, Dp(1;1)112 (homozygous stock)

724 Dp(1;$Y^{L}$)$sc^{S1}$ $sc^{S1}$ .$Y^{L}$/y.$Y^{S}$; y f:=; cn bw; (e/+)

725 Dp(1;3)126 v f; Dp(1;3)126/Payne, Dfd ca

* Dp(1;3)$w^{Vco}$ . . . . . 694

726 Dp(1;3)$sc^{J4}$ Dp(1;3)$sc^{J4}$/Df(1)$sc^{8}$, $w^{a}$

727 Dp(2;2)S Dp(2;2)S, (S ast) (S $ast^{4}$) net dp cl/Cy, En-S

* Dp(2;3)P . . . . . 661, 706

728 Qn(1)w Qn(1)w, $(w)_{5}$/y f:=

729 Qn(2;2)S Qn(2;2)S, $(ast)_{5}$, al ho/Cy, $S^{2}$ En-S

Inversions

Inversions-X

730 In(1)AB In(1)AB/y f:=

* In(1)AM . . . . . .12, 67, etc.

731 In(1)$B^{M1}$ In(1)$B^{M1}$, v $B^{M1}$(tan-like); see also 740, 741, etc.

732 In(1)$B^{M2}$ In(1)$B^{M2}$, $v^{rv}$ $B^{M2}$

733 In(1)$B^{M2}$ In(1)$B^{M2}$(rv) $f^{B15}$ (reinv.; mosaic)

734 In(1)$B^{M2}$ In(1)$B^{M2}$, $f^{B27}$ $B^{M2}$/ClB

* In(1)bb . . . . . 683, 684

| | | |
|---|---|---|
| * | In(1)ClB | In(1)Cl, sc $t^2$ v sl B (=ClB). .72, 676, etc. |
| * | In(1)ClB$^{36d}$ | In(1)Cl, sc $t^2$ v sl B$^{36d}$(=ClB$^{36d}$). . 802 |
| 735 | In(1)dl-49 | In(1)dl-49, ty-1 |
| 736 | In(1)dl-49 | In(1)dl-49, ty-1 $bb^1$/ y v f car |
| 737 | In(1)dl-49 | In(1)dl-49, $v^{Of}$ f |
| 738 | In(1)dl-49 | In(1)dl-49, y $fa^n$ |
| * | In(1)dl-49 | In(1)dl-49, y Hw $m^2$ $g^4$ . . . . . 752 |
| * | In(1)dl-49 | In(1)dl-49, y Hw $m^2$ $g^4$ $f^5$ . . . . . 677 |
| 739 | In(1)dl-49 | In(1)dl-49, y Su-Hw Hw $m^2$ $g^4$/y w f; (nub/+) |
| 740 | Ins(1)dl-49, $B^{M1}$ | Ins(1)dl-49, $B^{M1}$, l(1) J1 $sc^{J1}$ oc ptg $B^{M1}$/ In(1)$sc^{S1L}$ $sc^{8R}$, y $sc^{S1}$ $sc^8$ pn w ec rb cm $ct^6$ $sn^3$ $ras^2$ $g^2$ f sy od car 1/1(1)$J1^+$. Y (= "Maxy") |
| 741 | Ins(1)dl-49, $B^{M1}$ | Ins(1)dl-40, $B^{M1}$, sc v $B^{M1}$ (homozygous) |
| 742 | Ins(1)dl-49, $B^{M1}$ | Ins(1)dl-49, $B^{M1}$, y sc v cu-X $B^{M1}$ |
| 743 | In(1)en-bx | In(1)en-bx, en-bx/y f:= |
| * | Ins(1)FM1 | Ins(1)FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B (=FM1) . . 16, 25, etc. |
| * | Ins(1)FM3 | Ins(1)FM3, $y^{31d}$ $sc^8$ cm B 1 (=FM3) . . . 6, 47, etc. |
| * | Ins(1)FM4 | Ins(1)FM4, $y^{31d}$ $sc^8$ dm B (=FM4).74, 673, etc. |
| 744 | Ins(1)FM6 | Ins(1)FM6, $y^{31d}$ $sc^8$ dm B/y f:= |
| * | Ins(1)FMA3 | Ins(1)FMA3, $y^2$ (=FMA3) . . . 645, 647, etc. |
| 745 | In(1)N$^{264-84}$ | In(1)N$^{264-84}$, y/FM6, $y^{31d}$ $sc^8$ dm B |
| 746 | In(1)$rst^3$ | In(1)$rst^3$, $rst^3$ (homozygous) |
| 747 | In(1)$rst^3$ | In(1)$rst^3$ car bb |
| 748 | In(1)$sc^4$ | In(1)$sc^4$, y $sc^4$ |
| 749 | Ins(1)$sc^{4L}$, $sc^{8R}$ | Ins(1)$sc^{4L}$ $sc^{8R}$, y; see also 669 |
| 750 | In(1)$sc^7$ | In(1)$sc^7$, $sc^7$ |
| 751 | In(1)$sc^7$ | In(1)$sc^7$, $sc^7$ $w^a$ |
| 752 | Ins(1)$sc^7$, AM | Ins(1)$sc^7$, AM, $sc^7$/dl-49, y Hw $m^2$ $g^4$ |
| 753 | Ins(1)$sc^7$, AM | Ins(1)$sc^7$, AM, $sc^7$ car/ FM4, $y^{31d}$ $sc^8$ dm (without B) |
| 754 | Ins(1)$sc^7$, $B^{M1}$ | Ins(1)$sc^7$, $B^{M1}$, $sc^7$ $w^{43b}$ $B^{M1}$/y f:= |
| 755 | In(1)$sc^8$ | In(1)$sc^8$, $sc^8$ |
| 756 | In(1)$sc^8$ | In(1)$sc^8$, $sc^8$ cv v f/y f:= |
| 757 | In(1)$sc^8$ | In(1)$sc^8$, $y^{31d}$ $sc^8$ $w^a$ |
| 758 | Ins(1)$sc^8$, dl-49 | Ins(1)$sc^8$, dl-49, $y^{31d}$ $sc^8$ (homozygous) |
| * | Ins(1)$sc^8$, dl-49 | Ins(1)$sc^8$, dl-49, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B (= FM1) |
| 759 | In(1)$sc^9$ | In(1)$sc^9$, $sc^9$ Bx f t $w^a$ (homozygous) |
| 760 | In(1)$sc^{260-14}$ | In(1)$sc^{260-14}$, $sc^{260-14}$ |
| 761 | In(1)$sc^{260-22}$ | In(1)$sc^{260-22}$, $sc^{260-22}$ |
| 762 | In(1)$sc^{J1}$ | In(1)$sc^{J1}$; Del(1)24 |
| * | Ins(1)$sc^{S1}$, dl-49 | Ins(1)$sc^{S1}$, dl-49, y v B . . . . . 170 |
| * | Ins(1)$sc^{S1L}$, $sc^{8R}$ | Ins(1)$sc^{S1L}$, $sc^{8R}$, y $sc^{S1}$ $sc^8$ pn w ec rb cm $ct^6$ $sn^3$ $ras^2$ $g^2$ f sy od car 1 . . 740 |
| 763 | Ins(1)$sc^{S1L}$, S, $sc^{8R}$ | Ins(1)$sc^{S1L}$, S, $sc^{8R}$, $sc^{S1}$ $sc^8$ $w^a$ B (=Muller-5) |
| 764 | In(1)$w^{m4}$ | In(1)$w^{m4}$ (bb?) |
| 765 | In(1)$y^{3P}$ | In(1)$y^{3P}$ B |
| 766 | Ins(1)$y^{3PL}$, S $sc^{S1R}$ | Ins(1)$y^{3PL}$, S, $sc^{S1R}$/y f:= ; Cy/$sc^{19i}$ |
| 767 | In(1)$y^4$ | In(1)$y^4$, $y^4$ |
| * | In(1)$y^4$ | In(1)$y^4$, $y^4$ $w^a$ sn . . . . . 801 |

Inversions-2

| | | |
|---|---|---|
| 768 | In(2)$bw^{VDe1}$ | b In(2)$bw^{De1}$/b lt l cn mi sp |
| 769 | In(2)$bw^{VDe2}$ | In(2)$bw^{VDe2}$/Rev l |

2L Inversions

| | | |
|---|---|---|
| 770 | In(2L)Cy | In(2L)Cy, $al^2$ $ast^3$ b pr (does not carry Cy mutant) |
| * | In(2L)Cy | In(2L)Cy, Cy $dp^2$ b pr . . . . . 281, 340 |
| * | In(2L)Cyt | In(2L)Cyt, Su-S $dp^2$ pr . . . . . 280 |
| * | In(2L)NS | . . . . . 305 |
| 771 | In(2L)t | In(2L)t, esc c sp/SM5, $al^2$ Cy $lt^v$ $sp^2$ |
| 772 | In(2L)t | In(2L)t, lt l $L^4$ $sp^2$/ Pm, $ds^{33k}$ |
| * | In(2L)t | In(2L)t, l(2)R. . 397 |
| * | In(2L)t | Ins(2L)t, Roi.(2R)Cy, bw $sp^2$ or (= Roi) . . . . . 425 |
| 773 | In(2L)Tg | In(2L)Tg, Tg/SM5, $al^2$ Cy $lt^v$ $sp^2$ |

2L + 2R Inversions

| | | |
|---|---|---|
| 774 | Ins(2L+2R)Cy | Ins(2L+2R)Cy, $al^2$ En-S $cn^2$ $sp^2$ (does not carry Cy mutant) |

| | | |
|---|---|---|
| * | Ins(2L+2R)Cy | Ins(2L+2R)Cy, $al^2$ Cy $lt^3$ $L^4$ $sp^2$ . . . . 279, 815, 836 |
| * | Ins(2L+2R)Cy, $bw^{V34}$ | Ins(2L+2R)Cy, Cy, (2R) $bw^{V34}$. . . . . 337 |
| * | Ins(2L+2R)Cy | Ins(2L+2R)Cy, Cy, $dp^{Th}$ pr . . . . . 792 |
| * | Ins(2L+2R)Cy | Ins(2L+2R)Cy, Cy . . . .262, 641, 652 |
| * | | Ins(2L+2R)Cy, Cy $bw^{45a}$ $sp^2$ or $45^a$ . . . 231 |
| * | | Ins(2L+2R)Cy, Cy $dp^2$ . . . . . 292 |
| * | | Ins(2L+2R)Cy, Cy $dp^{Th}$ Bl $L^4$ $sp^2$. . . . 196 |
| * | | Ins(2L+2R)Cy, Cy En-S . . . 381, 698, etc. |
| * | | Ins(2L+2R)Cy, Cy $hk^2$ . . . . . 197 |
| * | | Ins(2L+2R)Cy, Cy $L^4$ $sp^2$ . . . . . 819 |
| * | | Ins(2L+2R)Cy, Cy pr . . . .411, 835, etc. |
| * | | Ins(2L+2R)Cy, Cy $sp^2$ . . . . . 660 |
| * | | Ins(2L+2R)Cy, Cy $S^2$ En-S . . . . . 729 |
| * | | Ins(2L+2R)Cy, Cy $S^2$ $dp^2$ En-S . . . . 323 |
| 775 | Ins(2L+2R)NS | Ins(2L+2R)NS, b mr/Cy |
| * | Ins(2L+2R)NS | Ins(2L+2R)NS, px sp . . . . . 696 |
| * | Cy-RNS | Ins(2L)Cy, Cy $dp^2$ pr. (2R)NS, 1 px 1(2)NS sp . . . . . 349 |

## 2LR Inversions

| | | |
|---|---|---|
| 776 | In(2LR)102 | In(2LR)102, $ds^W$/SM1, $al^2$ Cy $sp^2$ |
| * | In(2LR)dp | . . . . . 232 |
| * | In(2LR)Gla | . . . (= Gla) . . 221 |
| * | In(2LR)Pm | In(2LR)Pm, $ds^{33k}$ . . . 316, 342, etc. |
| * | $In(2LR)Pm^2$ | . . . . . 341 |
| * | In(2LR)Rev | . . . (= Rev) . . 769 |
| * | In(2LR)Rvd | . . . . . 712 |
| * | Ins(2LR)SM1 | Ins(2LR)SM1, $al^2$ Cy $sp^2$ . . (=SM1). . . 204, 206, etc. |
| * | Ins(2LR)SM5 | Ins(2LR)SM5, $al^2$ Cy $lt^v$ $sp^2$ . . (=SM5). 198, 202, etc. |
| * | Ins(2LR)U | . . . . (= U) . . 247 |

## 2R Inversions

| | | |
|---|---|---|
| * | In(2R)Cy | In(2R)Cy, $cn^2$ Bld . . . . . 343 |
| * | $In(2R)Mo^K$ | . . . . . 818 |
| * | In(2R)NS | . . . . . 349 |

## 3L Inversions

| | | |
|---|---|---|
| * | In(3L)D | . (= D) . 502, 652, etc. |
| * | $In(3L)D^3$ | . (= $D^3$). . . 460, 507 |
| * | In(3L)P | In(3L)P, gm . 537, 599 |
| * | In(3L)P | In(3L)P, Mé . (= Mé) . . . .517, 519, etc. |
| 777 | In(3L)P | In(3L)P, mot-36e/R |

## 3LR Inversions

| | | |
|---|---|---|
| * | In(3LR)Cx | .(= Cx). 462, 479, etc. |
| * | Ins(3LR)Cx, D | . . . . . 502 |
| 778 | In(3LR)sep | ri $p^P$, In(3LR)sep, sep |
| * | Ins(3LR)TM1 | Ins(3LR)TM1, Mé ri $sbd^1$ (=TM1). . . . . 439 |
| * | Ins(3LR)TM3 | Ins(3LR)TM3, $y^+$ $ac^+$ ri $p^P$ sep $bx^{34e}$ $e^s$ (=TM3). . . 534, 833 |
| * | $In(3LR)Ubx^{101}$ | . . . . . 556 |
| * | $In(3LR)Ubx^{130}$ | In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^s$ (= $Ubx^{130}$). . . . 467, 504, etc. |

## 3L + 3R Inversions

| | | |
|---|---|---|
| * | Ins(3L+3R)P | Ins(3L+3R)P, 1(3)PL 1(3)PR (= Payne) . . . .460, 473, etc. |
| * | | Payne, Dfd ca . . . .469, 470, etc. |
| * | Ins(3L+3R)P | .(= LVM).487, 503, 508 |
| * | Ins(3L)P, (3R)C | In(3L)P, Mé, In(3R)C, Sb e 1(3)e. . . 541 |

## 3R Inversions

| | | |
|---|---|---|
| 780 | $In(3R)Antp^B$ | In(3R)$Antp^B$, $Antp^B$/ TM1, Mé ri $sbd^1$ |
| * | In(3R)C | In(3R)C, cd . . . 557 |
| | | In(3R)C, e. . . . 450 |
| | | In(3R)C, e 1(3)e . . . .438, 509, etc. |
| | | In(3R)C, 1(3)a . . . .437, 466, etc. |
| | | In(3R)C, Sb e 1(3)e . . . .471, 494, etc. |
| * | In(3R)Cyd | In(3R)Cyd, Cyd . .(=Cyd) . . . 472 |
| 781 | $In(3R)D1^B$ | In(3R)$D1^B$, st $D1^B$/In (3R)$P^W$, st 1(3)W ca |
| * | In(3R)hp | In(3R)hp, hp. . . 491 |
| 782 | In(3R)Hu | In(3R)Hu, Hu $Sb^{Spi}$/ Payne |
| 783 | In(3R)Mo | In(3R)Mo, sr/Xa ca |
| * | In(3R)P | . . . . . 784 |
| 784 | $In(3R)P^{Fla}$ | In(3R)$P^{Fla}$ (homozygous) |

| | | |
|---|---|---|
| * | In(3R)$P^W$ | In(3R)$P^W$, st 1(3)W ca . . . . . 581, 781 |
| * | In(3R)spr, spr | . . . . . 464 |

Translocations-1;Y

| | | |
|---|---|---|
| 785 | T(1;Y)1E | T(1;Y)1E, y/y f:= ; cn bw |
| 786 | T(1;Y)2E | T(1;Y)2E/v car 1 (Stern #64)/y f;= ; cn bw |

Translocations-1;2

| | | |
|---|---|---|
| 787 | T(1;2)Bld | T(1;2)Bld, Bld/ClB (carries In(2R)Cy) |
| 788 | T(1;2)$f^{257-5}$ | T(1;2)$f^{257-5}$/In(1)AM |
| 789 | T(1;2)lt | T(1;2)lt/Cy (carries eq and possibly $su^3$-s) |
| 790 | T(1;2)$N^{264-10}$ | T(1;2)$N^{264-10}$/FM6, $y^{31d}$ $sc^8$ dm B |
| 791 | T(1;2)$sc^{S2}$ | T(1;2)$sc^{S2}$/Cy |
| 792 | T(1;2)$sc^{19}$ | T(1;2)$sc^{19}$/y f:= ; fes $sc^{19i}$ b pr/Cy, $dp^{Th}$ pr |

Translocations-1;3

| | | |
|---|---|---|
| 793 | T(1;3)263-4 | T(1;3)263-4, y sc $B^1$/ In(1)AM |
| 794 | T(1;3)"Del 143" | T(1;3)"Del 143" ru $e^s$ ca/CxF, ru h ca |
| 795 | T(1;3)$N^{264-6}$ | T(1;3)$N^{264-6}$, y/y w dm (=$N^6$) |
| 796 | T(1;3)04 | T(1;3)04/ClB |
| 797 | T(1;3)05 | T(1;3)05, D/y f:= |
| 798 | T(1;3)$ras^V$ | T(1;3)$ras^V$/y f:= |
| * | T(1;3)$sc^{J4}$ | . . . . . 807 |
| 799 | T(1;3)$sc^{260-15}$ | T(1;3)$sc^{260-15}$/FM6, $y^{31d}$ $sc^8$ dm B |
| 800 | T(1;3)v | T(1;3)v, v/FM6, $y^{31d}$ $sc^8$ dm B |
| 801 | T(1;3)v | T(1;3)v, sc cv v f/In (1)$y^4$, $y^4$ $w^a$ sn |
| 802 | T(1;3)$w^{Vco}$ | T(1;3)$w^{Vco}$, v f/$ClB^{36d}$ |

Translocations-1;4

| | | |
|---|---|---|
| 803 | T(1;4)$B^S$ | T(1;4)$B^S$/y f:= |
| 804 | T(1;4)$N^{8a}$ | T(1;4)$N^{8a}$/FM6, $y^{31d}$ $sc^8$ dm B |
| 805 | T(1;4)$sc^8$ | T(1;4)$sc^8$, B $w^a$/y f:= |
| 806 | T(1;4)$w^{m5}$ | T(1;4)$w^{m5}$/$ey^D$ |
| 807 | T(1;4)$w^{m5}$(1;3) $sc^{J4}$ | T(1;4)$w^{m5L}$; T(1;3)$sc^{J4R}$ (ClB) |
| 808 | T(1;4)$w^{258-18}$ | T(1;4)$w^{258-18}$, y/$ci^D$ |
| 809 | T(1;4)$w^{VD3}$ (=$w^{258-21}$) | T(1;4)$w^{VD3}$/FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B |
| 810 | T(1;4)$w^{258-21}$, y $w^a$ | T(1;4)$w^{258-21}$, y $w^a$/FM4, $y^{31d}$ $sc^8$ dm B |

Translocations-Y;2

| | | |
|---|---|---|
| * | T(Y;2)A | . . . . . 324 |
| 811 | T(Y;2)B | T(Y;2)B/b |
| * | T(Y;2)C | . . . . . 259, 361 |
| * | T(Y;2)E | . . . . . 195, 255 |
| * | T(Y;2)G | . . . . . 228 |
| * | T(Y;2)J | . . . . . 369 |
| 812 | T(Y;2)rl | T(Y;2)rl, lt cn/b lt bw |

Translocations-Y;2;3

| | | |
|---|---|---|
| * | T(Y;2;3)F | . . . . . 542 |

Translocations-2;3

| | | |
|---|---|---|
| 813 | T(2;3)101 | al T(2;3)101 $sp^2$/Cy, $L^4$ $sp^2$ |
| 814 | T(2;3)101 | ru h T(2;3)101 $e^4$ ro ca/Payne, Dfd ca |
| 815 | T(2;3)108 | al T(2;3)108 c $sp^2$/Cy, $al^2$ $lt^3$ $L^4$ $sp^2$ |
| 816 | T(2;3)109 | T(2;3)109 $p^P$/Payne, Dfd ca |
| 817 | T(2;3)A | Bl T(2;3)A; ru h D TA ss $e^s$/Payne |
| 818 | T(2;3)At | T(2;3)At, At/In(2R)$Mo^K$ |
| 819 | T(2;3)B | al T(2;3)B $sp^2$/Cy, $L^4$ $sp^2$ |
| 820 | T(2;3)B | T(2;3)B; ru h D TB ss $e^s$/Payne |
| 821 | T(2;3)$bw^{V4}$ | T(2;3)$bw^{V4}$/SM1, $al^2$ Cy $sp^2$ |
| 822 | T(2;3)$bw^{V5}$ | T(2;3)$bw^{V5}$/SM5, $al^2$ Cy $lt^V$ $sp^2$ |
| 823 | T(2;3)$bw^{VDe3}$ | T(2;3)$bw^{VDe3}$, Ubx bxd/ In(3LR)Cx |
| 824 | T(2;3)$bw^{VDe4}$ | T(2;3)$bw^{VDe4}$/SM5, $al^2$ Cy $lt^V$ $sp^2$ |
| 825 | T(2;3)C | T(2;3)C; ru h D TC ss $e^s$/Payne |
| 826 | T(2;3)E | T(2;3)E/SM5, $al^2$ Cy $lt^V$ $sp^2$ |
| 827 | T(2;3)Hn | T(2;3)Hn, Hn/In(3LR) $Ubx^{130}$, $Ubx^{130}$ $e^s$ |
| * | T(2;3)Mé | . . . . .514, 515, etc. |
| * | T(2;3)P | T(2;3)P, P. . 661, 706 |
| 828 | T(2;3)$p^{Gr}$ | T(2;3)$p^{Gr}$/SM1, $al^2$ Cy $sp^2$ |
| 829 | T(2;3)$Pu^4$ | T(2;3)$Pu^4$, $Pu^4$/C(3)x |
| 830 | T(2;3)rn | T(2;3)rn/SM1, $al^2$ Cy $sp^2$ |
| 831 | T(2;3)Dp-s | T(2;3)Dp-s, ho/Cy, En-S (hom. viable) |
| 832 | T(2;3)$S^L$ | T(2;3)$S^L$/Cy, En-S |
| * | T(2;3)$S^M$ | . . . . . 424 |
| 833 | T(2;3)$Sb^V$ | T(2;3)$Sb^V$, $Sb^V$/TM3, $y^+$ $ac^+$ ri $p^p$ sep $bx^{34e}$ $e^s$ |

| | | |
|---|---|---|
| * | T(2;3)Xa | .(= Xa) 442, 523, etc. |
| 834 | T(2;3)Xa | T(2;3)Xa/1(3)Xa R |

Translocations-2;4

| | | |
|---|---|---|
| 835 | T(2;4)a | T(2;4)a/Cy, pr; $ey^2$ |
| 836 | T(2;4)$ast^v$ | T(2;4)$ast^v$/Cy, $al^2$ $lt^3$ $L^4$ $sp^2$ |
| 837 | T(2;4)b | T(2;4)b/Cy, pr; $ey^2$ |
| 838 | T(2;4)d | al dp T(2;4)d px sp/Cy, pr; $ey^2$ |
| 839 | T(2;4)d | T(2;4)d/Cy, pr |

Translocations-3;4

| | | |
|---|---|---|
| 840 | T(3;4)a | D, T(3;4)a/Mé |
| 841 | T(3;4)A2 | T(3;4)A2/Mé, ca |
| 842 | T(3;4)A12 | T(3;4)A12/Cx, D |
| 843 | T(3;4)A13 | T(3;4)A13, ve ca/Mé, ca |
| 844 | T(3;4)A28 | T(3;4)A28, ve ca (homozygous) |
| 845 | T(3;4)c | T(3;4)c/Payne, Dfd ca |
| 846 | T(3;4)e | T(3;4)e/$Ubx^{130}$ |
| 847 | T(3;4)e | h th st, T(3;4)e, cu sr $e^s$ ca/Payne, Dfd ca |
| 848 | T(3;4)f | T(3;4)f/Mé |
| 849 | T(3;4)f | T(3;4)f, h th st cu sr $e^s$ ca/Payne, Dfd ca |

Transpositions

| | | |
|---|---|---|
| 850 | Tp(3)$bxd^{100}$ | Tp(3)$bxd^{100}$ ri/T(2;3)Mé |
| 851 | Tp(3)$sbd^{104}$ | Tp(3)$sbd^{104}$/$Ubx^{130}$ |
| 852 | Tp(3)$bxd^{107}$ | Tp(3)$bxd^{107}$, bx $bxd^{107}$ sr $e^s$/$bx^{34e}$ Mc |
| 853 | Tp(3)Vno | Tp(3)Vno/$H^2$ |

Biochemical Mutants

| | |
|---|---|
| 854 | y w spl sn (alaful-1)/M-5, $bb^1$ |
| 855 | y w spl sn (gluful-1)/M-5, $bb^1$ |
| 856 | y w spl sn bb (gluful-2)/M-5, $bb^1$ |
| 857 | y w spl sn bb (gluful-3)/M-5, $bb^1$ |
| 858 | y w spl sn (gluful-4)/M-5, $bb^1$ |
| 859 | y w spl sn bb (glufulproless-1)/M-5, $bb^1$ |
| 860 | y w spl sn (glufultyrless-1)/M-5, $bb^1$ |
| 861 | y B (glufultyrless-2)/M-5, $bb^1$ |
| 862 | y w spl sn bb (glufultyrless-3)/M-5, $bb^1$ |
| 863 | y w spl sn bb (glyful-1)/M-5, $bb^1$ |
| 864 | y w spl sn bb (glufultyrproless)/M-5, $bb^1$ |
| 865 | y w spl sn bb (phenylfultyrless-1)/M-5, $bb^1$ |
| 866 | y w spl sn bb (tyrless-1)/Ins(1)d1-49, $sc^{S1}$ oc ptg B |
| 867 | y w spl sn (tyrless-2)/M-5, $bb^1$ |
| 868 | y w spl sn (tyrproless-1)/M-5, $bb^1$ |
| 869 | y w spl sn bb (tyrproless-2)/M-5, $bb^1$ |
| 870 | y w spl sn bb (tyrproless-3)/M-5, $bb^1$ |

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### Department of Zoology

Chromosome 2

| | |
|---|---|
| 89 | b + + + |
| 90 | b pr + + |
| 91 | b pr stw + |
| 92 | b + + c |
| 93 | b + + sp |
| 94 | b pr + + sp |
| 95 | b pr stw + sp |
| 96 | b + + c sp |
| 97 | + + + c |
| 98 | + + + c sp |
| 99 | + pr stw + |
| 100 | + pr stw c |
| 101 | + pr stw c sp |
| 102 | + pr stw + sp |
| 103 | + + stw c |
| 104 | + + stw c sp |

Multichromosomal

| | |
|---|---|
| 105 | b + + +;$cd^{59}$ |
| 106 | b pr + +;$cd^{59}$ |
| 107 | b pr stw +;$cd^{59}$ |
| 108 | b + + c;$cd^{59}$ |
| 109 | b pr + + sp;$cd^{59}$ |
| 110 | b pr stw + sp;$cd^{59}$ |
| 111 | b + + c sp;$cd^{59}$ |
| 112 | + + + c;$cd^{59}$ |
| 113 | + + + c sp;$cd^{59}$ |
| 114 | + pr stw +;$cd^{59}$ |
| 115 | + pr stw + sp;$cd^{59}$ |
| 116 | + pr stw c;$cd^{59}$ |
| 117 | + pr stw c sp;$cd^{59}$ |
| 118 | + + stw c;$cd^{59}$ |
| 119 | + + stw c sp;$cd^{59}$ |

Deletions

Chromosome 1: 5

Chromosome 2: 10, 11, 12, 13, 15, 16, 17, 18, 19, 20, 21, 22, 24, 26, 28, 29, 30, 31, 32, 33, 34, 35, 36, 39, 40, 41, 43

Chromosome 3: 44, 46

Multichromosomal: 53

## COLLEGE PARK, MARYLAND: UNIVERSITY OF MARYLAND
### Department of Zoology

### Wild Stocks

This series of lines was originally derived from a single sampling near State College, Pennsylvania in the spring of 1955. 200 inseminated females were obtained, their progeny replicated three times, and raised at three temperatures 18°C, 22°C and 26°C (600 lines total). These temperature conditions were maintained until the winter of 1962 when all lines were raised at 25°C. Some of these lines are the source of the lxd gene.

18 lines remain:

| 18°C lines | | 22°C lines | | 26°C lines | |
|---|---|---|---|---|---|
| A2I | (18)° | A6IV | (22)° | A3V | (26)° |
| A2II | (18)° | A6X | (22)° | A3IV | (26)° |
| A3I | (18)° | AI8X | (22)° | A6I | (26)° |
| A3IX | (18)° | | | A6X | (26)° |
| A3X | (18)° | | | A17VI | (26)° |
| A6V | (18)° | | | A17VIII | (26)° |
| A6IX | (18)° | | | A2III | (26)° |
| | | | | A21IV | (26)° |

## BALTIMORE, MARYLAND: THE JOHNS HOPKINS UNIVERSITY

### Wild Stocks

b1 Amherst-34
b2 Canton-S
b3 Crimea
b4 Florida (inbred)
b5 Formosa
b6 Kyoto, Japan
b7 Lausanne-S
b8 Oregon-R
b9 Salta, Argentina
b10 Seto, Japan
b11 St. Louis-7
b12 Stephensville
b13 Swedish-b
b14 Tuscaloosa, Alabama
b15 Urbana-S
b15a Varese, Italy
b16 Woodbury, New Jersey

### Chromosome 1

c1 br $w^e$ ec rb $t^4$/Ins(1)$sc^8$ $In^{49}$, B $lz^s$ $w^a$ $y^{31d}$
c2 ec $ct^6$(s)car/ClB
c3 f B
c4 f/y
c4a $g^{53d}$
c5 lz/y
c6 sc cv dx v f
c7 sc $t^2$ v f Tu car/y f:=
c7a $v^1$ (suppressable)
c8 $w$
c8a $w^a$
c9 w m f
c9b $y^2$ $cho^2$
c10 y $ct^6$ $ras^2$ f
c10a y $sn^3$ $v^{36f}$ ($v^{36f}$- unsuppressed)
c10b $y^2$ $su^{51}c^{15}$ $ras^2$ $v^1$ f ($v^1$ - suppressed)

### Duplications

c11 Dp(1)$sc^{s1}$, y w f

### Inversions

c12 In(1)$In^{49}$, y $fa^n$
c14 Ins(1)$sc^4$ Ins $sc^{s1}$, y
c17 Ins(1)$sc^{s1}$ $sc^8$, B $w^a$
c18 Ins(1)$sc^{s1}$ $In^{49}$, B f v y/y f:=

### Lethals

c21 car $1^{(C1+1)}$/Ins(1) $sc^{s1}$ $sc^8$, B $w^a$
c22 car $1^{(B2+6)}$/Ins(1) $sc^{s1}$ $sc^8$, B $w^a$
c23 car $1^{(A3+3)}$/Ins(1) $sc^{s1}$ $sc^8$, B $w^a$

### Closed X's

c28 $X^{c1?}$ y/y f:=
c29 $X^{c2}$/y f:=
c30 $X^{c2}$, y v

### Chromosome 2

d1 a1 b c $sp^2$
d2 a1 dp b pr cn c px sp/Cy sp
d2a a1 dp b pr cn c px sp/Cy pr cn sp
d3 a1 Sp b $L^{34}$/Cy
d4 $ap^{(49j)}$/Cy
d4a b pr cn
d4b b Tft vg/b vg
d5 b vg
d6 B1 L/Cy
d6a B1 $L^2$/SM5, $a1^2$ Cy $1t^v$ $sp^2$
d7 bw
d7a cn Su-Pm/Cy cn vg Pm
d7b cn Su-Pm Tac/Pm (dp b c?)
d7c c px sp
d8 $dp^{02}$
d9 $L^2$
d10 M/Cy

d10a mi/Pm$^2$
d11 net S ho/Cy E-S
d11a pys
d11b Pfd/Ins(2L,2R)Cy S$^2$
d11c px s1t sp
d12 rn/Cy
d13 S Sp B1 L/Cy cn$^2$ sp
d14 stw$^2$/Cy
d14a Tac sp/Cy sp
d15 Tft/Cy sp
d15a Tft/Cy$^+$ sp

Deficiencies

d16 Df(2)bw$^5$/Cy sp
d17 Df(2)dp$^{v51}$/Cy

Inversions

d18 In(2R)bw$^A$/Cy
d19 Ins(2L,2R)Cy bw$^{V2}$/a1 dp b pr cn c px sp
d20 In(2LR)bw$^{V29}$/Cy
d21 In(2LR)bw$^{V30k1}$/Cy
d22 In(2R)bw$^{V30k10}$/Cy
d23 Ins(2L,2R)Cy bw$^{V34}$/b vg
d24 In(2)b bw$^{VDe\ 1}$/b 1t l cn mi sp
d25 In(2)bw$^{VDe\ 2}$/Rev 1
d26 In(2)bw$^{V13}$/Cy

Chromosome 3

e1 bar-3
e1a e$^{11}$
e2 G1 bx$^D$/Inv LVM
e2a G1 Sb/LVM
e3 Ly Sb/Inv LVM
e3b Ly Sb H/Inv LVM
e4 M(3)y G1/Inv LVM
e5 M(3)y Sb/Inv LVM
e6 p ss bx/T(2;3)Xa
e6a red
e6b ri p$^p$
e7 ru h th st cu sr e$^s$ ca
e8 Sb bx$^D$/T(2;3)Xa
e9 se
e10 se e
e10a se ss
e10b ss
e11 st$^{brk}$
e11a st

Chromosome 4

f1 Ce/ci ey$^R$
f2 spa
f3 sv$^n$
f4 bt ey$^R$ sv$^n$

Multichromosomal

g1 b(Su-er)$^+$bw; st er
g1a Swedish-b erupt
g1c b(Su-er)$^+$ Tft bw; st er
g1e cn bw; e
g1f ct$^{45e}$ v;bw;e;(ey$^R$)$^+$
g2 Cy/Pm ds$^{33k}$;H/Sb-C
g3 Cy pr cn/Pm ds$^{33k}$; H/Sb-C
g3a Cy/Pm; st er Su-tu
g3b Cy sp/a1 dp b pr cn c px sp;ci ey$^R$
g3c Cy/tu bw; st su-tu
g4 dp;e
g4a net;ru by
g5 pr cn;by
g6 pr cn;by;ci ey$^R$
g6a SM1, a1$^2$ Cy sp$^2$/Pm;Ubx/Sb
g7 Su-er tu bw;st er su-tu
g8a tu bw;er$^+$(su-tu)$^+$
g8c tu bw;Sb bx$^D$/T(2;3)Xa
g8d v$^1$/y;Su-er bw;st er (v$^1$- suppressable)
g8e y sn$^3$v$^{36f}$/y;Su-er bw;st er (v$^{36f}$-unsuppressed)
g8f tu-55G$^{JACOBS}$;st su-tu
g9 y;bw;e;ci ey$^R$
g9a v;bw;e
g9b y$^2$ v f;bw

Aberrations

g10 v;In(2R)bw$^{V2}$/v;+
g12 T(2;3)bw$^{V5}$ st/st
g13 T(2;3)bw$^{V5}$ st/T(2;3) p$^{Gr}$ st
g14 T(2;3)bw$^{VDe4}$/Cy
g15 T(2;3)Mé/ru h th st cu sr e$^s$ Pr ca
g16 T(2;3)p$^{Gr}$/Cy
g17 T(2;3)rn/Cy
g18 T(2;3;4)bw$^{V30k18}$ Ins (2LR)/Cy
g23 T(2;3)Sp;Dl Pr/pr cn; by
g24 T(2;4)/pr cn;ci ey$^R$

Tumor stocks

tu A$_2$ cito-p1-st
tu B$_3$ (Italy)
tu-55G$^{Jacobs}$
g8a see Multichromosomal listing

## MADISON, WISCONSIN: UNIVERSITY OF WISCONSIN
### Department of Zoology

The stock list remains essentially the same as that appearing in DIS 40:12 with the following changes:

Stocks Removed from List

33 ("Tester-1")y ac pn w rb wy$^2$ g$^2$ & y f:=;sc$^{191}$/Cy
34 ("Tester-2")y$^2$ w$^a$ cm wy$^2$ g$^2$ car & y f:=;sc$^{191}$/Cy
35 ("Tester-3")y rb cm ras$^2$ g$^2$ & y f:=; sc$^{191}$/Cy
43 ("sz 1z f")Y$^{Lc}$/1z$^3$ m f•Y$^S$ &$_6$y v f•=
45 ("sz y w")Y$^{Lc}$/y w•Y$^S$ & Y ct$^6$ f•=
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$^s$

Corrections

12 v m g & y f:=
15 sc$^{8}$•Y/y & y f:=
19 X$^{c2}$ y v bb$^-$/sc$^8$•Y$_D$& y$_{txI}$ f:=
73 S Sp cn$_2$M(2)S7 bw$^D$/dp$^{txI}$ Cy, InsO pr cn$^2$ sp

Additions

76 S Sp cn bw;dp$^{txI}$ Cy cn bw

## BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA
Department of Zoology

### Wild stocks

1 Canton-S
3 Samarkand
5 $+^3$
6 Oregon R-C

### Chromosome 1

100 B
101 $bb^1$ $w^e$/ClB(with floating Y)
102 br
103 br ec/$y^{3d}$
104 $Bx^3$
106 cm $ct^6$ $sn^3$
108 $ec_{49c}$
110 $Hw$/FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
111 In(1)dl49, y Hw $m^2$ $g^4$/y f
113 kz $g^2$ B/y
115 Df(1)$N^8$/dl49, y Hw $m^2$ $g^4$
116 sc ec cv $ct^6$ $vg^2$ f/FM3, $y^{31d}$ $sc^8$ dm B
117 sd
118 sple
119 sx $vb^2$ sy/FM4 (extra Y floating)
121 v car
122 $w_a$
123 $w_{bl}$
124 $w_{bl}$
125 $w_{bl}$ ec/FM4
126 $w^{cf}$
127 $w^{cf}$/FM
128 $y^2$ $w^{cf}$/FM4
129 $w^{ch}$ wy
130 $w^{ch}$ wy/FM4
131 $w^{co}$ $sn^2$
132 $w^{co}$/FM4
133 $w^e$
140 y
141 y ac/y
142 y ac $sn^3$ v
143 y ac $sn^3$ B
144 y ac $sn^3$ sx $vb^2$ sy/y $sc^{51}$ B v $w^a$ $sc^8$(dl49)
145 y ac v
150 y ac Dp $w^a$ $(w^a)_2$/$y^2$ sc $w^{56l}$ ec
151 y f:=/ y ac Dp $(w^a)_2$
155 y sc
156 y sc m $f^5$
157 y sc/y $ac^+$ $sc^+$·Y
159 y $sn^3$
160 y w
161 In(1)y In(1)w
165 $y^2$ cv v f
168 y w f:=/$w^a$ spl nd rb, Dp(1;2R)5167
170 M-5/y·
180 $X^{c2}$ f car/y f
181 y w/$w^{vc}$/w(ring)
183 $w^{vc}$/y w $lz^s$dl49/$sc^8$·Y
184 $y^2$ su-$w^a$ $w^a$ $Y^L$·$Y^S$/O x y v bb/O
185 X·$Y^L$ sc cv v f/y/$Y^S$
186 y w bb/X·$Y^S$, y w $Y^S$/$Y^L$·$bb^+$ $ac^+$ $y^+$ ($sc^8$)

### Chromosome 2

200 a px sp
205 al dp b pr c px sp/Cy pr
206 al dp b pr cn vg $ca_2$px bw mr sp/$S^2$ Cy $lt^3$ $pr^+$ Bl cn $L^4$ $sp^2$
208 b
212 bw
214 c
215 cg c/U
216 cl
218 cn bw
220 In(2L)t esc c sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
225 l(2)gl cn bw/Cy $al^2$ $lt^3$ $L^4$ $sp^2$
226 $L^4$
228 pr cn ix/SM5, $al^2$ $lt^v$ Cy $sp^2$
229 pr en
232 vg
233 $vg^{no}$

### Chromosome 3

300 $Antp^{49}$/$Ubx^{130}$
301 cp in ri $p^p$
302 cu
303 cv-c $sbd^2$
308 Gl Sb/LVM
309 gro/Pr ca
310 h
312 Ly/$D^3$
314 $p^p$
315 ru h st $p^p$ ss $e^s$
316 ru h th st cu sr $e^s$ ca
319 se
320 se h
321 se dn Sb/ LVM
322 se Ly dn/LVM
323 $ss_a$
324 $ss^a$-B
325 $ss^{a}$-$B^{d}$
327 $tet^{Bd}$ bilat
328 th st cp
329 th st Pc Scx $p^p$ ss/ TM1, Mé ri
340 In(3LR)TM; Mé/In(3LR)Ubx $e^s$
350 Pc/T(2,3)Mé

Chromosome 4

402 bt $ey^R$ $sv^n$
403 $bt^D/ci^D$
404 ci
405 $ci^w$
408 $ci\ ey^R$
412 $ey^2$
413 ey, ophthalmoptera
414 bubble-eye, eyeless
420 M-4/$ey^D$
421 $sv^n$

Multichromosomal

510 w;vg
511 y f/$X^{c2}$ t;en
512 y ac $sn^3$; $stw^3$ en
514 $sn^3$;cn bw;ri
516 y f;bw;e;ci $ey^R$
517 y;D/tra red
518 y w f:=/$y^{31d}$ $sc^8$ $f^{3n}$ v;Df(4), Dp $y^+$ $ac^+/ey^D$
519 ec;$sv^n$
520 b;$p^p$
521 Cy/Pm;D/Sb
522 vg;se
530 se h;ci $ey^R$
550 3N cm $ct^6$ $sn^4/FM_1$, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
552 3N $y^2$ sc $w^a$ ec/$FM_4$ Y

Translocations

603 T(1;2)Bld/ClB
606 T(1;2)$sc^{19}$/y f:=; fes $sc^{19i}$ b pr/Cy $dp^{th}$ pr
607 Xa/Sb Ubx
608 T(2;3) Met/dp
609 Met/Sb

Multiple inversions

A $sn^3$;Cy/cn bw;ri (Pasadena)
B y;Cy/cn bw;ri (Pasadena)
C $sn^3$;Cy/cn bw;ri (Berkeley)
D y;Cy/cn bw;ri (Berkeley)
H y;Pm/cn bw;ri (Pasadena)
J y;Gla/cn;ri
K $sn^3$;Gla/cn;ri
L y;In $bw^D$/cn bw;ri
M $sn^3$;In(2L)Cy bw/bw;ri

## EMPORIA, KANSAS: KANSAS STATE TEACHERS COLLEGE

Wild stocks

Oregon-R
Canton-S

Chromosome 1

y
$z_{Bwx}$
$w_{crr}$
$w_a$
$w_e$
w
$w_h$
$w_{sat}$
w
$rb_6$
$ct_3$
$sn_{37h}$
$lz^2$
ras
v
m
s
g
f
B
$y_2 ct^6$ $ras_3^2$ f
$y^2$ spl $sn^3$
$su^2$-s v
$z^{11G3}$
w m
w m f
$w^{ch}$ wy
amx lz/y Hw In(1) dl-49 $m^2$ $g^4$
$sc^{S1}$ B Ins $w^a$ $sc^8$

Chromosome 2

dp
ab
b
cn
en
vg
$L^2$
bw
al b c $sp^2$
b vg

Chromosome 3

ve
se
th
st
$e^{11}$
se ss k e ro

Chromosome 4

$ey^2$
pol
$sv^n$
ci $ey^4$

Multichromosomal

v;bw
SM1, Cy/Pm;$Ubx^{130}$/Sb
SM1/102;$Ubx^{130}$/Sb
$y^2$ $w^a$ spl ec;SM1/102;$Ubx^{130}$/Sb
y $w^{crr}$ $sp\text{-}w^1$;SM1;$Ubx^{130}$/Xa
$lx^D$/In(1)dl-49 $m^2$ $g^4$;Cy/Pm

Attached-X

$lz^3$/y f:=
f fu/y f:=
amx $lz^g$ v/y f:=
$y^2$ $w^a$ spl $sn^3$/y f:=
$y^2$ $sp\text{-}w^4$ spl $sn^3$/y f:=

Deficiencies

y $w^-$ $rst^3$/$Y^{303}$
y $w^-$ $rst^3$/y Hw In(1)dl-49 w lz
y $w^{258\text{-}11}$/y Hw In(1)dl-49 $m^2$ $g^4$
$y^2$ $w^{258\text{-}45}$/y Hw In(1)dl-49 $m^2$ $g^4$

Translocations

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

## DETROIT, MICHIGAN: WAYNE STATE UNIVERSITY
### Department of Biology

Wild Stocks

1 Oregon-R
2 Stephenville

Chromosome 1

101 Basc/y f:=
102 B In(1)AB/y f:=
103 $bb^1$ $w^e$/ClB
104 br
105 car
106 dm/y f:=
107 ec
108 f fu/y f:=
109 s
110 $w^{bl}$ ec/FM4
111 $w^{vc}$/y w $lz^s$ dl-49/$sc^8$·Y
112 $y^{3d}$/br ec tu-1
113 $y^{3d}$/y f:=
114 y ac w
115 y w m
116 y $w^a$ cv v m f car/y f:=

Chromosome 2

201 b
202 bw
203 cl
204 corr
205 corr b cn bw
206 cn bw
207 dp
208 $L^2$/Cy $sp^2$
209 vg

Chromosome 3

301 e
302 $e^s$
303 $e^{11}$
304 h th st $p^p$ cu sr $e^s$
305 Ly/$D^3$
306 se

Multichromosomal

401 y f:=;bw;e;$ey^R$
402 $L^2$/Cy $sp^2$;Ly Sb/$D^3$
403 tu-1;tu-3

Attached-X (no free Y)

601 $Y^SX{\cdot}Y^L$, v f B/br ec tu-1

## LEXINGTON, KENTUCKY: UNIVERSITY OF KENTUCKY
### Department of Zoology

Wild Stocks

1 Lexington, Kentucky, wild type.

## COLD SPRING HARBOR, NEW YORK: CARNEGIE INSTITUTION OF WASHINGTON

Note: Stocks are maintained primarily for distribution to students and teachers interested in performing the experiments outlined in Drosophila Guide, by M. Demerec and B. P. Kaufmann, published by Carnegie Institution of Washington (7th edition, 1961; second printing, 1962; third printing, 1964).

### Wild Stocks*

1 Canton-S$^{2}$
2 Oregon-R
3 Oregon-R-EL$^{2}$ (from East Lansing)
4 Swedish-b$^{8}$

*Superscript numerals refer to successive subcultures from a single pair whose $F_1$ progeny were examined cytologically to determine absence of gross chromosomal aberrations.

### Chromosome 1

5 B
6 bi
7 ec ct$^{6}$ v g$^{3}$
8 ec ct$^{6}$ v g$^{3}$/ClB
9 f
10 fw$^{H}$/y
11 m
12 v
13 s
14 w m f
15 w$_2$ m f/ClB
16 y$_2$ w$^{a}$ cv v f B
17 y$_2$ w$^{a}$ spl
18 y$^{2}$ w$^{a}$ spl bi

### Chromosome 2

19 bw
20 dp
21 c$_2$
22 L$_5$
23 L$^{5}$
24 vg

### Chromosome 3

25 e$^{p}$
26 p$^{p}$ by Sb$^{Spi}$/In(3R)C, l(3)a
27 se
28 se ss
29 st

### Chromosome 4

30 ey$^{2}$

### Multichromosomal

31 Ins(2LR)Cy/Pm$^{1}$, ds$^{33k}$; H/In(3R)C, Sb$_2$
32 Ins(2LR)SM1, al$^{2}$ Cy sp$^{2}$/ Pm$^{2}$, Ubx$^{130}$ e$^{s}$/Sb
33 y f;bw;e;ci ey$^{R}$
34 y;bw;e;ci ey$^{R}$

### Inversions

35 In(1)A99b
36 In(1)sc$^{S1}$ B InS w$^{a}$ sc$^{8}$
- In(1)ClB$_3$ (8, 15)
37 In(1)rst$^{3}$, y rst$^{3}$ car bb
38 Ins(2LR)Cy/L;Pm$_2$
- Ins(2LR)SM1, al$^{2}$ Cy sp$^{2}$/ Pm$^{2}$;Ubx$^{130}$ e$^{s}$/Sb (32)
39 In(3L)pers$_p$
- p$^{p}$ by Sb$^{Spi}$/In(3R)C, l(3)a (26)

### Translocations

40 T(2;3)S$^{M}$ Cy/vg$^{nw}$
41 Y$^{S}$X·Y$^{L}$, +/y$^{2}$ su-w$^{a}$ w$^{a}$ bb; Ore-R autosomes

### Closed-X

42 X$^{c2}$

### Attached-X

- fw$^{H}$/y (10)
- Y$^{S}$X·Y$^{L}$, +/y$^{2}$ su-w$^{a}$ w$^{a}$ bb; Ore-R autosomes (41)
- y f;bw;e;ci ey$^{R}$ (33)

## ARLINGTON, TEXAS: ARLINGTON STATE COLLEGE
Department of Biology

Virus-caused $CO_2$ sensitivity stocks: several strains

Delayed-recovery from $CO_2$ anesthesia strains: TDR-orange, TDR-1a, TDR-3, TDR-BC$_3$

## PITTSBURGH, PENNSYLVANIA: UNIVERSITY OF PITTSBURGH
### Department of Biology

#### Wild Stocks

1 Canton-S
2 Oregon-R (isogenic)
3 Samarkand
4 Lausanne-S

#### Chromosome 1

100 $B_5$
103 $f^5$
104 $Hw^{49c}/FM1$
105 $Hw^{49c}$ $f^5/ClB$
106 $Hw^{49c}$ $sn^3/ClB$
107 M(1)n/FM6
108 w
109 w m f
110 y
111 y ac v
112 y f:= & $Hw^{49c}$ $f^5$
113 y f:= & $Hw^{49c}$ $sn^3$
115 y Hw/Ins(1)$sc^{S1L}$, S, $sc^{8R}$ $sc^{S1}$ $w^a$ B $sc^8$
116 y w $sn^3$
117 y f:= & f B
119 y ac/y f:=
120 y ac
121 w B
122 $Hw^{49c}$ $sn^3/FM6$
123 y w f:= & Canton-S

#### Chromosome 2

200 al
202 bw sp
209 cn
211 dp
212 dp cn bw
213 $L^4$
217 net
222 vg
223 $stw^2$
225 bw
226 d/SM5, $al^2$ Cy $lt^v$ $sp^2$
227 d-b/Cy
228 d

#### Chromosome 3

301 e
302 h
303 ru h $e^s$
304 se $e^{11}$
305 ss
306 st
307 $ss^a$
308 se
309 ve h eyg cp
310 ru h th st cu sr $e^s$ Pr cn/TM1, Me ri
311 ry
312 B su-pr/In(3R)C, e(pr)
313 $su^2$-Hw bx bxd/TM1, Me ri
314 $Ubx^{130}$, $e^s$/Sb (Ubx/Sb)

#### Chromosome 4

400 $ey^2$

#### Multichromosomal

500 $al^2$ Cy $lt^3$ $L^4$ sp/Pm, $ds^{33k}$;$Ubx^{130}$, $e^s$/Sb (Cy L/Pm;Ubx/Sb)
501 bw;st
502 $bw^{75}$;st
503 l(2)55i/SM1, al Cy sp; pol
504 $sc^{S1}$ B $In^s$ $w^a$ $sc^8$;Ins SM1, $al^2$ Cy $sp^2$/dp b Pm $ds^{33k}$;C Sb/$Ubx^{130}$ $e^s$;pol
505 vg;$e^{11}$
506 v;bw
507 $Cy^{0}5$/Pm;Ubx/Sb
508 (TIN)$dp^{txI}$ Cy, Ins0 pr $cn^2$ T(2;3)Me, Ins ri $Sh^1$/ S Sp cn;ru h $D^3$ st In(3R)c e l(3)e

#### Translocations

600 M(3)S34/T(2;3)Me

#### Experimental Stocks

900 y $iso(2;3)_1$
901 $sn^3$ $iso(2;3)_2$
902 y $iso(2;3)_2$
904 y ac $iso(2;3)_2$
909 y f:= & FM6 $iso(2;3)_2$
910 y f:= & $Hw^{49c}$ $sn^3$ $iso(2;3)_2$
911 $Hw^{49c}$ $sn^3/FM6$ $iso(2;3)_2$

## MEDFORD, MASSACHUSETTS: TUFTS UNIVERSITY
### Department of Biology

#### Wild Stocks

Smarakand
Inbred (made coisogenic by Dr. Stern in 1948)
Medford

#### Chromosome 1

Muller-5

#### Chromosome 3

$ry^1$
e

#### Multichromosomal

st/st;bw/bw
$dp^{65}$ v/v;bw/bw
Cy;v/v;bw/bw
Cy/Pm
bw/bw;e/e

## NEW HAVEN, CONNECTICUT: YALE UNIVERSITY
### Department of Biology

#### Wild Stocks

1 Canton-S
2 Canton-S-C (inbred)
3 Cockaponsett Forest, Conn.
4 IF-38, Idaho Falls, Idaho
5 NB-1, New Britian, Conn.
6 OZL, New Haven, Conn.
7 Oregon-R
8 Oregon-R (highly inbred)
9 Oregon-K
10 Sevelen
11 Sevelen (highly inbred)
12 Swedish-B
13 Swedish-B (highly inbred)

#### Chromsome 1

14 B
15 B-reverted
16 bi
17 bi $ct^6$ $g^2$
18 car
19 $ct^6$
20 dor/ClB
21 dor/FM4
22 fa
23 fu/ClB
24 $fs^{Nas^A}$/M-5
25 g/ClB
26 $g^{50e}$
27 $g^{50e}$/y f:=
28 $Hw^{49c}$/M-5
29 l(1)48j/M-5
30 $lz^{50e}$
31 na/FM3, $y^{31d}$ $sc^8$ dm B l
32 $g^2$ pl/FM3, $y^{31d}$ $sc^8$ dm B l
33 $pn^2$
34 sc ec v g f/ClB
35 sc ec cv $ct^6$ v g f/ClB, v
36 sc ec v g f
37 $sc^{S1}$ B, In-S, $w^a$ $sc^8$ (M-5)
38 $sn^3$/y f:=
39 $sn^3$ v
40 $sn^3$ v B
41 $sn^4$ oc $ptg^3$/+:=
42 v
43 w
44 w ec
45 w m f
46 w spl
47 $w^a$
48 $w^a$ v B
49 $w^a$ v B/In(1)AM
50 $w^{bf}$
51 $w^{bf}$/FM4
52 $w^{bl}$
53 $w^e$
54 y sc $w^a$
55 $y^2$ sc $w^a$ ec/FM4, $y^{31d}$ $sc^8$ dm B (triploid)
56 $y^2$ sc $w^a$ ec/y f:=
57 $y^2$ v f
58 $y^2$ $w^a$ cv v f/M-5
59 $y^2$ $w^a$ cv $sn^{55a}$ v f/M-5
60 $y^2$ $w^a$ w/y f:=
61 $w^a$ $fw^{49c}$/y f:=

#### Chromosome 2

62 al
63 al b c $sp^2$
64 b
65 b cn vg
66 b vg
67 $bs^2$
68 bw
69 bw $bs^{cy}$
70 cn
71 cn bw
72 cn bw Kr/Pm
73 dp
74 dp $bw^a$
75 $L^2$/Cy $sp^2$
76 $ltd^{37b}$
77 $ltd^{37b}$ vg
78 M(2)$l^2$/SM1, $al^2$ Cy $sp^2$
79 M(2)z/In(2L)t, l(2)R
80 net al ex ds S ast/SM1, $al^2$ Cy $sp^2$
81 pr
82 rc
83 sca
84 vg
85 vg c
86 vg $Mt^A$ bw
87 $spd^{fg}$

#### Chromosome 3

88 $Dfd^{r-1}$
89 $e$
90 $e^4$ wo ro
91 $e^{11}$
92 $e^s$
93 Gl Sb/LVM
94 Ly/$D^3$
95 Ly Sb/LVM
96 l(3)tr Ubx/TM1, Mé ri $sbd^1$
97 Mé, Ins ri $Sb^1$/ru h D Ins CxF ca (h-24, Bloomington, 1957)
98 ru h th st cu sr $e^s$ ca
99 se
100 se e
101 ss
102 $ss^a$
103 st
104 $Ubx^{130}$ $e^s$/Xa
105 FMA3-D/tra

#### Chromosome 4

106 ci $ey^R$
107 $ey^2$
108 Scn/$ey^D$
109 $sv^{de}$/$ey^D$
110 $sv^n$
111 Cat/$ci^D$

#### Multichromosomal

112 $ct^{45e}$ v;bw;e;$ey^2$(1;2;3;4)
113 g;cn(1;2)
114 v;bw(1;2)
115 v;bw;e(1;2;3)
116 v;bw;e;$ey^2$(1;2;3;4)
117 $sc^{S1}$ B In-S $w^a$ $sc^8$;In SM1, $al^2$ Cy $sp^2$/dp b Pm $ds^{33k}$;C Sb/$Ubx^{130}$ $e^s$(1;2;3)
118 v;e(1;3)
119 w;e(1;3)
120 $w^a$ v;e(1;3)
121 $w^e$;cn(1;2)
122 y w;ant(1;2)
123 $y^2$ v f;bw(1;2)
124 bw;e(2;3)
125 bw;st(2;3).
126 cn bw;e(2;3)
127 cn;se(2;3)
128 dp;e(2;3)
129 Pm, dp b/Cy $sp^2$;Sb/CxF (ru h ca?)(2;3)
130 pr;$ey^2$(2;4)
131 e;$ey^2$(3;4)

#### Closed-X

132 $X^c$, y/y f:=

133 In($X^{c2}$)$w^{vc}$ f/dl-49, y w $lz^{s}$

Deficiencies

Chromosome 1

134 Df(1)$g^{1}$, f B/In(1)AM
135 Df(1)$L^{4-2}$/M-5
136 Df(1)$N^{8}$/y Hw $m^{2}$ $g^{4}$, dl-49
137 Df(1)$N^{45e}$/y Hw $m^{2}$ $g^{4}$, dl-49
138 Df(1)$N^{264-40}$/y Hw $m^{2}$ $g^{4}$, dl-49
139 Df(1)$w^{258-11}$, y/y Hw $m^{2}$ $g^{4}$, dl-49
140 Df(1)$w^{258-21}$/y Hw $m^{2}$ $g^{4}$, dl-49
141 Df(1)$w^{258-42}$, y/y Hw $m^{2}$ $g^{4}$, dl-49
142 Df(1)$w^{258-45}$/FM4, $y^{31d}$ sc dm B
143 Df(1)$w^{258-48}$, y/y Hw $m^{2}$ $g^{4}$, dl-49
144 Df(1)$w^{5513.2}$ spl/In(1) $sc^{S1}$ dl-49, v f B
145 Df(1)$w^{56d10.1}$ spl/In(1) $sc^{S1}$ dl-49, y f B
146 Df(1)$w^{59k13}$ spl($sn^{3}$)/ Dp(1)$w^{V}$, cb
147 Df(1)$y^{2}$ su-$w^{a}$ $w^{56l10.2}$/ In(1)$sc^{S1}$ dl-49, v f B
148 Df(1)y sc/M-5(Vogt)

Chromosome 2

149 Df(2)$bw^{5}$ $sp^{2}$/Xa
150 Df(2)$bw^{5}$ $sp^{2}$/SM1, $al^{2}$ Cy $sp^{2}$
151 Df(2)$bw^{VDe2L}$ $Cy^{R}$/Gla
152 Df(2)$Px^{2}$ bw $sp^{2}$/Cy, $al^{2}$ $lt^{3}$ $L^{4}$ $sp^{2}$
153 Df(2)$vg^{B}$/Cy, $L^{4}$ $sp^{2}$
154 Df(2)$vg^{C}$/Cy, $L^{4}$ $sp^{2}$
155 Df(2)$vg^{D}$/Cy, $L^{4}$ $sp^{2}$
156 Df(2)$vg^{S}$ cn/Cy, $al^{2}$ $lt^{3}$ $L^{4}$ $sp^{2}$

Duplications

157 Dp(1;1)Co

Inversions

see 20, 21, 31, 37, 49, 78, 79, 80, 96, 97, etc.

Translocations

158 T(Y;2)C/pr cn

## HAREN, GRONINGEN, NETHERLANDS: UNIVERSITY OF GRONINGEN
### Genetical Institute

Wild Stocks

Argeles
Bacup
Bannerdale
Canton-S
Curacao
Dis.
Dronfield
Groningen (3 strains)
Oregon (Davis)
Oregon-R
O S (Oregon x Samarkand)

Inbred Stocks

Bayfordbury
IsoCanton
Oregon (Sh 0)
Oregon (Mo)

Chromosome 1

B
ClB/$sc^{2}$ ec cv $ct^{6}$ v $s^{2}$ f car bb
ClB/fu $f^{55f}$
g
Muller-5
"Oster ♀"
"Oster ♂"
sc ct v f car + y:=
v
w
w m B
$w_{a}$ y
$w_{a}$
$w^{a}$ v
y
$y_{2}$ f car
$y_{2}$ v f
$y^{59b}$
w cv sn

Chromosome 2

al dp b pr c px sp
al dp b pr c px sp/Cy
b
b vg cn
b vg
$bw_{D}$ cn
$bw^{D}$
$CyL^{4}$/Pm (2 strains)
dp
dp-1 (Thoday)
J/$In^{65c}$
Pin
px sp
stw
$vg_{p}$
$vg^{p}$

Chromosome 3

$e_{11}$
$e^{11}$
H/LVM
$p^{53e}$
ri
RLy/In
ro
ru h th st cu sr $e^{s}$
ru h th st cu sr $e^{s}$ Pr ca/TM1 Me ri
se
se cp e
st
$ve_{2}$
$ve^{2}$

Chromosome 4

$ci^{D}$
$ci_{w}$
$ci^{57g}$
$ey^{R}$
$spa^{pol}$

Multiples

Cy "O"/Pm;$Ubx^{130}$/Sb; ci $ey^{R}$
dor/FM6;TM3/Sb
SM5/Bla;TM3/Sb
$v^{D}$ bw
v st
y bw $st_{ch}$ (2 strains)
y ec w spl;Cy; $Ubx^{130}$/Xa

## BUENOS AIRES, ARGENTINA: ATOMIC ENERGY COMMISSION
### Department of Radiobiology

#### Wild Stocks

W1 Buenos Aires
W2 Oregon R
W3 Isogenic stock (made at intervals from Oregon R)

#### Normal X Chromosomes

a1 B
a2 B Bx car/y f:=
a3 Bx
a4 ct g f
a5 $ct^6$ ras B/y f:=
a6 $g^W$ (subliminal allele)
a7 lz/y f:=
a8 m at
a9 oc ptg Tu/y f:=
a10 ras
a11 sc cv v f B/y f:=
a12 $sc^6$ car
a13 spl rb cx/y f:=
a15 w
a16 $w^{ch}$ rb/y w f
a17 $w^e$ sn/y f:=
a18 $w^e$ sn B/y f:=
a19 $w^t$ f
a20 $w^W$ f
a21 y
a22 y ac sc pn/y f:=
a23 y ac $t^2$·Dp($y^+$ $ac^+$ $sc^{S1}$)/y f:=
a24 y ac sc v/y f:=
a25 y $ct^6$ f
a26 y $ct^6$ ras/y f:=
a27 y cv v sd/y f:=
a28 y fa $wy^2$ $g^2$
a29 y v
a30 y sc $lz^g$ v f/y f:=
a31 y sn oc v B/y f:=
a32 y w $bb_3$
a33 y w $sn^3$
a34 y w f
a35 y $w^a$ m f car
a36 $y^2$ v f car

#### Inverted X Chromosomes

b1 Ins(1)dl-49, $B^{M1}$, $B^{M1}$
b2 In(1)$sc^8$, $sc^8$ $w^a$ bb
b3 Ins(1)$sc^8$, dl-49, $sc^8$ B/y f:=
b4 Ins(1)$sc^8$, dl-49, $sc^8$ v f car/y f:=
b5 Ins(1)$sc^{S1}$, dl-49, $sc^{S1}$ w v f/y f:=
b6 Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ (Insc)
b7 Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ $sd^{spa}$
b8 (winsc) Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ w/y f:=
b9 (Insn) Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ sn/y f:=
b10 (Binsc) Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ B/y f:=
b11 (Binsn) Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ sn B/y f:=
b12 Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ $w^a$ v f/ y f:=
b13 Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ $w^a$ v At/ y f:=
b14 (Basc) Ins(1)$sc^{S1L}$, S, $sc^{8R}$, $sc^{S1+8}$ $w^a$ B/ y f:=
b15 Ins(1)$sc^{S1L}$, S, $y^{3PR}$, $y^{3P}$ $sc^{S1}$ f/y f:=
b16 In(1)$sc^{V2}$, $sc^{V2}$/y f:=
b17 In(1)$B^{M1}$, $sn^3$ v $B^{M1}$/y w f
b18 In(1)$y^{3P}$, $y^{3P}$ B
b19 In(1)$sc^8$, $y^{S1}$ $sc^8$
b20 Ins(1)$sc^8$, dl-49, $y^{S1}$ $sc^8$ $w^a$ v f B/y f:=
b21 Ins(1)$sc^8$, S, $y^{S1}$ $sc^8$ $w^a$ f/y f:=
b22 In(1)dl-49, y $sn^{x2}$ $bb^1$/y f:=
b23 Ins(1)$sc^{S1L}$, $sc^{8R}$, y $sc^{S1+8}$
b24 (Inscy) Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, y $sc^{S1+8}$/ y f:=
b25 (Binscy) Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, y $sc^{S1+8}$ B/y f:=
b26 (winscy) Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, y $sc^{S1+8}$ w/y f:=
b27 Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, y $sc^{S1+8}$ $w^a$ v f/$sc^8$·Y
b28 Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, y $sc^{S1+8}$ $sn^{x2}$ B/y f:=
b29 Ins(1)$sc^{S1}$, dl-49, y $sc^{S1}$ v f B/y f:=
b30 Ins(1)$sc^{4L}$, $sc^{8R}$, y $sc^8$ w/$sc^8$·Y (in ♂ & ♀)
b31 Ins(1)$sc^{4L}$, S, $sc^{8R}$, y $sc^8$ w B/y f:=/$sc^8$·Y
b32 Ins(1)FM6, $y^{31d}$ $sc^8$ dm B/y f:=/$sc^8$·Y
b33 Ins(1)FM4, $y^{31d}$ w dm f/Y & FM4/FM4/Dp $w^+$ $dm^+$ ♀♀

#### Balanced X Chromosomes

c1 In(1)dl-49, y Hw m g/$N^8$
c2 Ins(1)$sc^{S1}$, dl-49, y $ct^1$ $lz^s$ B/w $sn^{5s}$ bb
c3 In(1)AM, $ptg^4$/In(1)$bb^-$, y $sl^2$ $bb^-$
c4 $ct^6$ v dy g f/In(1)A99, $sn^{33}$ f
c5 $w^e$ sn/ClB
c6 f fu/ClB

c7 un Bx/In(1)AM, $ptg^4$
c8 y oc ptg/Ins(1)$sc^{S1}$, dl-49, y $sc^{S1}$ v B/$sc^8$·Y
c9 Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $y^{-(Co-2)}$ $sc^{S1+8}$ B/$y$ $sn^5$ oc v·$Y^S$/$Y^{Lc}$
c10 y pn w cm $ct^6$ $sn^3$ oc $ras^2$ v dy $g^2$ f od car sw/Ins(1)$sc^{S1}$, dl-49, y $sc^{S1}$ y B
c11 $l^{J1}$ $sc^{J1}$ pn w rb cm $ct^6$ oc $ras^2$ v dy $g^2$ f od car sw/Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, y $sc^{S1+8}$ $sn^{x2}$ B/$Y^{Lc}$ ♀♀ & y $sn^5$ oc v·$Y^S$/$Y^{Lc}$ ♂♂ (jynd)
c12 (Maxy-old) $l^{J1+}$·Y/Ins(1)dl-49, $B^{M1}$, $l^{J1}$ $sc^{J1}$ $B^{M1}$/y ac pn w ec rb cm $ct^6$ $sn^3$ oc $ras^2$ v dy $g^2$ f Tu car
c13 Inp(1)$sc^{V1}$, v·$sc^{V1}$ $y^+$/Ins(1)$sc^8$, dl-49, $y^{31d}$ $sc^8$ v f B
c14 Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ $fw^{wr}$/In(1)AM, $y^2$
c15 X·Dp($sc^{V1}$ $y^+$ $ac^+$):$rb^{mott\ 48aH5}$/Ins(1)$sc^{S1}$, dl-49, y $sc^{S1}$ v B
c16 Ins(1)$w^{40aH1}$, $w^{40aH1}$·Dp($sc^{V1}$ $y^+$ $ac^+$)/Ins(1)$sc^{S1}$, dl-49, y $sc^{S1}$ v B
c17 Ins(1)ct-$l^{43aH1}$, ct-$l^{43aH1}$·Dp($sc^{V1}$ $y^-$ $ac^-$)/Ins(1)$sc^{S1}$, dl-49, y $sc^{S1}$ v B
c18 Df(1)$rb^{R15BH3}$, $rb^{R15BH3}$(ring X)/Ins(1)$sc^{S1}$, dl-49, $sc^{S1}$ v f
c19 Ins(1)$sc^{S1L}$, dl-49, $g^{I7Ba6}$,$sc^{8R}$, $sc^{S1+8}$ $g^{I7Ba6}$ B/$ct^n$ oc
c20 Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ $sn^{-(I9Bb5)}$ B/$ct^n$ oc
c21 Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ pn-(I10Ac4) B/$ct^n$ oc
c22 Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ ras-$v^{-(I7Cc8)}$ B/$ct^n$ oc
c23 Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, $sc^{S1+8}$ y ct(♀15B4) $sn^{x2}$ B/y oc ptg/$sc^8$·Y

## Altered X & Y Chromosomes

d1 $Y^S$/$g^2$ B·$Y^L$/y f:=
d2 $sc^{V1}$·$Y^S$/In(1)dl-49, y v B·$Y^L$/y f:=
d3 $Y^S$·$Y^S$No.2/y v f·$Y^L$/y f:=
d4 $y^3$·$Y^L$/y·$Y^S$/y f:=
d5 y $sn^5$ oc v·$Y^S$/$Y^{Lc}$ & Ins(1)ct-l, dl-49, y ct-l $sn^{x2}$·sc $ct^n$ oc ptg car (snoct)
d6 $Y^{Lc}$/oc ptg·$Y^S$ & Ins(1)ct-l, dl-49, y ct-l $sn^{x2}$·y $ct^n$ oc ptg car (snocty)
d7 $sc^8$·Y/y B/y f:=
d8 Y:$bw^+$/$X^+$;cn bw
d9 $Y^c$;$bw^+$/$X^+$;bw
d10 $sc^8$·Y:$bw^+$/$ac^3$;cn bw
d11 $l^{J1+}$·Y/$l^{L1}$ $sc^{J1}$ (extra Y in ♀)
d12 $X^+$/$B^S$·Y/y f:=
d13 y $w^a$/$B^S$·$w^+$·Y
d14 y $w^a$/$B^S$·Y·$sc^8$

## Attached X-Y (no free Y)

e1 $Y^S$·In(1)EN, y v·$Y^L$ ($sc^8$?Y)
e2 $Y^S$·In(1)EN, y·$Y^L$ $sc^8$ $y^+$ & $y^2$ su-$w^a$ $w^a$ bb
e3 $Y^S$·In(1)dl-49, y v f car·$Y^L$
e4 $Y^S$·In(1)EN, y B·$Y^L$ & $y^2$ su-$w^a$ $w^a$ bb
e5 $Y^S$·In(1)EN, sn oc ptg·$Y^L$ & sc $ct^n$ oc ptg car·Ins(1)ct-l, dl-49, y ct-l $sn^{x2}$

## Chromosome 2

f1 cn
f2 c
f3 b pr
f4 px vw mr sp/$S^2$ Cy $lt^3$ pr Bl $cn^2$ L $sp^2$
f5 net b cn crs/$dp^{txI}$ Cy pr Bl $lt^3$ $cn^2$ $L^4$ $sp^2$
f6 al dp b pr cn vg c a px bw mr sp/$S^2$ Cy $lt^3$ pr Bl $cn^2$ $L^4$ $sp^2$

## Chromosome 3

g1 red
g2 e
g3 gl
g4 ru h th st $p^p$ cn sr $e^s$/$Ubx^{130}$
g5 red Ubx ca Kpn/TM3

## Chromosome 4

h1 pol
h2 ci $ey^R$
h3 $sv^n$

## Multichromosomal

M1 (1,2) $y^2$ $t^2$;cn bw
M2 (1,2) y w f;tft
M3 (1,2) v;$bw^{VA}$/Bl $L^2$
M4 (1,3) w;T(2;3)Xa, Xa/Sb
M5 (1,3) y;red
M6 (1,4) In(1)AM, $y^2$/Ins(1)FM6, $y^{31d}$ $sc^8$ dm B/$sc^8$·Y;pol
M7 (1,4) $y^2$ $cho^2$/$B^S$·Y;ci
M8 (2,3) cn bw;e
M9 (1,2,3) $Y^{Lc}$/X·$Y^S$;bw;e
M10 (1,2,4) $y^2$ v;bw;$ci^D$/$ey^D$
M11 (1,3,4) f;bw sr $e^s$;pol
M12 (1,3,4) y/$B^S$·Y;red;ci
M13 (1,3,4) $y^2$ $cho^2$/$B^S$·Y/y f:=;red sbd;ci
M14 (1,2,3,4) y;bw;e;ci $ey^R$
M15 (1,2)(tester 1) y ac pn w rb $wy^2$ $g^2$/y f:=; $sc^{19i}$/Cy
M16 (1,2)(tester 2) $y^2$ $w^a$ cm $wy^2$ $g^2$ car/y f:=; $sc^{19i}$/Cy

M17 (1,2)(tester 3) y rb cm $ras^2$ $g^2$/y f:=; $sc^{19i}$/Cy

M18 (1,2)(maple) y ac sc pn w rb cm $ct^6$ $ras^2$ v $g^2$ f car/y f:=;$sc^{19i}$/In(2L) Cy, Cy lt

M19 (1,2) Ins(1)$sc^{8L}$, S, $sc^{4R}$, sc- $w^a$ B/ y f:=;$sc^{19i}$/Cy $cn^2$

M20 (1,2) Ins(1)$sc^4$, S, y $sc^4$ $w^a$;S $sc^{19i}$ Bl/Cy $L^4$ sp

M21 (1,2) $sc^{19}$-/$l^{J1}$ $sc^{J1}$;fes $sc^{19i}$ b pr/ Cy $dp^{txI}$ pr $cn^2$

M22 (1,3) $w^a$/y v f;tra/In(3)$D^{cxF}$,D

M23 (1,2) Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, y-(Co-2) $sc^{S1+8}$ B;$y^{+i}$(Co-2)

M24 (1,2) Ins(1)$sc^{S1L}$, dl-49, $sc^{8R}$, y-(Co-2) $sc^{S1+8}$ B;$y^{+}$(Co-2) tft/Cy

M25 (1,3) Df(1)$cm^{R8aH4}$(ring X)/y f:=; Dp(1;3)$sn^{R13AH1}$

M26 (1,2,3)(MI) Ins(1)$sc^{8L}$, S, $y^{3PR}$, $y^{S1+3P}$;dp b $Pm^1$/Ins(2LR)Cy, $al^2$ Cy $lt^3$ $cn^2$ $sp^2$;Ins(3)$D^{cxF}$, ru h D ca/ In(3R)Sb, Sb

M27 (1,2,3,4)FM6, $y^{31d}$ $sc^8$ dm B/In(1)AM, $y^2$;b pr Bl/Ins(2)SM1, $al^2$ Cy $sp^2$; Tp(3R)Vno, Vno;Ins(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^s$;pol

M28 Same as M27, but with $sc^8$·Y in ♂♂

M29 (1,2,3,4)FM6, $y^{31d}$ $sc^8$ dm B/In(1)AM, $y^2$;dp b $Pm^1$/SM1, $al^2$ Cy $sp^2$;In(3R) Sb, Sb/Ins(3LR)$Ubx^{130}$, $Ubx^{130}$;pol

M30 (1,2,3,4)Ins(1)$sc^{8L}$, S, $y^{3PR}$, $y^{S1+3P}$; autosomes same as M29

M31 (2,3,4) $X^+$;autosomes same as M29

M32 (1,2,3) Same as M30, but without pol

M33 (2,3) Same as M31, but without pol

M34 (2,3,4) b pr Bl/Ins(2LR)SM1, $al^2$ Cy $sp^2$;Tp(3R)Vno, Vno/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^s$;pol

M35 (2,3) Same as M34, but without pol

M36 (1,3,4) FM6, $y^{31d}$ $sc^8$ dm B/In(1)AM, $y^2$;Tp(3R)Vno, Vno/Ins(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^s$;pol

M37 (1,3,4) Same as M36, but with $sc^8$·Y in ♂♂

M38 (1,2,4) FM6, $y^{31d}$ $sc^8$ dm B/In(1)AM, $y^2$;b pr Bl/Ins(2LR)SM1, $al^2$ Cy $sp^2$; pol

M39 (1,2,4) Same as M38, but with $sc^8$·Y in ♂♂

M40 (1,2,3,4) FM6, $y^{31d}$ $sc^8$ dm B/In(1)AM, $y^2$;SM1, $al^2$ Cy $cn^2$ sp/T;Ins(3)$Ubx^{130}$, $Ubx^{130}$ $e^s$/T(2;3)13-1bb red sbd;pol

## COPENHAGEN, DENMARK: UNIVERSITY OF COPENHAGEN
### Institute of Genetics

#### Wild stocks

1 Oregon R
2 Rastad (Denmark)
3 Wisconsin-3
4 Wisconsin-4
5 Holte (Denmark)
6 Bøtø (Denmark)

#### Chromosome 1

1 y $v^{36f}$
2 B
3 $y^2$ $su^{51c}$-v $ras^2$ $v^1$ f
4 g f car & y f:=
5 w m f
6 f od sy car
7 ec ct v f
8 w
9 Basc

#### Chromosome 2

1 bw
2 b cn vg
3 vg
4 Cy/Pm
5 Cy L/Pm
6 cn
7 px sp
8 c wt px
9 b cn c bw
10 b pr c px sp
11 wt
12 al dp b c px sp/Cy
13 fj wt/Xa

#### Chromosome 3

1 D/H
2 st
3 e
4 st e
5 e ro

#### Chromosome 4

1 $ey^D$

#### Multichromosomal

1 cn bw;e
2 v;cn;st
3 vg;st
4 B;st
5 Cy L/Pm;Ubx/Sb
6 bw;st
7 v;bw
8 v;Cy/Pm
9 v;cn
10 y $v^{36f}$;px sp
11 ct v f;bw

## UPPSALA, SWEDEN: UNIVERSITY OF UPPSALA
Department of Genetics

### Wild Stocks

1 Algeria
2 Amherst-3
3 Bayfordbury
4 Boa Esperanca, Minas Gerais, Brazil
5 Canton-S
6 Crimea
7 Curitiba
8 Florida
9 Formosa
10 Gruta, Argentina
11 Hikone-R (resistant to BHC, DDT, parathione, nicotine)
12 Karsnäs
13 Kochi-R (resistant to parathione)
14 Oregon-R
15 Salvador, Bahia, Brazil
16 San Miguel, Buenos Aires, Argentina
17 Stäket
18 Tunnelgatan
19 Ultuna
20 Örebro

### Chromosome 1

101 B
102 B/y:=
103 BB car;$sc^8$ Y y f:=; $sc^8$ Y
104 f $B^i$/y f:=
105 ct
106 cv
107 cv $sn^3$
108 ec
109 ec ct v g
110 ec ct v f
111 f
112 f B od car/y f:=
113 f BB;$sc^8$ Y/y f:=; $sc^8$ Y
114 f $B^i$ $B^i$/y f:=
115 f od sy car
116 fu/y f:=
117 $g_2$
118 $g^2$ B
119 In(1)$w^{m4}$
120 lz/ClB
121 ma-l/y f:=
122 od car
123 rb
124 x $rb^{27-4}$ cv v $f^{3N}$/ y f:=
125 rb cx
126 sc z $w^{17G2}$ ec/y w f:=
127 sc z ec
128 sc $z^{mottled}$
129 $sc^{S1}$ B InS $w^a$ $sc^8$
130 $sc^{S1}$ InS $w^a$ $sc^8$
131 $sn^3$
132 sp-w
133 sp-$w^2$
134 su-$w^a$ $w^a$
135 v g
136 w
137 w cv
138 w cv $sn^3$
139 w $sn^3$
140 $w^a$ su-f
141 $w^{a4}$/y f:=
142 $w^{bf}$ $f^5$
143 $w^{bf2}$
144 $w^{bl}$
145 $w^{Bwx}$
146 $w^{ch}$ wy
147 $w^{co}$
148 $w^{co}$ $sn^2$
149 $w^e$
150 $w^{e2}$
151 $w^{e2}$ en-$w^e$/y f:=
152 $w^h$
153 $w^h$ ct
154 $w^h$ cv/y:=
155 $w^i$ vb
156 $w^{sat}$
157 y
158 $y^2$ ac $sc^+$ pn w rb cm $ct^6$ $sn^3$ $ras^4$ v m g f car/ Muller-5
159 y ec ct v f
160 y $rst^3$ car
161 y f Eb/$sc^{S1}$ B InS $w^a$ $sc^8$
162 y $sc^{S1}$ B InS $w^a$ $sc^8$(y M5)
163 $y^2$ sc $w^a$ $w^{ch}$ fa/y w f:=
164 $y^2$ sc $w^i$
165 $y^2$ sc $w^i$ $w^{ch}$
166 $y^2$ su-$w^a$ $w^{a2}$ $w^{ch}$ spl/ y f:=
167 $y^2$ $w^a$
168 $y^2$ $w^a$ w
169 $y^2$ $w^a$ ec
170 $y^2$ $w^{bf}$ spl $sn^3$/y f:=
171 z
172 sc z $w^{a3}$/y w f:=
173 z $w^{11E4}$

### Chromosome 2

201 bw
202 al b c sp
203 $bw^D$
204 Cy/Pm
205 Cy/S
206 fes Alu lt/$al^2$ Cy $lt^3$
207 $nw^2$/Cy RNS
208 pr
209 $S^2$ Cy pr Bl $cn^2$ $L^4$ bw sp/In-NSL In NSR px sp
210 vg
211 vg bw

### Chromosome 3

301 ca
302 cd
303 $D^3$/InP
304 D/Sb
305 $e^{11}$
306 lz s-u
307 $kar^2$
308 $ri_2$ ss
309 $ri^2$
310 $ri^2$ ss
311 ro
312 ru h st $p^p$ ss $e^s$
313 ry
314 $ry^2$
315 $ry^2$ cd
316 se
317 ss
318 st
319 st p
320 st ry
321 st ss $e^{11}$

### Chromosome 4

401 $ci^D$ pol/$spa^{Cat}$
402 $sv^n$

### Multichromosomal

501 $w^{ch}$;Su-$w^{ch}$/Cy (1;2)
502 $w^{col}$;bw (1;2)

503 $w^{e}$;cr-u/Cy (1;2)
504 $y^{S1}$ $sc^{8}$ InS $y^{3P}$;$al^{2}$ Cy $lt^{3}$ $sp^{2}$/dp b $Pm^{1}$;ru h $D^{3}$ InCXF ca/Sb In(3R) (1;2;3)
505 y' w spl;Cy;$Ubx^{130}$/Xa (1;2;3)
506 bw;cd(2;3)
507 bw;st(2;3)
508 cn bw;$e^{11}$(2;3)
509 Cy/S;D/InP(2;3)
510 L spl;th(2;3)
511 $L^{2}$/+, sp;th(2;3)
512 sp;th(2;3)

Deficiencies & Duplications

601 sc z $Df(1)w^{258-45}$/FM4
602 $y^{2}$ $Df(1)w^{258-45}$/FM4
603 $Df(1)w^{258-45}$, y w spl dm;$Dp(1;3)w^{Vco}$/y w f:=
604 $Dp(1;2R)w^{+51b7}$
605 $Dp(1;4)w^{+51c20}$
606 $Dp(1)w^{a}$/y w f:=
607 $Dp(w^{a4}/w^{a})$/y f:=
608 $Dp(w^{bf}/w^{a})$ec
609 sc $Dp(1)z^{59d15}$/y w f:=
610 z $Dp(w^{a4}/w^{a})$/y f:=

Translocations

701 $T(1;4)w^{m5}$/w;ci $ey^{r}$
702 $T(2;3)bw^{VDe4}$/Cy

Triploids

801 $\frac{y^{2}\ sc\ w^{a}\ ec}{sc^{8}\ dm\ B}$/FM6, $y^{31d}$,

## SEOUL, KOREA: CHUNGANG UNIVERSITY
### Department of Biology

Wild Stocks

1 Canton-S
2 Daekwanryung (Korea)
3 Damyang (Korea)
4 Heuksando-1 (Korea)
5 Heuksando-2 (Korea)
6 Kwangju-1 (Korea)
7 Oregon-R
8 Oregon-R-C
9 Oregon-S
10 Samarkand
11 Seoul-1 (Korea)
12 Seoul-2 (Korea)
13 Seoul-3 (Korea)
14 Suwon (Korea)
15 Swedish-C
16 Yangdong (Korea)

Chromosome 1

17 B
18 bo
19 $br_{3}$
20 $Bx^{3}$
21 cm
22 ec
23 fa
24 rg
25 sc cv v eq
26 sc cv v f
27 $t_{2}$
28 $t^{2}$ vf
29 v
30 $w_{a}$
31 $w_{bf2}$
32 $w_{ch}$
33 $w_{col}$
34 $w_{e}$
35 $w^{e}$ $bb^{1}$/ClB
36 y
37 y ac $v_{2}$
38 $y_{2}$sc $mf^{2}$
39 $y^{2}$ cv v $f_{8}$
40 Basc/y $sc^{8}$ $y_{3}$
41 Basc/y ac $sn^{3}$ cn

Chromosome 2

42 a px or
43 a px sp
44 ab
45 al
46 al bc $sp^{2}$
47 b
48 b lt wxt bw
49 b vg
50 bw
51 bw ba
52 Bl/$Cy_{a}$ $bw^{45a}$ $sp^{2}$ or
53 c
54 c wt px
55 cl
56 cn bw
57 Cy/Pm
58 ex
59 ho
60 $L_{4}$
61 $L^{4}$
62 pd
63 pr
64 rh
65 so
66 vg
67 wt

Chromosome 3

68 aa h
69 bul
70 ca
71 cu
72 D/GI
73 gl
74 h
75 jv
76 p
77 ra
78 ro
79 ru
80 se
81 se h
82 ss
83 st
84 th

Chromosome 4

85 bt
86 ci
87 ci gvl bt
88 ey

Multichromosomal

89 v;bw(1;2)
90 w;vg(1;2)
91 M-5;Cy/Pm;Sb/Ubx (1;2;3)
92 Cy/Pm;Sb/Ubx(2;3)
93 Cy/Pm;D/Bd(2;3)
94 vg;se(2;3)

Attached-X

95 $\frac{br\ ec}{}$/$y^{3d}$
96 $\frac{y}{g^{2}}$ ty

Duplications

97 Dp(2;3)S

Inversions

98 $Vg^{nw}$ Hia/$SM5_{2}$ $al^{2}$ Cy $lt^{L}$ $sp^{2}$
99 $Vg^{u}$/Roi, bw sp or

Translocations

100 T(1;2)Bld/ClB$^{D}$
101 T(2;3)Xa/Sb $bx^{D}$

## TOKYO, JAPAN: TOKYO METROPOLITAN UNIVERSITY
### Department of Biology

Wild Stocks

Oregon-R (isogenic; 466 generations)
Samarkand (isogenic; 577 generations)
48 strains maintained by mass culture

Chromosome 1

101 B
103 bb $Y^{bb}$
104 ec $ct^6$ $g^2$ $bb^1$/ClB
105 f $B^i$ $B^i$/y f:=
107 m
108 $sc^{S1}$ B Ins $w^a$ $sc^8$
109 sd mc
110 v
111 w
112 w m
113 $w^a$
114 $w^a$ m
115 $w^e$
116 y
117 y w m f
118 l(1)7/FH6, $y^{31d}$ $sc^8$ $w^a$

Chromosome 2

202 al dp b pr c px sp/Cy pr (all)
203 $ap^4$/Cy
204 bw
206 cn
207 cn bw
208 conditioned lethal/Cy
209 Cy
210 dp bw
211 dp x
212 $L^5$
213 $L^{52c}$ $vg^{no}$
214 $l(2)^{50}$/Cy
215 l(2)mat/SM5, $al^2$ $lt^v$ Cy $sp^2$
216 l(2)me/SM1, $al^2$ Cy $sp^2$
217 M(2)S7/SM5, $al^2$ Cy $lt^v$ $sp^2$
218 M(2)S11/Cy $bw^{v34}$
219 S Sp $ab^2$ ltd/NS, px sp
221 vg
222 $vg^{ni}$
223 $vg^{nG}$
224 $vg^{no}$
225 $vg^{np}$
226 $vg^D$/SM5, $al^2$ Cy $lt^v$ $sp^2$
227 $vg^U$/Roi, bw sp or
228 $vg^{nw}$ Hia/SM5, $al^2$ Cy $lt^v$ $sp^2$

Chromosome 3

301 $Bd^{49l}$/Sb
302 Bxl/Payne, Dfd ca
303 cu
305 $Dl^{49l}$/l(3)
306 $e^{11}$
307 $Gl^{53b17}$ H/LVM
308 Sd/l(3) (domi. vg-)
309 se
310 st
311 ve
312 N-X/Xa
313 l(3)tr Sb/In(3LR)$Ubx^{130}$
314 ry
315 $ry^2$
316 ca
317 ru h th st cu sr $e^s$ ca

Chromosome 4

402 $ci^D$/Cat
403 Scn/$ey^D$
404 $sv^n$

Multichromosomal

501 b;tx

Attached-XY

601 v f B $\overline{XY}$/$y^2$ su-$w^a$ $w^a$ bb

Deficiencies

701 Df(2)MS4/SM1, $al^2$ Cy $sp^2$
702 Df(2)MB/SM1, $al^2$ Cy $sp^2$
703 Df(2)$vg^C$/Rvd
704 Df(2)$vg^C$/SM5, $al^2$ Cy $lt^v$ $sp^2$
705 Df(2)$vg^B$/SM5, $al^2$ Cy $lt^v$ $sp^2$

## FREIBURG, GERMANY: ZOOLOGISCHES INSTITUT DER UNIVERSITÄT

Note: see the list of Max Planck-Institut für Biologie, Tübingen.

## STOCKHOLM, SWEDEN: UNIVERSITY OF STOCKHOLM
### Institute of Genetics

#### Wild Stocks

1 Canton S
2 Karsnäs
3 Oregon
4 Skaftö

#### Chromosome 1

101 B
102 cv
103 cv $sn^3$
104 ct$^6$
105 ec ct v f
106 f
107 m
108 m f
109 $pn^8$
110 sc
111 sc cv
112 sc cv v f
113 $sc^{S1}$ cv v car
114 $sc^{S1}$ B $In^S$ $w^a$ $sc^8$
115 $sc^{S1}$ $In^S$ $w^a$ $sc^8$; y sc$^8$ Y
116 $sn^3$
117 v
118 w
119 $Y{\cdot}w^+/y\ w^a$
120 w cv
121 w cv $sn^3$
122 $w^a$ $sn^3$
123 $w^a$
124 $w^a$ B ♂ x y f:= ♀ $ec^2$
125 $w^h$
126 w
127 $y_4$
128 $y^{329}$
129 y
130 y ac$^8$ sc pn sn; sc$^8$ Y
131 y ac sc$^4$ pn w rb cm $ct^{S1}$ $sn^3$ ras$^4$ v m g f car/$sc^{S1}$ B $In^S$ $w^a$ $sc^8$ ♀ x $sc^{S1}$ B $In^S$ $w^a$ $sc^8$ ♂
132 y ec ct$^6$ v f
133 y ec ct v f;$sc^8$ Y
134 $y^2$ eq;Df(Y)Y$^{bb}$
135 y f car
136 y f:=/$sc^8$ Y x y Muller 5/$sc^8$ Y
137 y Hw m g f $Eb^8$/$sc^{S1}$ B $In^S$ $w^a$ $sc^8$ ♀ $sc^{S1}$ B $In^S$ $w^a$ $sc^8$ ♂
138 $y^{16}$;$sc^8$ Y
139 y $sc^4$ $In^S$ $sc^{S1}$ (extra Y in ♀)
140 y $sc^4$ $sc^8$
141 y $sc^8$ B f In dl49 v ♂ y f:=$_{S1}$ ♀
142 y $sc^{S1}$ $In^S$ $w^a$ $sc^8$
143 y $sc^{S1}$ B $In^S$ $w^a$ $sc^8$
144 $y_S$ v f·= ♀ Y·Y In dl49 y v f car ♂
145 y v g f
146 y w $sn^3$
147 y w sn;$sc^8$ Y
148 y w sn/y w sn/$sc^8$ Y ♀ x y w sn $sc^8$ ♂
149 y w spl sn

#### Chromosome 2

201 a px sp
202 al b c sp
203 al dp b pr c px sp
204 al dp b pr cn vg c a px bw $mr_2$ $sp^4$/$S^2$ Cy $lt^3$ pr Bl $cn^2$ $L^4$ $sp^2$
205 $al^2$ Cy $lt^3$ $L^4$ $sp^2$/Pm
206 al S ast ho/Cy E-S
207 b cn vg
208 b pr vg
209 bw$^D$
210 cn bw
211 Cy/Pm
212 dp b
213 px bw mr sp/$ds^{33k}_2$ $L^4$ Pm
214 $S^2$ Cy pr Bl $cn^2$ $L^4$ bw sp/In NSL In NSR px sp
215 S $Sp_4$ Bl $bw^D$/$al^2$ Cy $lt^3$ $L^4$ $sp^2$
216 vg

#### Chromosome 3

301 $cu^3$
302 $D^3$/InP
303 Gl Sb/LVM
304 K-pn
305 ru h st $p^p$ ss $e^s$
306 ru se h st $p^p$ ss $e^s$
307 se
308 ss
309 st
310 st ss $e^{11}$
311 W

#### Chromosome 4

401 ci $ey^R$

#### Multichromosomal

501 bw;st
502 cn bw;$e^{11}$
503 sp;th
504 $sc^{S1}$ B $In^S$ $w^a$ $sc^8$;sp;th
505 $sc^{S1}$ B $In^S$ $w^a$ $sc^8$;cn bw;$e^{11}$
506 T(1;2)$B^{bd}$/$Cy_4$ ♀ M2e/Cy ♂
507 T(2;3)$bw^V$ $De^3$/Cy
508 v;bw
509 vg;st ss
510 $y_{S1}$ ec$^8$ ct v f;sp
511 y $sc^8$ $In^S$ $y^{3p}_1$;$al^2$ $Cy_3$ $lt^3$ $sp^2$/dp b Pm;ru h $D^3$ In CxF ca/Sb In(3R) "Marked Inversions"

## TURKU, FINLAND: UNIVERSITY OF FINLAND
### Department of Genetics

The stock list remains unchanged (see DIS 40).

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

### Wild Stocks

Oregon-R
Otaru
Sapporo (2 strains)

### Chromosome 1

w
v
w B

### Chromosome 2

Cy

### Chromosome 3

se

### Multichromosomal

vg/se

## LEIDEN, NETHERLANDS: GENETISCH LABORATORIUM DER RIJKSUNIVERSITEIT

### Wild Stocks

1 Kolmar
2 Leiden

### Chromosome 1

101 B
102 ClB/sc ec cv ct$^{6}$ v s$^{2}$ f car bb
103 cv
106 m
107 sc$^{S1}$ B InS w$^{a}$ sc$^{8}$
108 sn$^{3}$
109 v$_{2}$
110 y$^{2}$ su-w$^{a}$ & v f$^{3n}$ car:=
111 w
112 + & w:=
113 w cv sn
114 sc ct v wy f car & y:=
115 f & v f$^{3n}$ car:=
120 z$^{58g}$

### Altered Y's

151 Y$^{lc}$/X·Y$^{s}$ "sterilizer +"
152 Y$^{lc}$/X·Y$^{s}$;vg "sterilizer vg"
153 Y$^{lc}$/X·Y$^{s}$;spa$^{pol}$ "sterilizer spa$^{pol}$"
154 Y$^{lc}$/X·Y$^{s}$;vg;spa$^{pol}$ "sterilizer vg;spa$^{pol}$"

### Chromosome 2

201 al dp b pr c px sp
202 al dp b pr c px sp/Cy
204 Bl$_{60g}$L/Cy
205 bw$_{D}$
206 bw
208 cn
209 cn bw
209a cn vg bw
210 crc cn/Pm cn
211 crc cn/Pm
213 px sp
215 vg

### Chromosome 3

301 e
302 Gl/Ubx$^{130}$
303 h gs th
304 ltr/Ubx$^{130}$
305 Ly/D$^{3}$
308 ru h th st cu sr e$^{s}$
309 ru h th st cu sr e$^{s}$ Pr ca/TM1, Mé ri
310 ve$_{2}$
311 ve$_{2}$
312 ve$^{2}$ se

### Chromosome 4

402 ci$^{57g}$
403 ci$^{w}_{D+G}$ spa$^{pol}$/spa$^{Cat}$
404 ci$_{D}$ D
405 ci$_{R}$/ey
406 ey
408 spa$^{pol}$

### Multichromosomal

501 Cy/Pm;D/Sb
502 Cy "Oster"/Pm;Ubx$^{130}$/Sb;ci ey$_{R}$
503 dor/FM6;TM3/Sb
504 ec br;ix/Cy
505 SM5/Bla;TM3/Sb
505a v;cn
506 vg;spa$^{pol}$
508 y;bw;st
509 y ec w$^{ch}$ spl; (Cy;Ubx$^{130}$/Xa)

## BERLIN-DAHLEM, GERMANY: INSTITUT FÜR GENETIK DER FREIEN UNIVERSITÄT BERLIN

### Wild Stocks

1 Berlin wild K
2 Berlin wild B
3 England

### Chromosome 1

4 B
5 ClB/+
6 car
7 cv
8 f
9 In(1)dl-49, ty-1 $bb^{1}$/y v f car:=
10 l(1)7/dl-49, y Hw w $lz^{s}$
11 m
12 sc ec ct
13 sc ec ct v g f
14 $sc^{S1}$ InS B $w^{a}$ $sc^{8}$
15 v
16 w
17 $w^{a}$
18 $w^{bf}$
19 $w^{e}$
20 $w^{m4}$
21 w $sn^{3}$
22 $w^{co}$ $sn^{2}$
23 $w^{ch}$ wy
24 wy
25 y cv v f
26 y w
27 y y:=/+

### Chromosome 2

28 al dp
29 b cn vg
30 bw
31 $L^{2}$/Cy
32 $tu^{g}$
33 vg

### Chromosome 3

34 $bx^{34e}$
35 $Dfd^{rL}$
36 $e^{11}$
37 jv se
38 Ly/$D^{3}$
39 ri
40 ru h st Dfd $p^{p}$ ss $e^{s}$
41 st
42 Tu

### Chromosome 4

43 $bt^{D}$/$ci^{D}$
44 ci $ey^{R}$
45 $ey^{2}$

### Multichromosomal

46 Bld $w^{a}$/w;Cy
47 cn;ss
48 vg;$e^{11}$
49 $sc^{S1}$ 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
50 $y^{S1}$ $sc^{8}$ InS $y^{3P}$;$al^{2}$ Cy $lt^{3}$ $sp^{2}$/dp b $Pm^{1}$; ru h $D^{3}$ InCXF ca/Sb In(3R)
51 y f:=;bw;e;pol

### Altered Y

52 Multi ♂: $sc^{8}$·Y/y In49 B;$bw^{D}$ ♂ & y f:=;$bw^{D}$ ♀
53 Multi ♀: X·Y InEN y;st
54 Sterilizer +: $Y^{LC}$/X·$Y^{S}$ ♂ & X·$Y^{S}$ ♀
55 Sterilizer cn bw;e: $Y^{LC}$/X·$Y^{S}$;cn bw;e ♀

### Selected for DDT-resistance

56 Berlin wild, DDT-resistant 1

### Unanalyzed

57 $CO_2$-sensitive

## NEW DEHLI, INDIA: INDIAN AGRICULTURAL RESEARCH INSTITUTE
### Division of Botany

ocks are as listed in DIS 40:25, with the following additions:

1 $w_{a}$
2 $w^{a}$
3 $w^{e}$
4 car f ras ct & y v f

## BIRMINGHAM, ENGLAND: UNIVERSITY OF BIRMINGHAM
### Department of Genetics

Collected Stocks:

1 Edinburgh
2 Wellington
3 Florida

Inbred for 15-600 generations:

4 Oregon
5 Samarkand
6 6C/L

Chromosome 1

6 B
8 w
9 w m B
10 y v f

Chromosome 2

11 cn
12 vg
13 b cn vg
14 dp cn bw
15 Cy/al dp pr c px sp

Chromosome 3

16 se
17 e
18 st
19 st $p^p$
20 se cp e

Chromosome 4

21 $ey^2$

Multichromosomal

22 v;bw
23 vg;st $p^p$
24 v;cn
25 y;bw;st

Rearrangements

26 ClB/+
27 Muller-5
28 Mé Sb e/He
29 Cy L/Pm; H/Sb

Attached-X

30 y x w

## SEOUL, KOREA: SEOUL NATIONAL UNIVERSITY
### Department of Zoology

Wild Stocks

Canton-S
Oregon-R
Oregon-S
Seoul-S

Chromosome 1

bo
br
cm
ec
fa
sc cv v f
$t^2$ v f
v
w
$w^a$
$w^e$ $bb^1$/ClB
y
M-5/y $sc^8$ y

Chromosome 2

ab
al dp b pr c px sp/Cy, pr
b lt wxt bw
b vg
c
c wt px
Cy/Pm
L
$L^4$
pd
pr

Chromosome 3

aa h
cv-c $sbd^2$
gl
Gl Sb/LVM
h
jv
p
ro
ru
ru h th st cu sr $e^s$ ca
se h
ss
st
th st cp

Chromosome 4

ci gvl bt
ey
ci gvl $ey^R/sv^n$
spa

Multichromosomal

v;bw (1;2)
w;vg (1;2)
bw;st (2;3)
Cy/Pm;D/Sb (2;3)
Cy/Pm;Sb/Ubx (2;3)
lys rc;ss (2;3)
vg;se (2;3)

Inversions

Muller-5

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

### Wild Stocks

1 Canton-S (iso)
2 Kochi
3 Mexico
4 New York
5 Oregon-R

### Chromosome 1

101 B
102 car
103 cm
104 f
105 $lz^{50e}$
106 $rb_3$
107 $sn^3$
108 v
109 w
110 $w^a$
111 $w^{co}$ $sn^2$
112 y Oregon-R
113 y w $w^a$
114 y w f
115 y w m f

### Chromosome 2

201 b gp
202 bw
203 cl
204 cn
205 cn bw
206 cn Cy/Pm
207 cn fes(K)bw/Cy
208 cn vg bw
209 Cy
210 dp
211 ft
212 Gla/Cy pr
213 l(2)gl cn bw/Cy
214 $L^2$
215 px
216 sca cn
217 vg

### Chromosome 3

301 bar-3
302 $ca^2$
303 Dp/In(3L)p, In(3R) C, Sb e l(3)e
304 $e^{11}$
305 gl-1
306 jv
307 ld
308 se
309 se ss
310 ss
311 $ss^a$
312 st
313 st ss
314 $tx^{52j}$
315 ve

### Chromosome 4

401 $ey^2$
402 gvl
403 pol

### Multichromosomal

1;2
501 v;bw
502 v;cn
2;3
503 b;se
504 bw;st
505 bw;st ss
506 $cl^{57j}$;$ss^a$
507 cn;st
508 Cy/Pm;Sb e/$Ubx^{130}$ es
509 $S^2$/Pm;Sb e/$Ubx^{130}$ es
510 vg;$e^{11}$
2;4
211 ft;pol
2;3;4
512 bw;st;$sv^n$
513 cn;ss;gvl

### Attached-X

601 yy:+ (Oregon-R)

### Special Stocks

(A) Insecticide-resistant

701 Hikone-R (multiple) (Japan)
702 HL2-Q (multiple) (USA)
703 KSL (multiple) (Sweden)
704 RI8-A (multiple, intermediate) (USA)
705 RI8-B (multiple, intermediate) (USA)
706 RI8-H (multiple, intermediate) (USA)
707 TG-57 (multiple)(Korea)
708 WMB (multiple) (Japan)
709 WMB-150 (multiple) (Japan)
710 WMD-7-100-42 (multiple) (Japan)
711 bw;NS-R (nicotine sulfate) (Japan)
712 $cn^{55a}$;NS-R (nicotine sulfate) (Japan)
713 TDE-R50 (TDE) (Japan)

(B) Amylase

801 $Amy^1$ (Hikone)
802 $Amy^1$ (Kyoto)
803 $Amy^1$ (Oregon-R)
804 $Amy^1$ (Suyama)
805 cn $Amy^1$ bw
806 cn $L^2$ $Amy^1$ bw
807 $Amy^{1.3}$ ($L^2$)
808 cn $L^2$ $Amy^{1.3}$ bw
809 $L^2$ $Amy^{1.3}$
810 $Amy^{1.6}$ (Suyama)
811 $Amy^{2.6}$ (Hikone)
812 cn $Amy^{2.6}$ bw
813 $Amy^{3.6}$ (Kyoto)
814 $Amy^{1.4}$ (w)
815 $Amy^{4.6}$ ($ad^{60}$)

## MELBOURNE, AUSTRALIA: UNIVERSITY OF MELBOURNE
Department of Genetics

Unchanged, refer to DIS 34.

## UTRECHT, NETHERLANDS: GENETISCH INTSTITUUT VAN DE RIJKSUNIVERSITEIT

### Wild Stocks

1a Oregon K
1b Wageningen

### Chromosome 1

2 $cm_{55}$ $ct^{6}$ $sn^{3}$ & y w f:=
2a $cs^{26-48}$ & y $w_{3N}$ bb•=
3 car $f^{3N}$ & y f:=
4 cv $f^{3N}$ & y f:=
5 $fu$/ClB
6 $g^{2}$
7 pn
8 ras dy
9 $rb_{27-4}$
10 rb cv v $f^{3N}$
11 $sc_{S1}$ cv v f
12 sc B In S $w^{a}$ $sc^{8}$
13 "tester 1" y ac pn w rb $wy^{2}$ $g^{2}$ & y f:=;$sc^{19i}$/$Cy_{2}$
14 "tester 2" $y^{2}$ $w^{a}$ cm $wy^{2}$ $g^{2}$ car & y f:=;$sc^{19i}$/$Cy_{2}$
15 "tester 3" y rb cm ras $g^{2}$ & y f:=;$sc^{19i}$/Cy
16 w sn B
16a w sn
16b w cv sn
16c $w_{S1}$
17 $y^{S1}$ $sc^{8}$ B f In(1)49 v
18 y $w_{a}$ m B
19 $y_{C2}$ $w^{a}$ cv v f
19a X y B & y f:=

### Chromosome 2

19b $sc_{8}$ ec cv $ct^{6}$ v $g^{2}$ f/$FM_{6}$
19c $ct^{n}$ $oc_{1}$ $12^{3}$ v/$FM_{6}$ $ct^{6}$ $v^{6}$
19d v dy $g^{2}_{2}$ sd
19e v dy $g^{2}$ sd $f^{36a}$

### Chromosome 2

28 b pr vg
28a b cn vg
28b b cn vg bw
28c b pr cn vg
29 bw
30 Bl L/Cy
31 dp
32 dp b cn bw
32a $cn_{8}$ bw
33 $dp^{Th}$ Cy, In-L pr $cn^{2}$ In Cy R-O/In s-NSL Ins-NSR p x sp
34 $dp^{Th}$ Cy cn bw/S Sp cn bw
35 J/In(2L)t, l(2)B
35a S/Cy, EN-S
35b $S^{R}$/$ds_{33K}$ Pm

### Chromosome 3

36 e
36a cu
36b gs
37 h ri
38 l tr/e In(3R)In(3L)
39 Mio/In(3R)Sb
39a ss
40 st

40a st e
40b st ss
40c st $ss_{r}$ $e^{s}$
41 st $Sb^{r}$ $e^{s}$ ro ca
41b ro
41c ca

### Chromosome 4

42 $ci_{D}$
43 $ci^{D}$/$spa^{Cat}$

### Multichromosomal

44 y $sc^{S1}$ In 49 $sc^{8}$;dp b cn bw
45 $w^{a}$;tra/D In sc x F & y v f:=;tra/D In sc x F
46 cn bw;e
47 Cy/Pm;Cx, D/In(3R)Sb

### Stocks selected for abnormal abdomen

48 (AA)DCxF/Me Sb
49 (AA)Cx, D/In(3R)Sb

### Deficiencies

52 $Df(1)N^{8}_{264-105}$/dl-49, y Hw $m^{2}$ $g^{4}$
53 Df(1)N(dm)/dl-49, y Hw $m^{2}$ $g^{4}$
54 $Df(1)N^{8}_{264-39}$ $w^{ch}$/FM4, $y^{31d}$ $sc^{8}$ dm B

### Altered Y's

20 X•$Y^{+}$ In ENy;st(no free Y)"multi ♀"
21 l $J^{+}_{1}$•Y/l J1 $sc^{J1(+)}$ $sc^{8}$ In(1)49 v ptg oc $B^{M1}$/$y^{S1}$ $sc^{S1}$, In car odsy f $g^{2}$ dy v $ras^{2}$ $sn^{3}$ $ct^{6}$ $cm_{8}$ rb ec w l pn sc ("Maxy-v")
22 sc•Y/y Hw In(1)49 v ptg oc $f^{S1}$ $B^{M1}$/$y^{S1}$ $sc^{S1}$, In car odsy f $g^{2}$ dy v $ras^{2}$ $sn^{3}$ $ct^{6}$ cm $rb_{8}$ ec w l pn sc
23 sc•Y/y In 49 B;$bw^{D}$ ♂ & y f:=;$bw^{D}$ "Multi ♂"
24 $Y^{Lc}$/X•$Y^{s}$;bw "sterilizer +"
25 $Y^{Lc}$/X•$Y^{s}$;bw "sterilizer bw"
26 $Y^{Lc}$/X•$Y^{S}$;dp "sterilizer dp"
27 $Y^{Lc}$/X•$Y^{S}$;cn bw;e "sterilizer cn bw;e"

## VIENNA, AUSTRIA: UNIVERSITY OF VIENNA
Department for General Biology

See DIS 35:34

## HARWELL, DIDCOT, BERKS., ENGLAND: MEDICAL RESEARCH COUNCIL
### Radiobiological Research Unit

Wild stocks

1 Oregon-K
2 Oregon-R

Inbred lines

3 light ($F_{134}$)
4 Oregon-S ($F_{134}^{360}$)
5 straw ($F_{134}$)

Chromosome 1

6 B
7 $sc^{S1}$ B Ins $w^a$ $sc^8$
8 w
9 y

Chromosome 2

10 al dp b pr c px sp/Cy
11 a px
12 a px pd bw
13 b
14 bw
15 cn
16 Cy/Bl $L^2$
17 ds dp/Cy
18 dp
19 el
20 el b
21 el b pr lt ltd cn/Cy
22 el b pr lt ltd cn a px pd bw/Cy
23 hk pr
24 ho ed cl
25 lt
26 ltd
27 ltd $cn_3$
28 lt $stw^3$
29 net
30 net al ex ds S ast shv ho rub/SM1 $al^2$ Cy $sp^2$
31 pd
32 pd bw
33 pr
34 pr $stw^3$/Cy
35 pym/Cy
36 rl
37 sp
38 $stw^3$
39 vg

Chromosome 3

40 cp
41 ri

Closed-X

42 $X^{c2}$ ec f/y f
43 $X^{c2}$ y B/s $c^8$ Y y f/$sc^8$ Y

Multichromosomal

44 $y^2$, FMA3;al S ast ho/SM1
45 y $sc^{S1}$ In49 $sc^8$;bw;st pp

Triploid

46 $y^2$ sc $w^a$ ec/FM4, $y^{31d}$ $sc^8$ dm B

## VARANASI, INDIA: BANARAS HINDU UNIVERSITY
### Department of Zoology

Wild Stocks

a) Canton-S
b) Oregon-R
c) Kerala

Chromosome 1

ClB
$X^{c-2}$
$w_i$
$w_a$
$w_{bl}$
$w_e$
$w_{co}$
$w_h$
$w^{82}$ - y $sc^{S1}$ B $In^{49}$ $ct^{ns}$ $sc^8$

Chromosome 2

vg

g49 - $dp^{tx1}$ Sp $ab^2$/$S^2_{tx1}$ ls Cy $In_2$Cy L
B - fes ms(b)cn sp/$dp^{tx1}$ $Cy^1$ $cn^2$
135 - S $fes_T$Sp ms ta cn mr crs/$dp^{tx1}$ $Cy^1$ $_2cn^2_4$
g67 - $ls_T dp^1$ Sp ms ta cn crs/$S^2$ Cy Bl $cn^2$ $L^4$ $sp^2$
g45 - $dp^1$ Sp cn bw sp/$S^2$(1st)Cy, In L cn bw sp CyD

Chromosome 3

se cu
seh
$ss^a$
$Ly/D_3$

Chromosome 4

$ey^2_w$
ci

Multichromosomal

fs 13 - $y^+_S$ $ac^+$ $sc^8$ Y/y B;$bw^D_L$;$st^+$ ♂ & y f:=;$bw^D$ ♀
j 102 - $Y^L$ X In EN In49 y Y;st (no free Y)

## MILANO, ITALY: UNIVERSITA' DI MILANO
### Istituto di Genetica

#### Wild Stocks

1 Berlino
2 Canton-S
3 Chieti-v
4 Crkwenica
5 Gaiano
6 Jaslo O.C.
7 Moltrasio
8 Oregon-R
9 Pavia
10 S. Maria
11 Sevelen
12 Suna
13 Urbana
14 Valdagno
15 Varese

#### Chromosome 1

16 $B$
17 $N^{B-S}$
18 $ptg^{2}$
19 sc ec ct v g f
20 sd
21 $v_{a}$
22 $w_{bl}$
23 $w_{e}$
24 w
25 y w

#### Chromosome 2

26 b cn
27 a px sp
28 ab
29 b cn vg
30 $blt_{S}$
31 $blt^{S}$
32 bsp
33 bw ba
34 c wt px
35 cn
36 cn c wt px
37 dp cl b
38 $ft_{2}$
39 $ll^{2}$
40 net
41 $so_{2}$
42 $so^{2}_{C}$ b cn
43 $So^{C}$
44 spt
45 $sp^{2}$ $bs^{2}$

#### Chromosome 3

46 $cp_{3}$
47 $gl^{3}$
48 mwh
49 obt
50 ru b st $p^{p}$ as $e^{s}$
51 ru
52 ve

#### Multichromosomal

53 $px^{43j}$ oo;ru jv se st ca
54 y;al bw sp

#### Not localized

55 tg (formerly $abab^{49}$)

#### Inversions on X

56 ClB/+
57 l(1)7/dl49 y Hw $m^{2}$ $g^{4}$
58 Muller-5
59 Muller-5/lozenge

#### Inversions on 2

60 Cy sp/Pm
61 Cy E-S/S
62 Cy $pr^{2}$/d b
63 Cy $cn^{2}$ bw sp/Gla In LR
64 Gla/spd gt-4

#### Inversions on 3

65 H/Sb sr In(3R) Mé
66 ltr/Sb sr In(3R) Mé
67 Mé ca/ru cu ca
68 ve h th C3 G Sb Ubx/st C3 G ca

#### Multichromosomal

69 Cy $L^{4}_{S1}$ sp/Pm;H/Sb sr In(3R)Mé
70 y $sc^{S1}$ In49 $sc^{8}$;bw;st $p^{p}$

#### Deficiencies

71 Df(1)N y/dl49 y Hw $m^{2}$ $g^{4}$
72 Df(2)$Px^{2}$ Df(2)Px, bw sp/SM1, $al^{2}$
73 Df(2)$bw^{5}$ Df(2)$bw^{5}$ $Cy_{5}sp^{2}$/Xa
74 Minute(2)Bridges
74 M(2)33a/$al^{2}$ In Mis Cy $cn^{2}$ $sp^{2}$

#### Special Stocks

76 "sz e" $Y^{Lc}$/X·$Y^{S}$ & y v f·=;e
77 "sz w" $Y^{Lc}$/$X^{w}$·$Y^{S}$
78 FMA 3/$w^{a}$ v; tra/In(3LR)Ubx
79 $y^{2}$ $Sn^{wa}$ $w^{a}$ bb·= & s f B·Y

#### Stocks selected for tumor manifestation

80 tu A1
81 tu B1
82 tu B3
83 tu C1
84 tu C2
85 tu C3
86 tu C4
87 tu C5
88 tu D
89 tu $So^{C}$
90 tu Aspra
91 Freckled/Curly
92 q 156 melanotic

## JERUSALEM, ISRAEL: HEBREW UNIVERSITY OF JERUSALEM
### Laboratory of Genetics

Stock list the same as listed in DIS 37:38 and 39:56

## MEXICO CITY, MEXICO: NATIONAL COMMISSION OF NUCLEAR ENERGY
### Laboratory of Genetics of Drosophila

**Wild Stocks**

a1 México
a2 Florida
a3 Oregon-R
a4 Canton-S

**Chromosome 1**

b1 B
b2 w m f
b3 m
b4 w
b5 y
b6 $y\ w^a$
b7 f
b8 v
b9 w m f/ClB
b10 sc cv v f
b11 sc cv v f B/y f:=
b12 $ma_3l$
b13 $lz^3$/y f:=
b15 y v
b16 $y_2w\ sn^3$
b17 $y^2$ v
b18 $y_2w^a$
b19 $y^2\ w^a$
b20 gt $w^a$
b22 y $w^a$ m f
b23 y pn w cm $ct^6$ $sn^3$ oc ras$^2$ v $dy^1$ $g^2$ f od car sw/y $sc^{S1}$ B In-49 v
b25 y cv v f car
b27 dow/FM6, $y^{31d}$ sc dm B

**Scute Alleles**

c1 y $sc^{s1}$ B f In-49 y & y f:=
c2 $y^{s1}$ $sc^8$ B f In-49 v

**Combinations of Similar Scute Inversions**

d1 M-5;$sc^{S1}$ B In-$S_8w^a$ $sc^8$
d2 $sc^{S1}$ In-S $w^a$ $sc^8$ (appears spontaneously in line M-5)

**Altered Y**

f2 y w $sn^3$;$sc^8_{m4}$ Y
f3 $sc^8$ Y/y w
f4 $y^2\ w^a/sc^8_3$ Y
f5 $sc^8$ Y/ac$^3$
f6 "new facl";$Y^{LC}$/In-49 ptg oc $B^{M1}$/$sc^{s1}$ $sn^{3.1}$ w $sc^8$ ♀ & $Y^{LC}_L$/y sn oc ptg v;$Y^S$ ♂
f7 sc·$Y^L$/y In-49 v f·Y;e

**Chromosome 2**

g1 bw
g3 cn
g4 dp
g5 $L^2$
g7 Cy/+

**Chromosome 3**

h1 se ss
h2 st
h3 e
h4 $se_2$
h5 $ry^2$
h7 ve h th
h8 $e^s$ $ca^{nd}$/TM1
h9 $e^s$ $ca^{nd}$/In(3RL)$ca^v$
h10 $e^s$ $ca^{nd}$/$ca^v$
h11 ss
h12 C3G
h14 ru st C3G $e^s$ (iso 3) (b pr)
h15 ru st C3G sr $e^s$

**Chromosome 4**

i1 $ey^2$

**Multiple Chromosomes**

j1 Cy/Pm;H/Sb
j2 $y^{s1}$ $sc^8$ In-49 $sc^8$;dp bw;st $p^p$
j3 bw;st
j4 y;bw;e;ci $ey^R$
j6 y f:=;bw;e;ci $ey^R$
j7 Cy/Pm;D/Sb
j8 $y^{s1}$ $sc^8$ B f In-49 v; $ey^2$

**Closed X**

k1 $X^c_{c2}$y/y f:=/$y^+$ Y
k2 $X^{c2}$, y v

**Attached (compound) Chromosomes**

m1 $Y^S$ X·$Y^L_2$ In(1)EN, $Y^s$ B y· $Y^L$/$y^2$ su-$w^a$ $w^a$ bb/O

**Special Cultures**

n1 sen $CO_2$ Sensible al $CO_2$, Collected in Mexico, D. F.

**Triploids**

t1 $y^2$ $sc^8_{sg}$ $w^a$ ec/$FM^4$, $y^{31d}$ dm B

## CAMBRIDGE, ENGLAND: UNIVERSITY OF CAMBRIDGE
### Department of Genetics, Milton Road

**Chromosome 1**

1 y f car

**Chromosome 2**

2 b cn vg
3 dp cn bw

**Chromosome 3**

4 se cp e
5 th cp
6 th st cp
7 ve h eyg cp

CHALFONT ST.GILES, BUCKS., ENGLAND: CHESTER BEATTY RESEARCH INSTITUTE
Institute of Cancer Research, Department of Genetics

Wild Stocks

1 Oregon K

Chromosome 1

2 acc
3 $amb^2$
4 bi amx ras/y v f:=
5 bis
6 $br^4$
7 brc
8 brc Oce cop smd
9 bz
10 ccw
11 ccw sma up bz
12 clm
13 cm ny un/y v f:=
14 crt
15 $dd^3$
16 dfw
17 dlv
18 dm rux if/y v f:=
19 dow/FM6
20 $drw^2$
21 dsh
22 $dvr^3$
23 dwg
24 ec cv m f Bx/y v f:=
25 ec dx
26 ff
27 fin
28 fla
29 g ty r sy/y v f:=
30 $gg^3$
31 hdp
32 kz sd od car/y v f:=
33 lac
34 lf
35 m f b z bb
36 mel
37 mgt
38 mk
39 mo
40 msc
41 $n^{fah}$
42 mo $n^{fah}$ mo
43 nrs
44 obl
45 Oce
46 omm
47 ot
48 peb na
49 pn shf oc ptg/y v f:=
50 pun
51 r (complementing series)
52 $re^2$
53 rea
54 ref
55 rg scp t sbr
56 rsi
57 rud
58 $sc^{Fah}$
59 sc ct wy g f car/y v f:=
60 sc v f car/y v f:=
61 sc v ClB/sc ct v f car
62 shm
63 sl
64 sla
65 slc
66 sld
67 slm
68 sma
69 smd
70 splw
71 spx
72 sta rb cx lz/y v f:=
73 sts
74 stt
75 svr vs sn/y v f:=
76 swb
77 ta
78 tc
79 thl
80 thv
81 tnb
82 trb
83 tw dy/y v f:=
84 Tu
85 Tu omm
86 unp
87 us
88 w
89 $w_t$ spl s
90 $w^t$ fw
91 $wa^2$
92 $ws^2$
93 ww
94 y ac fu
95 y fa dwx g pl

Chromosome 2

96 al dp b pr
97 Cy/Bl L
98 dp b cn bw
99 dp b pr stw (c) px sp
100 lt stw

Chromosome 3

101 $Dl^7/In(3LR)Ubx^{130}$ $Ubx^{130}$ $e^s$
102 Mé In(3L)cu sr $e^s$ ca/ "rucuca"
103 se cp e
104 st in ri $p^p$
105 tu $36^a$ st sr $e^s$ ro ca

Chromosome 4

106 ar/$ci^D$
107 $bt_{361}$
108 $ci^{361}$
109 $ey^2$
110 gvl
111 ltd
112 spa
113 spa pol
114 $sv^n$

Multichromosomal

115 bw;e;pol;(2;3;4)
116 dp;e;pol;(2;3;4)
117 y $sc^{S1}$ $In^{49}$ w $sc^8$; dp; e;pol;(1;2;3;4)
118 y v f:=;bw;e;pol;(1;2;3;4)
119 y v m f;bw;e;pol;(1;2;3;4)

Inversions

120 $sc^{S1}$ B InS $w^a$ $sc^8$
121 In(2LR)$Pm^2$/In(2LR)Gla
122 In(1)$sc^8$ $w^a$
123 In(1)$sc^8$ $w^a$;Cy/Bl L
124 In(1)$sc^8$ $w^a$;tu $36^a$ st sr $e^s$ ro ca
125 In(1)$sc^8$ y31d $w^a$
126 y $sc^{S1}$ $In^{49}$ $sc^8$
127 y $sc^{S1}$ $In^{49}$ $sc^8$;dp b cn bw
128 y $sc^{S1}$ $In^{49}$ w $sc^8$

Special Stocks

129 $Y^S$ X(FR1)$K^S$ y cv v f/RM y/$B^S$ Y $y^+$

Translocations

130 T(3;4)A 28, ve ca (homozygous)

## ANZYO, AICHI-KEN, JAPAN: NAGOYA UNIVERSITY
### Department of Animal Breeding

Wild stocks

1 Anzyo-Aichi
2 Chausuyama-Aichi
3 Hachijojima
4 Hikosan-Kyushu
5 Hiroshima
6 Hita-Kyushu
7 Onogo-Shikoku
8 Oregon
9 Suzuka-Mie
10 Yonekawa-Yamaguchi

Chromosome 1

11 Bx
12 f
13 $m_{58i}$
14 $m^{S1}$
15 $sc^{S1}$ B InS $w^a$ $sc^8$
16 $sc^{S1}$ B InS $w^a$ $sc^8$ $l^{(1)99}$ /y w m f
17 v
18 $w_{52l}$
19 $y^{52l}$
20 y w m
21 y w m f
22 y w m f/$y^{ClB}$

Chromosome 2

23 b
24 bw
25 bw(from population bell No.33)
26 bw vg
27 cn
28 Cy/bw (M)
29 Cy/bw (T)
30 Cy bw/bw
31 Cy/$l^{(2)50c}$
32 Cy bw/$l^{(2)50c}$
33 Cy/Pm
34 $dp^x$
35 $dp^v$ b
36 Pm b
37 Pm/$l^{(2)50c}$
38 $vg$
39 $vg^{Nw}$ Hia/T(2,3)$S^M$ Cy

Chromosome 3

40 cu
41 $e_{62h}$
42 $e^{62h}$
43 Sb

Multichromosomal

44 Cy/$l^{(2)50c}_{(2)50c}$;Sb
45 Cy/$l^{(2)50c}$;Sb cu/cu
46 Pm/$l^{(2)50c}$;cu
47 v;bw

Unanalyzed

48 Dichaete like
50 brown like
51 Jaunty like

## NAPLES, ITALY: UNIVERSITY OF NAPLES
### Instituto di Biologia Generale e Genetica

Wild Stocks

Bisignano
Canton
Lecce
Oregon-R
Pavia
Roma
Sciolze

Chromosome 1

B
Muller-5
v
w
$w^a$
$w^{bf}$
$w^{bl}$
$w^{co}$
$w^i$
$w^t$
y

Chromosome 2

b
b cn vg
bw
cl
cn
cn bw
Cy/Pm
pr
vg

Chromosome 3

cd
Sb/+
Sb/In(3R)l(3)Na
se
st

Multichromosomal

w;vg

## CHANDIGARH, PUNJAB, INDIA: PUNJAB UNIVERSITY
### Department of Zoology

D. melanogaster (4 strains)

## CALCUTTA, INDIA: UNIVERSITY OF CALCUTTA
### Department of Zoology

Wild Stocks

a1 Canton-S
a2 Oregon-R
a3 P Ceylon
a4 Nai-C

Chromosome 1

b1 y
b2 $w_a$
b3 $w_{bo}$
b4 $w_{co}$
b5 $w_e$
b6 $w_h$
b7 $w_i$
b8 $w^1$
b9 B
b10 sx $vb^2$ sy/FM4(Y)
b11 y $ac_1$ $sn^3$ sx $vb^2$ $sy^a$/y $sc^8$ $sc^1$ In $dl^s$ B v $w^8$
b12 $w^{vc}$/y w $lz^s$ dl49/$sc^8$ Y.(Floating Y)

Chromosome 2

c1 vg
c2 bw
c3 cg c/U
c4 pr en

Chromosome 3

d1 se h
d4 $e^s$
d5 th st pc Scx $p^p$ ss/ TM1, Mé ri
d6 D/dsx red
d7 tra/In(3LR)$Ubx^{130}$(FMA3)
d8 red e

Chromosome 4

e1 $ey^2$
e2 $ci^w$
e3 M-4/$ey^D$

Multichromosomal

f1 y;D/tra red
f2 3N $y^2$ sc $w^a$ ec/FM4 $sc^8$ y;cn/cn/cn

## ADELAIDE, SOUTH AUSTRALIA: UNIVERSITY OF ADELAIDE
### Department of Genetics

Wild stocks

1 Canton-S

Chromosome 1

2 B
3 car
4 ct v f
5 $g^2$
6 Basc
7 rb cx
8 sc cv f
9 sc cv v f
10 sd
11 v
12 $v^{61j}$
13 w
14 $w^{a55b}$
15 $w^{sat}$
16 w ct f
17 w m f
18 y
19 y w spl
20 y $w^a$ sc ec
21 y/$lz^{57j}$
22 $X^{c2}/sc^{S1}$
23 X $y^S/Y^{LC}$
24 $vs^{64j}$

Chromosome 2

25 al
26 al dp b pr c px sp/Cy
27 b j
28 b vg
29 bw
30 cn
31 dp
32 dp b j
33 fj wt/Xa T(2;3)
34 ho
35 vg
36 S/Cy En-S
37 S Gp Bl $bw^D$/Cy $cn^2$ lc
38 Sp J/SM5 $al^2$ Cy $lt^v$ $sp^2$

Chromosome 3

39 ca
40 $e^4$
41 $e^4$ wo ro
42 Ly/$D^3$
43 R Ly/In(3L) P, gm
44 ss
45 ss $e^4$ ro
46 st

Chromosome 4

47 ci $ey^R$
48 $ey^2$

Multichromosomal

49 Basc;Cy $ds^{33k}$/Pm;H/Sb
50 y;Cy $ds^{33k}$/Pm;H/Sb
51 v;bw
52 y w;dp
53 bw;$e^4$
54 bw;st
55 dp;$e^4$
56 vg;$e^4$

## ROMA, ITALY: CITTÀ UNIVERSITARIA
### Istituto di Genetica

#### Wild Stocks

A1 Oregon
A2 Marzi

#### Chromosome 1

B1 car bb
B2 pn
B3 $r^R$/y f:=
B4 sc cv v f B/y f:=
B5 sc z ec
B6 sw
B7 w
B8 $w^a$
B9 $w^{Bwx}$
B10 $w^{cf}$
B11 $w^{cp}$
B12 $w^r$
B13 $w^{cf}$/y f:=
B14 $w^e$ dy/y f:=
B15 en $w^e$/y f:=
B16 y
B17 y $l^{59}$
B18 y ac sc pn/y f:=
B19 y cv v g/y f:=
B20 $y\ fa^n\ sn^3$
B21 $y^2$ v ma-1
B22 $y^2\ w^{cf}$
B23 $y^{31d}\ v^{0f}\ w^a$ f sn/y f:=
B24 y $l^{259}$/s-5/$sc^8$·Y

#### Chromosome 2

C1 b cn c bw
C2 b cn vg bw
C3 Bl L/Cy
C4 Bl $L^2$/SM5, $al^2$ Cy $lt^v\ sp^2$
C5 $bw_D$
C6 $bw^D$
C7 cn bw
C8 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
D5 se ss k $e^s$ ro

#### Chromosome 4

E1 ci $ey^R$
E2 pol

#### Multichromosomal

F1 bw;st (2;3)
F2 Cy/Pm;Sb/Ubx (2;3)
F3 sc cv v f B;ci $ey^R$ (1;4)
F4 y;pol (1;4)
F5 y;ru h th st $p^p$ cu sr $e^s$ (1;3)
F6 $y^2$ cho;lys rc (1;2)
F7 y;bw;st (1;2;3)
F8 al $L^4$ Cy sp/Pm;H/Sb sr In(3R)Mé (2;3)

#### Translocations

H1 T(1;4)$B^S$(16 A 1), $y^2$ cv v $B^S$ car/y f:=

#### Inversions

I1 In(1)dl-49, w lz
I2 In(1)dl-49, y Hw $m^2$/fa $N^{22a}$
I3 In(1)dl-49, y Hw $m^2$ $g^4$/ Df(1)$N^8$
I4 In(1)$sc^{4L}$, $sc^{8R}$, y $sc^{4+8}$ cv v f/y f:=
I5 In(1)$sc^7$, AM
I6 In(1)$sc^8$, dl-49, $y^{31d}$ $w^a$
I7 In(1)$w^{m4}$/Df(Y)$y^{bb}$
I8 In(1)$w^{m4L}$, $rst^{3R}$, y $w^-$ $rst^3$/ Dp(1;2R)$w^{51b7}$/y w f:=
I9 In(1)481(12-F;14B), y $bb_1{}^{481}$/ FM6, $y^{31d}$ $sc^8$ dm B/$sc^8$·Y

#### Deficiencies and Duplications

L1 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$^-$·$Y^L$/RA,(ND-27)v f/Y"
M2 X·$Y^S$(A-3), sc cv v·$Y^S$/y v f/ $y^{Lc}$
M3 $Y^S$X·(FR-1),$Y^S$ y cv v f/ y f:=/Y
M4 $Y^S$X·(P-7), In(1)EN, $Y^S$ y f/ y v f/Y

#### Attached XY Chromosomes

N1 $XY^L$·$Y^S$(108-9), $y^2$ su-$w^a$ $w^a$ $Y^L$·$Y^S$/y v bb/0
N2 $XY^S$·$Y^L$(115-9), $y^2$ su-$w^a$ $w^a$ $Y^S$·$Y^L$ $y^+$/y v bb/0
N3 $XY^S$·$Y^L$(129-16), $y^2$ su-$w^a$ $w^a$ $Y^S$·$Y^L$ $y^+$/y v bb/0
N4 $Y^S$X·$Y^L$, Ins(1)EN, dl-49, $Y^S$ car f v y·$Y^L$
N5 $Y^S$X·$Y^L$, Ins(1)EN, dl-49, $Y^S$ car f v y·$Y^L$/$y^2$ su-$w^a$ $w^a$ bb/0

#### Altered Y Chromosomes

P1 $Y^{Su-Var}$ In(1)$w^{m4}$ w
P2 Y $w^+$ (Y 900)/y $w^a$
P3 Y $w^+$ (Y 303)/y $w^a$
P4 $y^+$ Y/y $w^a$
P5 $B^S$ Y/y f:=/$y^2$ su-$w^a$ $w^a$ bb
P6 $y^+$ Y $w^+$ (11a)/y $w^a$

## KWANGJU, KOREA: CHUNNAM NATIONAL UNIVERSITY
### Department of Biology

Stock list unchanged. See DIS 40:21

## LOUVAIN, BELGIUM: THE UNIVERSITY
### Janssens Laboratory for Genetics

Inbred Temperature Lines

1 line raised at 25°C for 112 generations (Abeele - Belgium)
3 lines raised at 18°C for 93 generations (Gabarros - Spain)
2 lines raised at 25°C for 225 generations
4 lines raised at 18°C for 117-127 generations
These two last items came originally from Dept. of Botany and Plant Pathology, Pennsylvania State University (see DIS 34).

**Wild Stocks**

Abeele (Belgium)
Canton-S
Gabarros (Spain)
Oregon
Swedish-B
Watou (Belgium)

**Chromosome 1**

B
Basc
w

**Chromosome 2**

bw
ho
sca
$sp^2$ $bl^2$
$stw^2$
vg

**Chromosome 3**

bv
ca
cp in ri $p^p$
e

jv
ri
ro
ro ve
ru h th st cu sr $e^s$ ca (rucuca)
ve jv h $H^n$ th st cu sr $e^s$ ro ca (vecuroca)
ve jv h $H^n$

**Multichromosomal**

Basc;ri e
$ri;stw^2$
w;ri

## BAYFORDBURY, HERTFORD, ENGLAND: JOHN INNES INSTITUTE

**Wild Stocks**

1 Bayfordbury
2 Hampton Hill
3 Samarkand
4 Teddington

**Inbred Lines**

5 Bayfordbury (A)
6 Bayfordbury (B)
7 Oregon (v marker)
8 Samarkand
9 b pr

**Chromosome 1**

10 v
11 w
12 yw

**Chromosome 2**

13 b pr

**Multichromosomal**

14 Cy $L^4$/Pm;H/Sb

## THESSALONIKI, GREECE: UNIVERSITY OF THESSALONIKI
### Department of Biology

**Wild Stocks**

Oregon-K
Berlin wild

**Chromosome 1**

y
$pn^2$
w

$w^e_{bf}$
$w^n$
fa
sn
v
m
f
Muller-5
XXY

**Chromosome 2**

Cy
dp
b
pr
cn
vg
bat
bw

bs
Cy $L^4$/Pm

**Chromosome 3**

ve
jv
se
st

cu
ry
ss
e
Bd

**Chromosome 4**

ey

## JOHANNESBURG, SOUTH AFRICA: UNIVERSITY OF THE WITWATERSRAND
### Department of Zoology

### Wild Stocks

1 Bethulie
2 Bloemfontein
3 Canton-S
4 CapeTown
5 Cedara
6 Drakensberg
7 Florida
8 Graaff-Reinet
9 Grahamstown
10 Inhaca Island
11 Johannesburg
12 Kalahari
13 Kariba Dam
14 Limpopo
15 Magaliesberg
16 Nelspruit
17 Nyasa Lake
18 Oregon-R
19 Stanford Lake
20 Stellenbosch
21 Tzaneen
22 Umgazi River
23 West Rand
24 Zoutpansberg

### Chromosome 1

25 B
26 $Bx^3$
27 bo
28 $car_2$
29 $car^2$
30 cm
31 cm car
32 cm $g^3$ car
33 ct v
34 $ct_6$ v dy g
35 $ct^6$
36 cv ct
37 cv sc
38 ec
39 f
40 $f_5$ BB
41 $f^5$ m
42 $f^5$ v
43 $g_2$
44 $g^2$
45 $g_3^3$
46 $g^3$ f
47 m $f_2$
48 $ras^2$
49 rb
50 rb cm $g^3$
51 rb cm car
52 rb cx
53 rb $g_3^3$
54 rb $g^3$ car
55 sc ec cv $ct_6^6$ v
56 sc ec cv $ct^6$ $v_{31d}$ $g^2$ $g^8$ f/FM3 $y^{sc^a}$ dm B l
57 svr $w^a$
58 $v_{36f}$
59 $v^{36f}$
60 w
61 w m
62 w m f
63 $w_a$
64 $w_{a2}$
65 $w_{a3}$
66 $w_{a4}$
67 $w_{bf}$ $rb_5$
68 $w_{Bwx}$ $f^5$
69 $w_{bl}$
70 $w_{ch}$
71 $w_{co}$
72 $w_{col}$ $sn^2$
73 $w_e$
74 $w_{e2}$
75 $w_{e3}$
76 $w_e$ $g^3$
77 $w_h$ rb car
78 $w_{sat}$
79 $w_t$
80 $w^w$ $fw^5$
81 $w^w$ $f^5$
82 $w^w$ rb
83 y $_4$
84 y $g^4$
85 y m
86 y pn
87 y w
88 $y_2$ w m
89 $y_2$ $su\text{-}w^a$ $w^a$ bb
90 $y^2$ $w^a$ w
91 z $w^{E4}$

### Chromosome 2

92 al dp b pr Bl $c_2$ px sp/SM1 $al^2$ Cy $sp^2$
93 a $sp^2$
94 b
95 b pr cn
96 b pr cn a
97 $bw_{2b}$
98 $bw_D$
99 $bw^D$
100 c
101 c px
102 $cn_{35k}$
103 $cn^{35k}$
104 $cl_{50a}$
105 $cl^{50a}$
106 dke c
107 dp
108 lt std/Cy $sp^2$
109 ltd
110 net
111 pd
112 $pr_{42d}$
113 pr
114 $px_2$
115 $sf^2$
116 sp
117 Su-H/Cy, pr
118 tk $sf^2$ abb
119 $vg_{dn}$
120 $vg_{ni}$
121 $vg_{np}$
122 vg

### Chromosome 3

123 ca
124 cd
125 cu kar
126 cp
127 D/Gl
128 $e_s$
129 $e^s$
130 gl
131 ma fl
132 mah
133 p
134 $p^p$ cu
135 $p^p$ cu sr $e^s$
136 res
137 ru
138 ry
139 se
140 ss bx
141 ss bx $su^2$-ss
142 $st^{sp}$
143 $su^B$-pr/In(3R)C, e;pr
144 th st
145 th st $p^p$

### Chromosome 4

146 bt

### Multichromosomal

147 bw; ci ey
148 bw; st
149 Cy/Pm, $ds^{33k}$;H/In(3R)Mo;sr
150 $g_3^3$;bw
151 $g_3^3$;st
152 $g^3$;st $p^p$
153 $ras^2$;st
154 rb;bw
155 car;se
156 vg;se
157 $w^e$ rb;se
158 $w^w$;cd
159 y;bw;e;ci ey

### Attached-X

160 f B/$su^{S2}$-v-pr v
161 y/+
162 $\frac{y^2\ su\text{-}w^a\ w^a\ bb/}{sc^{4L}\ sc^{8R}}$

### Closed-X

163 $X^{c2}$, cv v f/ClB

Deficiencies

165 Df(1)$w^{258-11}$

Inversions

166 In(1)A99b
167 In(1)dl-49, y $fa^n$
168 In(1)$rst^3$, y $rst^3$ car bb
169 In(1)$w^{m4}$
170 In(1)$w^{m4}$;st
171 In(1)$sc^{8}$, $S_a$, $sc^{8R}$ $sc^{S2}$ $sc^{8}$ w B

Translocations

172 T(1;3)04/ClB
173 T(1;4)$w^{m5}$

## HELSINKI, FINLAND: UNIVERSITY OF HELSINKI
### Institute of Genetics

Wild Stocks

3 Haaga
4 Oregon-K
5 Oregon-R-S
6 Porvoo
7 Swedish-b

Chromosome 1

8 B
10 bi $ct^6$ $g^2$
10a ec ct v f
11 f
12 fu/ClB
13 $g^2$ f B & y
14 In(1)dl-49, y $fa^n$
16 In(1)$sc^4$, y $sc^4$
17 In(1)$w^{m4}$
20 $ras^2$
21 rb cx
22 s
23 sc cv v f
24 sd (;se)
25 $sn^3$
26 spl
27 w
28 $w^e$ sn/ClB
29 $w^{ch}$ wy
30 y ac v
31 y In(1)dl-49 f car & y f:=
32 y $sn^3$ bb
33 y v f
34 z

Chromosome Y

35 $X{\cdot}Y^L/Y^S$
36 In(1)$w^{m4}$ & extra Y
38 In(1)$w^{m4}$;rl & extra Y

Chromosome 2

39 al dp b pr c px sp
42 Bl $L^2$/Cy
43 $cn^2$ InCyR cg $sp^2$/Ins NS px sp
44 $dp^T$ $ab^2$ pr Bl rn NSR mr/$al^2$ Cy $cn^2$ $L^4$ $sp^2$
45 $dp^{tx}$ Sp $cn^2/S^2$ Cy $cn^2$ (homoz. InCyR)
46 ex
47 fj
48 fj px
50 Ns, b mr/Cy
51 rl
52 rl
53 $stw^2$
54 vg

Chromosome 3

55 $Bd^G$/In(3R)C, l(3)a
56 $D^3$/Payne
58 $e^{11}$
59 Gl Sb/LVM
60 In(3R)$Dl^B$, st $Dl^B$/In (3R)$P^W$, st l(3)W ca
61 In(3R)$P^{FLA}$ (homozygous)
62 Ly Sb/LVM
63 Mé, InL Sb/ru h D InsCXF
64 R Ly/In(3L)P, gm
65 se
66 se app
67 se $rt^2$ th/Mé, InL
69 tra/Mé, T23
70 W Sb/InsCXF

Chromosome 4

71 $ci^W$
72 spa

Multichromosomal

74 Cy/Pm;D/Sb
75 vg;e
76 $w^{m4}$;Cy/$ap^4$ vg
77 $w^{m4}$;Cy/blt

Deficiencies

78 Df(2)MS-4/SM1, $al^2$ Cy $sp^2$
79 Df(2)MS-8/SM1, $al^2$ Cy $sp^2$
80 Df(2)MS-10/SM1, $al^2$ Cy $sp^2$
81 Df(2)$rl^{10a}$ lt cn/Cy
82 Df(2)$rl^{10a}$ lt cn/Pm, $al^4$ $ds^{33k}$ $lt^-$ $bw^{V1}$

Translocations

84 T(2;3)rn/Cy
85 T(2;3)rn/Cy $sp^2$
86 T(2;3)rn/Cy $cn^2$ $sp^2$
87 T(2;3)rn/Cy Bl $cn^2$ $L^4$ $sp^2$
88 T(2;3)rn In(2R)M/Cy $cn^2$ $sp^2$
89 T(2;3)rn $L^2$/Cy
91 T(2;3)Xa/Sb Ubx

## LYON, FRANCE: UNIVERSITÉ DE LYON
### Faculté des Sciences, Zoologie Expérimental

Wild Stocks Oregon R Lyon Algérie

## LIVERPOOL, ENGLAND: UNIVERSITY OF LIVERPOOL
### Department of Genetics

Chromosome 1

w sn m
B
$y\ w$
$w^a$

w m f
ClB/w m f
$w^a \hat{} w^a$

Chromosome 2

dp b cn c bw

dp cn bw
b cn vg
Cy $L^4$/Pm
Cy $L^4$/d b

Chromosome 3

e
se cp e

Multichromosomal

y;bw;st
$y\hat{y}$;bw;st
y;Cy $L^4$/Pm;st
M-5;Ly/Mé

## CHIBA-SHI, JAPAN: NATIONAL INSTITUTE OF RADIOLOGICAL SCIENCES
### Division of Genetics

Wild stocks

1 Oregon-R
2 Samarkand
3 Tokyo

Chromosome 1

4 B
5 ec ct$^6$ $g^2$ $bb^1$/ClB
6 $sc^{S1}$ B Ins $w^a$ $sc^8$
7 v
8 v f B XY/$y^2$ su-$w^a$ $w^a$ bb
9 w
10 y w m f

Chromosome 2

11 cn bw
12 Cy/Pm
13 vg

Chromosome 3

14 $e^{11}$
15 se
16 ss
17 st

Chromosome 4

18 $ci^D$/Cat

Multichromosomal

19 $sc^{S1}$ B Ins $w^a$ $sc^8$;Cy/Pm;Sb/Ubx
20 $X^{c2}$ v & y:= (bw;e;ey)
21 y $sc^{S1}$ In49 $sc^8$; bw;st $p^p$
22 $sc^8$ Y/y In 49 B & y f:=;$bw^D$
23 $sc^8$ Y/y B & y f:=

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

Wild Stocks

Oregon-K
Madras

Chromosome 1

1 $sc^{S1}$ B InS $w^a$ $sc^8$
2 w

Chromosome 2

3 Cy Bl $L^2$
4 dp b cn bw
5 vg

Chromosome 3

6 e

Attached-X

7 $sc^{S1}$ B InS $w^a$ $sc^8$ &
y v f:=

Multichromosomal

8 bw;st
9 y $sc^{S1}$ In49 sc;bw;st

## SANTIAGO, CHILE: UNIVERSIDAD DE CHILE, DEPARTAMENTO DE GENETICA
### Instituto de Biología "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

## BLINDERN, NORWAY: UNIVERSITY OF OSLO
### Institute of General Genetics

The stock list remains essentially the same as that appearing in DIS 40:27, with this change:

Removed from List

58 ma fl

## MANCHESTER, ENGLAND: CHRISTIE HOSPITAL & HOLT RADIUM INSTITUTE
### Paterson Laboratories

Wild Stock

Birmingham

Inbred Lines (no. generations)

Oregon v (265)
b pr (165)

Chromosome 1

v/y v f car:=
Muller-5
sc ec cv ct vg/In(1)dl49, y Hw $m^2$ $g^4$
sc v f car/Cl B
sc ec cv
ct v g
$y_2$ cv v f
$y_2$ sc car·Dp $sc^{v1}$, $y^+$
$y^2$ sc $v_8$ f·Dp $sc^{v1}$ $_{B1d}$ $sc^8$ $y_a/B^S$·Y
In(1)$sc_8$, $y^8$ $sc^8$ w
In(1)$sc_8$, $sc_8$ v f car
In(1)$sc^8$, $sc^8$ cy v f/In(1) dl49, y Hw $m^2$ $g^4$

Chromosome 2

b pr vg
$dp_{Th}$ b cn bw
$dp^{Th}$ Cy cn bw/S Sp cn bw
al dp b pr c px sp/Cy
2L dp;2R px

Chromosome 3

3L $rs^2$ $h^2$;3R
3L ri;3R sr

Multiple Stocks

dp;$e_4$
$Cy_{VA}L^4$/Pm;H/Sb
bw $^{VA}$/Bl $L^2$;st
v;bw $^{VA}$/Bl $L^2$
$y_2$/FM6 or $sc^8$·Y;SM 1/+
$y^2$ AM/FM6 or $y^+$·Y;SM1/Bl; Vbx $^{130}$/Vno;pol

## LONDON, ENGLAND: UNIVERSITY COLLEGE LONDON
### Department of Zoology

**Wild Stocks**

Kenya (inversion on X)
Oregon

**Chromosome 1**

BB$^{3}$
B$^{8}$
N/dl49 y Hw m$^{2}$ g$^{4}$

ras f
v
w$_{a}$
w$_{bl}$ m
w$_{e2}$
w$_{m4}$
w
w sin B
y & oc$_{bl}$ pt y pr shif
y & w wy f

**Chromosome 2**

bw
cn
cn bw
ho
Pt/Cy
vg

**Chromosome 3**

Mc/Sb$_{a}$
Sb ss
ss$_{a}$
ss
st
ve
ve ss$_{a}$

**Multichromosomal**

v;bw

## STRASBOURG (BAS-RHIN), FRANCE: UNIVERSITÉ DE STRASBOURG
### Faculte des Sciences, Laboratoire de Zoologie

**Wild Stocks**

Oregon-R-C

**Chromosome 1**

B
v
w
y

**Chromosome 2**

cn
b

**Chromosome 3**

DcxF/Dfd
e
se

**Chromosome 4**

ey

**Multichromosomal**

vg;cn
w;e
y;e
y;se
Cy/Pm;H/SbC

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

**Wild Stocks**

a1 Calcutta University (originally from Oregon Stock)
a2 Kerala
a3 Guptipara (W. Bengal)

**Chromosome 1**

b1 y
b2 w
b3 B

**Chromosome 2**

c1 vg

**Chromosome 3**

d1 se h
d2 ss$^{a}$

**Chromosome 4**

e1 ey$^{2}$
e2 ci$^{w}$

## CLERMONT-FERRAND, FRANCE: M. HOVASSE LABORATOIRE DE ZOOLOGIE ET BIOLOGIE GENERALE
### Faculte des Sciences, Laboratoire de Zoologie

Oregon-R-C vg w e B

## SEOUL, KOREA: YONSEI UNIVERSITY
### Department of Biology

Note: Stocks presently maintained unchanged from DIS 37:42 with the following exceptions:

**Lost:** L
D/Gl
th

**Added:** st in ri $p^p$
Bxl/Payne, Dfd ca

## DUNEDIN, NEW ZEALAND: UNIVERSITY OF OTAGO
### Department of Botany

**Wild Stocks**

1 Oregon R-C

**Chromosome 1**

2 B
3 B m
4 f
5 m
6 v
7 w
8 $w_a$ m
9 $w^a$
10 $w_a^a$ m
11 $w^a$ m f
12 y sc m f
13 Muller-5
14 In(1)$w^a$ f

**Chromosome 2**

15 al dp b pr c px sp
16 bw
17 bw cn
18 bw vg
19 cn
20 cn dp
21 dp
22 vg

**Chromosome 3**

23 cp
24 cu
25 cu e
26 cu se
27 e
28 e se
29 e se cu
30 $e^s$
31 Gl Sb/LVM
32 ru h th st cu sr $e^s$ ca
33 se
34 st

**Multichromosomal**

35 v; bw
36 bw; st
37 Cy/Pm; D/Sb
38 vg; se

**Translocations**

39 T(1;2)Bld, Bld/ClB (carries In(2R)Cy)

## PÔRTO ALEGRE, BRAZIL: UNIVERSIDADE DO RIO GRANDE DO SUL
### Laboratório de Genética Animal

**Wild Stocks**

Eldorado (Rio Grande do Sul)

**Chromosome 1**

w
$w^h$
$w^{bl}$
$w^e$
v
y v
sc cv v f

**Chromosome 2**

cn
vg
L
dp
st/bw

**Chromosome 3**

p
se
e

## PRAGUE, CZECHOSLOVAKIA: INSTITUTE OF EXPERIMENTAL BOTANY
### Department of Plant Physiology and Genetics

**Wild Stocks**

Suchumi
Oregon-K
Hikone-r

**Chromosome 1**

y
Basc
ClB/w
$sc^8$ y $B^S$
y $sc^{S1}$ $In^{49}$ w $sc^8$
XXy v f/sc ct v f car

**Chromosome 2**

cn vg b
dp b cn bw

**Chromosome 3**

Cy/Bl e
$L^4$

**Multichromosomal**

bw e pol

NEW MUTANTS

Report of C. Tokunaga

Msc: Multiple sex comb Tokunaga, 64a 3-48.0. Spontaneous. One male with strongly expressed multiple sex comb from a line selected for - usually weak - heterozygous expression of the second chromosome mutant esc.

Msc causes the differentiation of extra sex combs on the basitarsi of second and third legs as well as the differentiation of transverse bristle rows between the longitudinal rows 6 and 8 on each basitarsus and the distal part of the tibia as described in other extra sex comb genes. A marked difference between the action of Msc and the other extra sex comb genes is that Msc affects sex comb differentiation of the first leg. As shown in Table 1, the average sex comb teeth number of the first leg of Msc is markedly decreased, and the relative length of the basitarsus is greater than in wild type or esc or $esc^D$ males. Although the absolute numbers of teeth in second and third legs vary with the genetic background, the greater similarity between second and third leg sex combs in Msc in contrast to the strong gradient in esc/esc and $esc^D$/+ seems to be a constant feature. As the standard errors in Table 1a show the expression of Msc in the second and third leg sex combs is less variable than in the other extra sex comb types.

Table 1.--Parts (a) and (b) are based on the same sample of flies. The gene $esc^D$ was formally called $Esc^2$ (DIS 38:80).

a. Number of teeth in sex comb.

| | 1st leg | 2nd leg | 3rd leg | Number of legs |
|---|---|---|---|---|
| Oregon R.C. | 10.75±0.78 | 0 | 0 | 20 (20 ♂♂ left) |
| y, esc/esc | 10.9 ±0.81 | 6.3±2.26 | 2.3±2.05 | 40 (20 ♂♂) |
| y ac, $esc^D$/+ | 11.6 ±1.23 | 5.1±2.03 | 2.9±2.31 | 40 (20 ♂♂) |
| b, Msc/+ | 5.9 ±0.75 | 4.1±0.97 | 3.4±1.28 | 40 (20 ♂♂) |

b. Length of basitarsus and ratio of lengths.

| | 1st leg | 2nd leg | II/I | 3rd leg | III/I |
|---|---|---|---|---|---|
| Oregon R.C. | 28.2±0.5 | 44.4±0.7 | 1.57 | 48.3±0.8 | 1.71 |
| y, esc/esc | 24.9±1.0 | 39.1±2.0 | 1.57 | 42.5±1.6 | 1.71 |
| y ac, $esc^D$/+ | 28.5±1.2 | 42.8±2.7 | 1.50 | 49.9±2.3 | 1.75 |
| b, Msc/+ | 29.3±1.3 | 40.2±3.1 | 1.37 | 44.1±3.7 | 1.50 |

The following data (Table 2) indicate that Msc is dominant and homozygous lethal

| Genotype of Parents | Number of Pair Matings | $F_1$ phenotype ♀ | $F_1$ phenotype ♂ |
|---|---|---|---|
| Sb/Msc | 1 | Sb: 99 | Sb, Msc: 104 |
| Pm/+, Sb/st cp Msc | 2 | Pm, Sb: 30; Sb: 21 | Pm, Sb: 21; Msc: 24 |
| Pm/+, Sb/Msc $bx^{34e}$ | 6 | Sb: 170; bx: 1* | Sb, Msc: 159; Sb: 4** |

* Presumably crossovers between Msc and Sb. ** Presumably low penetrance of Msc.

In general, the penetrance of Msc is very high in comparison with other extra sex comb genes (see also Table 1a). The penetrance of Msc is markedly lowered when combined with Dichaete as illustrated by the $F_1$ of a pair mating between a Sb/D ♀ and a Msc/+♂. The phenotype of $F_1$ males were: Sb: 42; Sb,Msc: 38;D: 71; D, Msc: 3. There are indications that the penetrance of Msc can also be changed by environmental conditions.

Linkage tests with the third chromosome markers, st in ri $p^p$ and ss, indicate that Msc is located between ri and p, very close to p.

Analysis of salivary gland chromosomes indicates that Msc is associated with a small inversion from 84B to 84F.

Report of A. Chovnick and J. Talsma

db: dark body 3-44.4. First occurred as three dark bodied, Bar eyed daughters from the cross: Basc/ma-$1^{bz}$ x $1^{J1}$/$sc^{8}$ Y. Homozygous lethal in males with death occurring in late pupae. Rare male survivors are dark bodied and do reproduce.

Report of R. M. Valencia

The following are mutations (all but one of which are associated with rearrangements) found in a series of experiments carried out by Dr. H. J. Muller, J. I. Valencia and R. M. Valencia at Indiana University during the years 1946-1953, in which mutations were recovered at specific loci in the X chromosome. Since I have acquired these cases and they appear in my present stock list, I think that the information pertaining to them (even though incomplete in some cases) should be made available. Salivary analyses were made by J. I. Valencia. These have not been re-checked recently, but the stocks are phenotypically as expected.

$w^{40aH1}$: white. Induced by X-rays (5000r) in an X chromosome bearing a Dp for $y^{+}$ $ac^{+}$ attached to the right arm ($sc^{V1R}$). Multiple rearrangement with breaks in 4C4, 17B7-8, 18E2-3 and the Dp tip. Lethal.

$ct^{43aH1}$: cut. Same treatment and chromosome as above. Two inversions, 4B4 to 7B3-4 and 10D6-7 to 20B or C. Lethal.

$rb^{mott\ 48aH5}$: ruby-mottled. Same treatment and chromosome as above. Deletion 3E3-4 to 11A7-8 transposed in inverted order to XR.

$g^{17Ba6}$: garnet. Induced by hard X-rays (dose?) in a "Binsc" X chromosome. Inversion 12B15 to 19F. Male sterile.

$ras\text{-}v^{17Cc8}$: raspberry-vermilion. Same treatment and chromosome as above. Deficiency for 9E4 to 10A5-6.

$sn^{I9Bb5}$: singed. Same treatment and chromosome as above. 3C2 to 7C9 inserted in 3L 72A-B. Locus of white not included.

$pn^{I10Ac4}$: prune. Same treatment and chromosome as above. Deficiency 2C9-10 to 3A2-3.

$cm^{R8aH4}$: carmine. Induced by X-rays (?) in a ring X ($X^{c2}$). Deficiency for 6E.

$sn^{R13aH1}$: singed. Same treatment and chromosome as above. 6C to 7C8-9 inserted in chromosome 3.

$rb^{R15BH3}$: ruby. Deficiency 4B3-4 to 4D4-5.

$sc^{S1L}$ $ct^{\varphi 15B4}$: cut. Induced by X-rays (4000r) in developing oocyte of "jynd" female, in y B d1-49 $sn^{X2}$ $sc^{8R}$ X chromosome. Non-lethal.

Report of M. J. Mayo

$vs^{64j}$: vesiculated-64 Sex linked recessive X-ray induced in Canton-S sperm. One or both wings crumpled and only partially expanded, occasionally blistered but not otherwise affected. Expression variable and penetrance incomplete (about 77%). No difference between sexes. Similar to vs in appearance and position but not tested for allelism.

Report of D. J. Komma

L-a$^{Zw}$ Low activity-auxocompensation for G-6-PD Chromosome 1. Conceivably more than one mutant. Hemizygous males and heterozygous females have about 50% of normal activity of glucose-6-phosphate dehydrogenase, homozygous females about 30%. Not an operator gene mutation, as enzyme production by both chromosomes is reduced in heterozygotes. Effect is suppressed in females homozygous for transformed, suggesting that the L-a$^{Zw}$ locus may be part of the mechanism of dosage compensation for G-6-PD. Has been found on the same chromosome with both the ZwA and ZwB structural alleles of G-6-PD. Seems to be quite common, as it has been found in several different inbred stocks.

Report of J. B. Courtright

Octanol dehydrogenase: a mutant for dehydrogenases of six, seven and eight carbon primary alcohols in Drosophila. Two types of Drosophila strains have recently been reported that differ in the electrophoretic mobility of alcohol dehydrogenase (ADH) isozymes when ethanol is used as the substrate in the staining mixture (Nature 204:906, 1964; Science 149: 80, 1965; J. Exp. Zool, in press). The locus responsible for this variant ADH pattern was mapped on the second chromosome (Science 149:80, 1965).

When octanol was used as the substrate, a mass homogenate of OreR flies was found to contain, in addition to the seven ADH bands, three "octanol dehydrogenase" (ODH) bands which migrate less cathodally than ADH (J. Exp. Zool., in press). Single fly electrophoresis showed that the OreR stock consists of three types of flies: individuals containing one ODH band only, migrating "fast" cathodally (F), individuals containing a single band migrating more slowly to the cathode (S), and imdividuals containing three bands, one slow, one fast, and one intermediate.

We have now localized the gene responsible for the ODH variant pattern on the third chromosome. Reciprocal crosses had shown that the ODH trait is not sex-linked. When single OreR flies (F) were crossed with Cy/Pm;Sb/Ubx (S) flies, all $F_1$ individuals had three bands. In $F_2$, only those flies that were homozygous for the OreR third chromosome had the F band. Such flies were now crossed to ru h th st cu sr e$^s$ ca, which were S with respect to ODH. The crossovers obtained in $F_2$ place the ODH locus between st (44) and cu (50), tentatively at 49.4.

The ODH bands can be demonstrated on electropherograms not only with octanol, but also with hexanol and heptanol. Since octanol esters are present in plant oils, it is possible that the function of ODH is the utilization of these energy sources. Benzyl alcohol has been reported as a substrate also (J. Exp. Zool., in press). Apparently the benyl alcohol reaction is not associated with ODH, but with a different band, which also migrates to the cathode, and has been termed "pyridoxal band" (see J. B. Courtright, this issue of DIS).

Report of W. D. Kaplan

T(1;2L)D1 From $H^3$ deoxycytidine-fed 1st instar male larva. Breaks at 6F and 26C. Lethal in male.

T(1;2R)D2 From $H^3$ deoxycytidine-fed 1st instar male larva. Breaks at 8B and 46B. Lethal in male.

T(1;3L)D3 From $H^3$ deoxycytidine-fed 1st instar male larva. Breaks at 4F and 62A. Lethal in male.

In(1)D1 From $H^3$ deoxycytidine-fed 1st instar male larva. Breaks at 13B and 16A. Lethal in male.

Report of K. K. Kidd

$w^{64g3}$: white$^{64g3}$ K. K. Kidd, 1964g. 1-1.5. This spontaneous mutation occurred in a stock containing vermilion, garnet, and forked and showed an ivory phenotype. The $w^{64g3}$ phenotype is indistinguishable from garnet when young but darkens to a deep carnation color with age. No sex or temperature differences have been observed. $w^{64g3}$/Basc ($w^a$) females had the garnet-like phenotype of the homozygote.

$w^{FM6}$: white in FM6 balancer. K. K. Kidd, 1964a. Spontaneous mutation inseparable from the FM6 inversion (Grell and Lewis, DIS 30:71) with a typical white phenotype. Salivary analysis shows no change in the FM6 inversion pattern. The stock is maintained as a balancer for a sex-linked lethal.

Modified FM6 balancers (See Grell and Lewis, DIS 30:71 for original description)

"FM6,w": Ins(1)FM6, $y^{31d}$ $sc^8$ $w^{FM6}$ dm B. K. K. Kidd, 1964a. See this DIS, this section above.

"FM6,$dm^+$": Ins(1)FM6, $y^{31d}$ $sc^8$ $dm^+$ B. K. K. Kidd, 1963. Recovered in a balanced lethal stock, this is either a spontaneous reversion to the wild-type allele of diminutive in the FM6 inversion or a rare recombinational separation of the mutant from the inversion. All other mutants are still present in the multiple inversion, which salivary analysis showed to be identical to the original FM6. The viability of this stock is greater than the regular FM6, and since the females are fertile, the stock is maintained homozygously.

"FM6, w $dm^+$": Ins(1)FM6, $y^{31d}$ $sc^8$ $w^{FM6}$ $dm^+$ B. K. K. Kidd, 1964h. The intact FM6 inversion with w added and dm missing. This, the stock is maintained homozygously. The productivity and viability are very good. It is felt that this stock will be valuable for use in the detection of sex-linked lethals much as "Basc" (Muller-5) which this stock surpasses in cross-over suppression. This stock arose by recombination in heterozygous "FM6, w"/ "FM6, $dm^+$" females. Approximately 3.2% recombination was found between w and dm, as would be expected since no inversion breaks occurred between these two loci.

Report of M. Ashburner and G. Hudson

$gl^{63a14}$: glass 63a14 Spontaneous in line J54 derived from a single wild female from Japan.

$gl^{63f6}$: glass 63f6 Spontaneous, found six months after $gl^{63a14}$ in line J58 also derived from a single wild female from Japan. Both mutants like gl but eye colour darker and pigmented area larger. Male eyes darker than female. Malphigian tubules wild type. No crossing over between $gl^{63a14}$ and gl (1235 flies), $gl^3$ (1546 flies), $gl^{60j9}$ (1108 flies), or $gl^{63f6}$ (2565 flies). Male heterozygotes $gl^{63a14}$/gl have more pigment than either parent. $gl^{63a14}$ and $gl^{63f6}$ are probably identical. Viability and fertility good. RK1

Report of G. K. Manna

held-out This mutant, which was described under melanogaster in DIS 40, belongs under ananassae.

Report of Tufts University

$dp^{65}$ Spontaneously arose in a vermilion; brown stock. Characteristic truncate wings, and dorsal vortices.

Report of Sara H. Frye

y ac (yellow-achaete) or (achaete-yellow) ac y chromosome 1-0.0, occurred singly in $F_1$ male from irradiated series (for details, see Frye, research note, this issue DIS) but could be of spontaneous origin; yellow body color, yellow wings, and yellow bristles (i.e., yellow-one phenotype, symbolized y) and posterior dorsocentral bristles missing (i.e., achaete-one phenotype, symbolized ac). Progeny test of the original double marker mutant male with y f:= virgin females and progeny tests of the $F_1$ double marker mutant males with y f:= virgin females showed that males bearing In49 $B^{M1}$ chromosome with the double marker mutant phenotype (i.e., y ac In49 $B^{M1}$) to be of good viability and fertility. Separate allelism tests for yellow or achaete were performed by the following crosses and observing the daughters:

1. $\frac{Y}{y}$ ac In49 $B^{M1}$ ♂ X y w ♀ - daughters were phenotypically yellow (y) and non-achaete ($ac_{M1}$). Backcross of $F_1$ yellow virgin daughters ($ac^+$) to $P_1$ males showed that the y ac In49 $B^{M1}$ chromosome is homozygous-female-viable.

2. $\frac{Y}{y}$ ac In49 $B^{M1}$ ♂ X $ac^3$ $w^a$ ♀ - daughters were phenotypically achaete (ac) and non-yellow ($y^+$). Backcross of $F_1$ achaete virgin daughters ($y^+$) to $P_1$ males showed that y ac In49 $B^{M1}$ chromosomes are homozygous-female-viable.

Results of 23 separate fertility tests for $X\text{-}Y^S$ exchanges were negative. Tests for $X\text{-}Y^L$ exchanges or an involvement with a gross structural change such as a translocation or an inversion have not been conducted. Existing stock cultures were sent from Irvine, Kentucky to 45 American and European investigators including the two stock centers, Pasadena and Philadelphia (see Frye, materials available, this issue DIS). (This work was partially supported by the estate of the author's deceased father, Dr. H. J. Comley, of Irvine, Kentucky and PHS-5TI-GM-337-05.)

Report of W. W. Doane

A system has been worked out for the Amy alleles that will not only conform with that of Kikkawa, based on agar gel electrophoresis, but will also apply to separations by acrylamide gel disc electrophoresis (see research note, this issue). According to this scheme, the $Amy^4$ allele described by Kikkawa should be called $Amy^{1.4}$.

$Amy^{1.2}$: $Amylase^{1.2}$ $_2$ Doane 64e6. A highly inbred strain containing the mutant $adp^{fs}$, balanced against SM1, $al^2$ Cy $sp^2$, was found to include this Amy allele linked to $adp^{fs}$. Its electrophoretic banding pattern is characterized by two major isozymes in the "1" and "2" positions. A band of very weak activity precedes the faster migrating, major one and appears in the "0" position. Total α-amylase activity in this is generally quite high, lying intermediate between $Amy^{4.6}$ and $Amy^{1.3}$. Individuals varying greatly in activity, especially females, but this may be an effect of the $adp^{fs}$ mutant linked to it.

Report of R. J. MacIntyre

$Acph\text{-}1^A$: Acid phosphatase-$1^A$ MacIntyre, 1964. 3-101.4±0.1

$Acph\text{-}1^B$: Acid phosphatase-$1^B$ MacIntyre, 1964. 3-101.4±0.1. These are two codominant, interacting alleles controlling the structure of electrophoretic variants of a non-specific acid phosphatase. The demonstration of the enzyme is best performed in starch gels prepared in 0.05 M tris-hydrochloric acid buffer, pH 8.7. A voltage drop of 5-6 v/cm for 5-6 hours is sufficient to separate the variant enzymes. The stain solution contains in acetate buffer at pH 5.0 the substrate, alpha naphthyl phosphate, and Fast Blue BB as the dye coupler. Individuals homozygous for $Acph\text{-}1^A$ produce a single, intense band which migrates 1 1/2 cm from the origin. Homozygous $Acph\text{-}1^B$ individuals under the same conditions show a band at about 3 cm from the origin. $Acph\text{-}1^A/Acph\text{-}1^B$ heterozygotes produce both parental bands plus a more intensly staining, intermediate or hybrid enzyme. $Acph\text{-}1^A$ has been found only in one recently collected polymorphic strain from Commack, New York. RK1 even with homogenates of single 2nd instar larva, 3rd instar larva, pupae, or adults.

Report of Ross J. MacIntyre

Locus of the structrual gene for 3rd larval instar alkaline phosphatase (Aph) For a description of this gene-enzyme system and the staining procedures used to detect the enzyme in starch gel, see Beckman and Johnson (Genetics 49: 829-835 1964). The buffers used in the gels and for the electrolyte, however, are described in Wright (Genetics 47: 787-801 1963). In the fromer communication, the gene was localized only on the left arm of the 3rd chromosome of D. melanogaster.

In order to insure homozygosity of $Aph^F$ and $Aph^S$ alleles in the stocks used for mapping the gene, single pair matings were set up, and six larva from each mating were analyzed for their Aph phenotype. Two stocks containing 3rd chromosome mutant marker genes were established from matings in which all six offspring showed the "fast" band when run adjacent to suitable "control" larva ("control" stocks, known to be homozygous for "fast" and "slow" alleles, were obtained from Dr. T. R. F. Wright). These stocks, set up from single pair matings and considered to be homozygous for $Aph^F$, were ru h th st cu sr $e^s$ ca (rucuca) and h th st ss. In the same way, a wild type strain homozygous for $Aph^S$ was derived from an Ore-R stock.

To map the gene, recombinant male progeny from the appropriate testcrosses— + + + + + + + + ($Aph^S$)/ ru h th st cu sr $e^s$ ca ($Aph^F$) females x "rucuca" ($Aph^F$)/($Aph^F$) males and + + + + + ($Aph^S$)/ h th st ss ($Aph^F$) females x h th st ss ($Aph^F$)/h th st ss ($Aph^F$) males— were individually crossed to virgin females homozygous for $Aph^F$. From each of these single male crosses, six 3rd instar larva were analyzed for their Aph genotypes. If all six were $Aph^F$ homozygotes, the crossover chromosome from the recombinant male was considered to have carried the $Aph^F$ allele. If one or more Aph heterozygotes appeared in the six larva analyzed, the crossover chromoso carried the $Aph^S$ allele. Only very rarely (1 in 64) would six homozygotes be randomly selected from the offspring of a cro between a heterozygous male and a homozygous female. This rare event would not significantly affect the results, which are summarized in the accompanying table.

| Recombinant Chromosome | Number which carried $Aph^F$ | Number which carried $Aph^S$ |
|---|---|---|
| 1. ru + + + + + + + | 0 | 9 |
| 2. ru h + + + + + + | 0 | 9 |
| 3. ru h th st + + + + | 2 | 3 |
| 4. ru h th st cu + + + | 3 | 0 |
| 5. + + + + cu sr $e^s$ ca | 1 | 1 |
| 6. + + + + + sr $e^s$ ca | 0 | 16 |
| 7. + + + + + + $e^s$ ca | 0 | 9 |
| 8. + + + + + + + ca | 0 | 9 |
| 9. h + + + | 0 | 9 |
| 10. + th st ss | 9 | 0 |
| 11. h th st + | 60 | 11 |
| 12. + + + ss | 7 | 36 |

Table 1. Results from the analyses of crossover chromosomes for Aph allele.

The more meager data from the "rucuca' testcross shows that the Aph locus certainly lies between st and sr and perhaps to the left of cu ( see recombinant chromosomes #3-6 in the table). Recombinant chromosomes involving a crossover between st and ss (#'s 11 and 12) corroborate this conclusion, and on the basis of these 114 chromosomes analyzed, a locus for Aph was calculated. 15.8% (18 of 114) of the crossovers occurred between st and Aph. 15.8% of the map distance between st (44.0) and ss (58.5) is approximately 2.3 map units. Thus, Aph lies at 46.3 ± 0.5 (s) on Chromosome III. Beckman and Johnson (1964. Hereditas 51, 212-220) also reported 2.9% recombination between the structural gene for Esterase C and Aph. The locus for Est C may be therefore at 43 or 49 on the genetic map.

Report of G. R. Johnson

Location of tu-1, a wild type isoallele. Of 129 forked males produced by females of the genotype f, su-f/tu-1 11 were found to also carry tu-1. Using the interval 7.8 between f and su-f, tu-1 is located at 63.4 (64.5-11/129 of 7.8). Consistent with the location of tu-1 to the left of su-f is the fact that females Df(1)ma-1/tu-1 produce the maternal effect characteristic of females tu-1/tu-1.

## NEW YORK, NEW YORK: COLUMBIA UNIVERSITY

Essentially the same as DIS 34:59, except:

D. paulistorum

Centro-American race (7 strains)
Amazonian race (33 strains)
Andean-South Brazilian race (26 strains)
Orinocan race (19 strains)
Guianan race (2 strains)
Plus several strains not yet assigned to a definite race.

D. dominicana

New Guinea (1 strain)

D. serrata

Australia (9 strains)
New Guinea (5 strains)
New Britain (1 strain)

D. birchii

Australia (1 strain)
New Guinea (4 strains)
New Britain (1 strain)

(Note that the Calypso race is now deleted)

## VEZNECILER, ISTANBUL: ISTANBUL ÜNIVERSITESI
### Department of General Zoology

Drosophila species collected during 1962-1964:

D. melanogaster
D. simulans
D. busckii
D. immigrans
D. subobscura
D. fenestratum
D. funebris
D. limbata
D. nitens
D. confusa
D. unimaculata

Due to the zoogeographical situation of Istanbul we expect to find other species also.

## PÔRTO ALEGRE, BRAZIL: UNIVERSIDADE DO RIO GRANDE DO SUL
### Laboratório de Genética Animal

D. willistoni

Wild strains

Eldorado G (Rio Grande do Sul), Vila Atlântica (São Paulo), Maranguape (Ceará)

| Chromosome 1 | Chromosome 2 | Chromosome 3 |
|---|---|---|
| w | abb bw (analyzer stock) | p(In)/lethal (analyzer stock) |
| | SHk abb bw (In)/lethal (analyzer stock) | p/normal |
| | Star Emarginata | aristapedia |
| | Em ph | |

Other Species

D. cardinoides (Eldorado G, Rio Grande do Sul); D. capricorni (Ilha das Cobras, Brasília); D. cubana (Tefé, Trinidad 330); D. equinoxialis (Içana, Tefé); D. fumipennis (Paranaí); D. insularis (St. Kitts Islands, Guadalupe Islands); D. kikkaway (Eldorado A, Rio Grande do Sul); D. lebanonensis (Eldorado, Rio Grande do Sul); D. montium; D. nebulosa (Pedras de Una, Guaporé); D. paulistorum (Içana); D. pararepleta (Itatiaia, Ilha das Cobras); D. polymorpha (Boa Viagem, Jacú, Pascoal, Itapeva); D. tropicalis (Palma).

## DEKALB, ILLINOIS: NORTHERN ILLINOIS UNIVERSITY
### Biological Sciences Department

Several samples of both D. immigrans and D. tripunctata

## TUCSON, ARIZONA: UNIVERSITY OF ARIZONA
### Department of Zoology

Cardini group

cardini sub group

D. acutilabella: Cuba; Florida(3); Jamaica(3); Haiti(4)
D. cardini: Florida(2); Chile; Mexico; Trinidad
D. cardinoides: Mexico; Trinidad(2); Brazil
D. neocardini: Brazil(2)
D. neomorpha: Honduras; Panama(2); Colombia; Trinidad
D. parthenogenetica: Mexico(4); Colombia(2); Nicaragua; Trinidad(2); Honduras
D. polymorpha: Colombia(3); Trinidad; Brazil(4)
D. procardinoides: Bolivia

dunni sub group

D. antillea: St. Lucia
D. arawakana: Montserrati; Guadeloupe: St. Kitts
D. belladunni: Jamaica(4)
D. caribiana: Martinique
D. dunni: St. Thomas; Puerto Rico
D. nigrodunni: Barbados
D. similis: St. Vincent

Macroptera group

D. macroptera: Huachuca Mts. (Arizona)

Melanogaster group

D. melanogaster: Tucson (Arizona)
D. simulans: Tucson (Arizona)
D. succinea: Canelas (Durango, Mexico)

Obscura group

D. persimilis: Porcupine Flats (California)
D. pseudoobscura: Tucson: Hawley Lake; Catalina Mts.; Santa Rita Mts. (Arizona)

Nannoptera group

D. pachea: Sonoita, Caborca, Hermosillo, Guaymas, Empalme, Kino, Desemboque, Esperanza (Sonora, Mexico); Comondu (Baja California, Mexico)
D. nannoptera: Puebla (Mexico)

Repleta group

D. aldrichi: Navajoa (Sonora, Mexico)
D. arizonensis: Tucson (Arizona); Guaymas, Empalme, Esperanza (Sonora, Mexico)
D. eohydei: Bucaramanga (Colombia)
D. hamatofila: Tucson (Arizona); El Alamo ranch (Sonora, Mexico)
D. hydei: Chinipas (Chihuahua, Mexico)
D. longicornis: Alamos, El Alamo ranch (Sonora, Mexico)
D. melanopalpa: Santa Rita Mts. (Arizona)
D. meridiana: Chinipas (Chihuahua, Mexico); Gurymas (Sonora, Mexico)
D. mojavensis: Chocolate Mts. (California); El Barril ranch, La Paz (Baja California, Mexico); Sonoita, Desemboque, Tiburon Island, Empalme, Esperanza (Sonora, Mexico)

D. neohydei: Carpentero (Venezuela)
D. nigrohydei: Portal (Arizona)
D. nigrospiracula: Tucson, Organ Pipe Nat'l. Monument (Arizona); San Felipe (Baja California, Mexico); Magdelena, Hermosillo, Guaymas, Empalme (Sonora, Mexico)
species C: Canelas (Durango, Mexico)
species M: Tucson, Organ Pipe Nat'l. Monument (Arizona)

## ROCHESTER, NEW YORK: UNIVERSITY OF ROCHESTER
### Department of Biology

D. busckii - There are more than 300 stocks of this species. They include X-chromosomal and autosomal mutants (with visible effects) in various combinations, as well as a number of special stocks in which lethals are associated with chromosomal aberrations. Dominant and recessive markers, associated with inversions and other types of chromosomal aberrations, are available for each chromosome. There are also more than 25 strains from geographically remote populations, and about 50 stocks with second aberrant chromosomes extracted (isolated and balanced) from flies of these populations.
D. hydei - 3 strains from Rochester, N.Y., Salinas, Calif., and Buenos Aires, Argentina
D. mercatorum - dark and light forms from Rochester, N.Y.
D. nigrospiracula - from Buenos Aires, Argentina
D. paramelanica - from Rochester, N.Y.
D. robusta - from Rochester, N.Y.
D. transversa - from Rochester, N.Y.
Scaptomyza graminum - from Corvallis, Oregon
Megaselia scalaris - from Rochester, N.Y.

## EMPORIA, KANSAS: KANSAS STATE TEACHERS COLLEGE

| D. virilis | | Multichromosomal |
|---|---|---|
| Wild Stocks | Brazil<br>Chile<br>Texmelucan | b;tb gp$^2$;cd pe |
| Argentina | | |

## AMHERST, MASSACHUSETTS: AMHERST COLLEGE

D. affinis: Amherst 1954
D. busckii: Amherst 1958
D. funebris: Amherst 1954
D. hydei: Amherst 1956
D. immigrans: Amherst 1962
D. melanica: Amherst 1952
D. robusta: Amherst 1960
D. simulans: Amherst 1961

## COLD SPRING HARBOR, NEW YORK: CARNEGIE INSTITUTION OF WASHINGTON

D. busckii: wild-type B
D. virilis: China-a

## RICHMOND, VIRGINIA: MEDICAL COLLEGE OF VIRGINIA

D. ananassae

Wild:

Puerto Rico

D. equinoxialis

Wild:

Brazil
Puerto Rico

Chromosome 1

sh
w

D. mirim

Wild:

Puerto Rico

D. nebulosa

Wild:

Puerto Rico

D. paulistorum

Wild:

Brazil
Colombia
El Salvador
Honduras
Trinidad
Venezuela

D. peninsularis

Wild:

Puerto Rico

D. prosaltans

Wild:

Jamaica

D. sturtevanti

Wild:

Haiti
Trinidad

D. tropicalis

Wild:

Brazil
Puerto Rico

Chromosome 1

Delta
ro se
ro w

D. willistoni

Wild:

Argentina
Brazil
Cuba
Dominican Republic
Florida
Haiti
Jamaica
Puerto Rico
Trinidad

## CALCUTTA, INDIA: UNIVERSITY OF CALCUTTA
### Department of Zoology

D. ananassae

Wild Stock

1 a6 Calcutta

Chromosome 2

2 bw vs $ss^a$

Chromosome 3

3 pc

Multichromosomal

4 In(3)M pr pc px fu
5 stw px pc fu

Special selected strains with crossing over in males

6 px pc (6a)
7 px pc (6a) high
8 px pc (3)
9 px pc (6a-13)
10 px pc (6a-4)

D. bipectinata
D. simulans

## NEW HAVEN, CONNECTICUT: YALE UNIVERSITY
### Department of Biology

D. acanthoptera
D. affinis: Bethany, Conn.; Greenwich, Conn.; Sleeping Giant, Conn.
D. algonquin: Bridgeport, Conn.; West Haven, Conn.
D. americana americana: Independence, Ohio; Western
D. americana texana: Florida
D. ananassae: Cristobal
D. bifasciata: sex-ratio; Pavia normal
D. borealis: Kent, Conn.
D. busckii: Lankenau (Abington, Pa.)
D. duncani: New Canaan, Conn.
D. equinoxialis: Puerto Rico, normal and sex-ratio
D. flavomontana: Yampa River, Colo.
D. funebris: Rexburg, Idaho: Stockholm, Sweden; Upperville, Va.; white eye; Yucatan
D. gibberosa: South Mexico
D. hydei: Chile; New Haven, Conn.; Vera Cruz; Zurich, Switzerland; Lobe mutant (Gloor)
D. immigrans: DeKalb, Illinois; New Haven, Conn.; North Cannaan, Conn.; Sharon, Conn.; Washington, Conn.
D. lacicola: Fairbanks, Minn.
D. lebanonensis
D. littoralis: Switzerland
D. melanica: St. Louis, Mo.
D. mirim
D. montana: Cottonwood Canyon, Utah
D. nebulosa: Haiti, normal and sex-ratio
D. nigrohydei
D. micromelanica: Cold Spring Harbor, N.Y. Marlborough, Conn.; Stafford, Conn.
D. novamexicana
D. paramelanica: Hamden, Conn.; Killingsley, Conn.
D. paulistorum: Belem; Bucamaranga; Cantareiras; Lancetilla; Trinidad
D. persimilis: Whitney, Calif.
D. polychaeta
D. prosaltans: Belem; Chilpancingo (stellata)
D. pseudoobscura: Pinion Standard
D. quinaria: Kent, Conn.
D. repleta: Philadelphia, Pa.; Prospect, Conn.
D. robusta: Fairfield, Conn.; Hebron, Conn.; Kent, Conn.; Lexington, Conn.; New Canaan, Conn.
D. simulans; Lankenau
D. tripunctata: Bridgeport, Conn.; Fairfield, Conn.
D. virilis: Japan
D. willistoni: Barbadoes-3; Belem; Recife-3; Recife Pop. 168; ebony;pink; white eyes; sex-ratio

Zaprionus vittiger: South Africa

## LINCOLN, NEBRASKA: THE UNIVERSITY OF NEBRASKA
### Zoology and Physiology Department

D. affinis: Florida, Nebraska, Ontario
D. algonquin: Minn., Ontario, Vermont
D. azteca: Arizona, California, Guerrero (Mexico)
D. athabasca: Alaska, British Columbia, Colorado, Ohio, Idaho, Massachusetts, Minnesota, New Mexico, North Carolina, Oregon, Ontario, Pa., Vt., and Wash.

## LEXINGTON, KENTUCKY: UNIVERSITY OF KENTUCKY
### Department of Zoology

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

## TOKYO, JAPAN: TOKYO METROPOLITAN UNIVERSITY
Department of Biology

### D. ananassae

Wild Stocks

20 strains maintained by mass culture.

Mutants

1 w
2 y f
3 f;px st
4 b
5 pxd
6 ru
7 bw-R
8 j
9 ps
10 se

### D. bifasciata

Wild Stocks

124 strains in 34 localities. Most of them were originated from single inseminated females.

Mutants

Sex-linked: a, Ac, acp, y, f
Autosomal: or, orr, cn, cu
Cytoplasmic sex-ratio: 18 strains from 10 different localities in Japan and one strain from Italy.

### Other Species

| | | |
|---|---|---|
| D. ambigua | Wild | 1 strain |
| D. angularis | Wild | 1 strain |
| D. auraria Race A | Wild | 31 strains |
| D. auraria Race B | Wild | 16 strains |
| D. auraria Race C | Wild | 14 strains |
| D. busckii | Wild | 8 strains |
| D. chinoi | Wild | 1 strain |
| D. funebris | Wild | 4 strains |
| D. hydei | Wild | 5 strains |
| D. immigrans | Wild | 8 strains |
| D. kikkawai | Wild | 2 strains |
| D. kuntzei | Wild | 2 strains |
| D. lacertosa | Wild | 2 strains |
| D. lutea | Wild | 17 strains |
| D. maculinotata | Wild | 1 strain |
| D. miranda | Wild | 1 strain |
| D. nasuta | Wild | 2 strains |
| D. nigromaculata | Wild | 1 strain |
| D. obscura | Wild | 1 strain |
| D. persimilis | Wild | 1 strain |
| D. pseudoobscura | Wild | 4 strains |
| D. pseudoobscura | Mutant | 3 strains |
| D. pulchrella | Wild | 1 strain |
| D. rufa | Wild | 1 strain |
| D. simulans | Wild | 2 strains |
| D. suzukii | Wild | 4 strains |
| D. takahashii | Wild | 37 strains |
| D. tristis | Wild | 1 strain |

| | | |
|---|---|---|
| D. virilis | Wild | 14 strains |
| D. virilis | Mutant | 7 strains |

## CHANDIGARH, PUNJAB, INDIA: PUNJAB UNIVERSITY
### Department of Zoology

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

## SAPPORO, JAPAN: HOKKAIDO UNIVERSITY
### Department of Zoology

D. histrioides: Sapporo (2 strains), Nopporo (1), Toya (1)
D. busckii: Sapporo (1)
D. ananassae: India (1)
D. kikkawai: India (1)
D. auraria race A: Sapporo (1)
D. auraria race B: Nopporo (1)
D. brachynephros: Sapporo (1)
D. unispina: Sapporo (1)
D. nigromaculata: Sapporo (1)
D. histrio: Fuyushima (1)
D. immigrans: Sapporo (1)
D. funebris: Sapporo (1)
D. virilis: Sapporo (1), Okushiri Is. (1)
D. lacertosa: Fuyushima (1), Toya (1), Ohnuma (1), Rebun Is. (3)
D. moriwakii: Jōzankei (1)
D. okadai: Nopporo (1)
D. neokadai: Nopporo (2)
D. sordidula: Sapporo (2), Ohnuma (1)
D. pseudosordidula: Nopporo (2), Toya (1), Ohnuma (1)
Scaptomyza pallida: Yamada-Onsen(1), Hawaii(1)
Scaptomyza monticola: Yamada-Onsen (1)

## SANTIAGO, CHILE: UNIVERSIDAD DE CHILE, DEPARTAMENTO DE GENETICA
### Instituto do Biología "Juan Noé"

D. busckii: Chile (La Serena)
D. camaronensis: Child (Azapa)
D. funebris: Chile (La Serena, Valdivia, Tierra del Fuego y Punta Arenas)
D. gasici: Chile (Arica), Bolivia (Cochabamba), Colonbia (Bogotá)
D. gaucha: Brazil (M. Capoes, C. de Jordan and Taimbas), Argentina (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: Child (Copiapó, Vallenar, La Serena, El Tabo, Vina del Mar, Olmué, Bellavista, Arryán, Los Alpes, Colbuń, Los Quenes, Chillán), Argentina (Mendoza)
D. simulans: Perú (Lima), Chile (Arica)
D. viracochi: Perú (Machu-Picchu), Colombia (Bogotá)
D. virilis: Chile (Santiago)

## LEIDEN, NETHERLANDS: GENETISCH LABORATORIUM DER RIJKSUNIVERSITEIT

D. hydei
D. eohydei
D. neohydei
D. nigrohydei
D. bifurca
D. buzzatii
D. mulleri
D. repleta
D. mercatorum
D. victoria

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

D. ananassae: 24 strains from different parts of West Bengal. 1 strain from Hazaribagh forest in Bihar. 1 strain from Mikir hills in Assam.

D. bipectinata: seven strains

D. malerkotliana

## MEDFORD, MASSACHUSETTS: TUFTS UNIVERSITY
### Department of Biology

D. colorata
D. paulistorum, curata C
D. persimilis, 17 strains
D. pseudoobscura
  CH-7 strains
  AR-10 strains

Mutant Strains

Chromosome 1

Pointed

Chromosome 2

Lobed

Chromosome 3

Bare

Chromosome 4

Curly

D. serrata, cairns
ribaul

## ANZYO, AICHI-KEN, JAPAN: NAGOYA UNIVERSITY
### Department of Animal Breeding

| | | |
|---|---|---|
| 51 | D. auraria | Anzyo-Aichi |
| 52 | " | Kiso |
| 53 | " | b(black) |
| 54 | D. hydei | Suginami-Tokyo |
| 55 | D. montium | Kanazawa |
| 56 | D. virilis | Nagoya |
| 57 | " | Tobetsu-Hokkaido |
| 58 | " | Tokyo |

## BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA
### Department of Zoology

700 D. simulans

wild

800 D. virilis

Pasadena wild

scarlet

900 D. montium Abidiaw

950 Zaprionus vittiger

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

D. virilis

Wild stock

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 stock

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

Other species

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

## TURKU, FINALND: UNIVERSITY OF TURKU
### Department of Genetics

D. simulans

Wild stock

Chromosome 3

jv se st pe
st pe

## BERLIN-DAHLEM, GERMANY: UNIVERSITÄT BERLIN
### Institut für Genetik

58 D. funebris: wild
59 D. busckii: wild
60 D. hydei: wild
61 D. simulans: v
62 D. virilis: wild

## SEOUL, KOREA: YONSEI UNIVERSITY
### Department of Biology

1 D. virilis (Korea)
2 D. auraria
  a A-14 (Japan)
  b A-15 (Japan)
  c H.K.D. (race B)

## HAREN, GRONINGEN, NETHERLANDS: UNIVERSITY OF GRONINGEN
### Genetical Institute

D. ambigua
D. ananassae
D. andalusiaca
D. buskii
D. bifasciata
D. duncani
D. funebris
D. hydei
D. immigrans
D. lebanonensis
D. littoralis
D. mercatorum
D. nebulosa
D. obscura
D. pseudoobscura
D. repleta
D. simulans
D. subobscura
D. virilis

Arrowhead + Chiricahua gene arrangements

## VARANASI, INDIA: BANARAS HINDU UNIVERSITY
### Department of Zoology

Wild Stocks

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

D. ananassae

Chromosome 1

$y_a$
w
vs

Chromosome 2

cu b se
cu se
b se
cu b
b
cu
se
ic
cu bw
$blo^a$
ss

Chromosome 3

px pc
stw pc
stw px
stw
px
pc

Unlocated mutants

dct
sp
ci
arch

## SEOUL, KOREA: CHUNGANG UNIVERSITY
### Department of Biology

D. auraria - Type A (15)
Type B (3)
Type C (10)
D. busckii (3)
D. nigromaculata (2)
D. pseudoobscura
D. suzukii (5)
D. virilis (5)

## MEXICO CITY, MEXICO: NATIONAL COMMISSION OF NUCLEAR ENERGY
### Laboratory of Genetics of Drosophila

A1 D. busckii
A2 D. pseudoobscura

## BAYFORDBURY, HERTFORD, HERTS, ENGLAND: JOHN INNES INSTITUTE

D. simulans

## UPPSALA, SWEDEN: UNIVERSITY OF UPPSALA
### Department of Genetics

D. littoralis
D. hydei
D. funebris
D. subobscura
D. americana
D. simulans
D. texana
D. virilis
D. pseudoobscura

## BARCELONA, SPAIN: UNIVERSIDAD DE BARCELONA
### Centro de Genética Animal y Humana

D. ambigua Spanish stocks
D. busckii Spanish stocks
D. buzzatii Armentera (Spain)
D. cameraria Canary Islands
D. funebris Bilbao
D. hydei Barcelona
D. immigrans Barcelona
D. obscura Bilbao
D. phalerata Several Spanish stocks
D. repleta Barcelona
D. simulans Several Spanish stocks
D. subobscura Several Spanish stocks, Mutant stocks
D. testacea Spanish stocks

## MILANO, ITALY: UNIVERSITÀ DI MILANO
### Instituto di Genetica

D. simulans

Wild Stocks

1 Aspra
2 Morro Bay
3 Pavia

Stocks selected for tumor manifestation

4 tu B1
5 tu Aspra

## SYDNEY, AUSTRALIA: UNIVERSITY OF SYDNEY
### Department of Animal Husbandry

Note: same as in DIS 40:31

## LYON, FRANCE: UNIVERSITÉ DE LYON
### Faculté des Sciences, Zoologie Expérimental

D. funebris
D. busckii

## FREIBURG, GERMANY: ZOOLOGISCHES INSTITUT DER UNIVERSITÄT

Note: see list of Max Planck-Institut für Biologie, Tübingen

## NEW MUTANTS

### simulans

Report of R. J. MacIntyre

Acph-$1^A$: Acid phosphatase-$1^A$ MacIntyre, 1965. 3-133.7±2.1.

Acph-$1^B$: Acid phosphatase-$1^B$ MacIntyre, 1965. 3-133.7±2.1. These are two codominant, interacting alleles controlling the structure of electrophoretic variants of a non-specific acid phosphatase. Methods of demonstrating the enzyme are included in the report of R. J. MacIntyre in the section on the new mutants of melanogaster of this issue. Under the conditions specified in that report, homozygous Acph-$1^A$ flies produce a single band which migrates about 3 1/2 cm from the origin or slightly ahead of the Acph-$1^B$ band from D. melanogaster. The enzyme from homozygous Acph-$1^B$ individuals migrates 5 cm from the origin. Heterozygotes at the Acph-1 locus produce both bands characteristic of the homozygotes and a more heavily staining intermediate or "hybrid" enzyme. Several wild type stocks monomorphic for each of the "homozygous" patterns have been found. Both alleles produce enzymes which show a marked reduction in their rates of migration in starch gels prepared in a tris-EDTA-boric acid buffer. The Acph-1 locus in D. simulans is considered to be homologous with the Acph-1 locus in D. melanogaster (MacIntyre, R. J. manuscript in preparation). RK1, even with homogenates of single 2nd instar larva, 3rd instar larva, pupae or adults.

### hydei

Report of W. W. Doane

Several strains of D. hydei were analyzed for differences in electrophoretic banding patterns formed by their $\alpha$-amylases. Three strains in this laboratory (New Haven, Zurich, and Vera Cruz) are homozygous for an allele whose enzyme assumes the position of "7" in the proposed scheme for Drosophila amylases separated by disc electrophoresis (see research note, this issue). Another strain, Chile, is homozygous for an allele whose enzyme lies in the "8" position. A single strain of D. nigrohydei was tested and found to contain amylase that migrated to the band "6" position characteristic of certain isozymes described in D. melanogaster.

### ananassae

Report of G. K. Manna

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

## RESEARCH NOTES

Barigozzi, C. and M. Sari. University of Milan, Italy. Two modes of transmission of Freckled.

New data have been collected proving the existence of two modes of transmission of Freckled phenotype in the same individual.

Crossing, in single pair, Frd/Cy L to +/+ flies, both having the same 1st chromosome marked with y w, three classes of individuals are derived: Frd/+, Cy L/+ (in equal proportions) and a variable amount (1-10%) of Cy L/+ (therefore lacking the Frd factor, which has been definitely located at 102) which show a Frd phenotype, restricted to a few black masses, at typical anatomical localizations (head, legs, thorax, etc.). These flies transmit the character unlinked to chromosomes, through both gametes. This condition is indicated as (Frd). The presence of a given X seems important to stabilize the phenomenon. While the highest proportion of (Frd) occurs with an X marked with y w, a lower frequency is obtained with another X marked with v; no (Frd) flies result when M-5 is present.

The transmission of (Frd) seems to be permanent. (Frd) genotype is interpreted as due to an extrachromosomal unit, transmitted close (or within) the nucleus, since both gametes are equally efficient.

(Frd) may be conceived, hypothetically, either as an extrachromosomal stage of Frd, or as a constantly extrachromosomal entity, whose multiplication (and, therefore, whose degree of manifestation) is controlled in the strongest way by the presence of Frd, and, more weakly, by the genes located in the 1st chromosome.

Scharloo, W., and W. Vreezen. Universities of Groningen and Leiden, Netherlands.

Selection for 5th vein interruption in a Hairless mutant.

Selection for a larger 5th vein interruption was practised on the mutant $H^{57c}$ introduced in the Kaduna cage population. Progress was steady until no 5th vein material was left posterior to the 2nd crossvein. Then progress was almost halted (generations 8-12) and the variability very small. After the appearance of some individuals with a break proximal to the 2nd crossvein, advance was resumed and the variability increased again. In generation 7 the first individuals appeared which showed a 5th vein interruption without the presence of $H^{57c}$. The penetrance and expression of these assimilants increased steadily, even when progress in $H^{57c}$ expression on which selection was practised, was apparently halted in generations 8-12.

It can be concluded that the lack of progress and the small variability in these generations is the reflection of a zone of canalization in 5th vein formation around its junction with the 2nd crossvein. This is supported by the result of introduction of chromosomes with dominant markers in stocks obtained from the selection line by relaxation at different stages. The effect of the modifiers introduced in this way seems to depend on the expression range and is very small when they act in the neighbourhood of the 2nd crossvein.

Beardmore, J. A. and W. Kramer. University of Groningen, Netherlands.

Selection responses in different environments.

The choice of environment in selection experiments is of considerable theoretical and applied interest. Falconer (Genet. Res. 1, 1960) on the basis of experiments with mice, suggested that for good overall expression of the selected character, selection should be practised in an environment unfavourable to the expression of this character.

In order to test this thesis and in an effort to establish in how far the genes involved in determining the same character in different environments are the same, an experiment involving selection for number of abdominal chaetae on the 5th sternite of D. melanogaster has been started. High, low and unselected control (H, L and C) lines are grown at 22°C and 28°C, giving in all six lines.

The mean values for the character at S 16 are as follows:

| | | | | | |
|---|---|---|---|---|---|
| H 28 | 26.54 | C 28 | 19.91 | L 28 | 16.42 |
| H 22 | 25.36 | C 22 | 20.84 | L 22 | 15.34 |

1. Selection is clearly effective in both environments.
2. Selection response is greatest in the environment whose effect acts against that of selection (28° for H, 22° for L).

Cross tests in the other temperature were made at S 16 and the values for the six lines are:

| | 22° | 28° |
|---|---|---|
| L 22 | 15.65 | 14.99 |
| L 28 | 17.51 | 15.97 |

| | 22° | 28° |
|---|---|---|
| H 22 | 24.48 | 24.14 |
| H 28 | 24.79 | 25.51 |

| | 22° | 28° |
|---|---|---|
| C 22 | 20.84 | 20.40 |
| C 28 | 21.94 | 19.91 |

1. Selection response is largely maintained in the new environment using, as a basis of comparison, deviation from control in both environments. This might indicate that the genes selected in the two conditions are broadly similar.

2. The effect of shifting from one environment to the other is not reciprocally equal in any of the three comparisons, 28° lines being more sensitive than 22° lines in all

cases. The H 28 line has fewer bristles at 22°C than at 28°C, suggesting that some of the genes effective at the latter temperature have a diminished effect at 22°C. The magnitude of this diminution in effect is probably of the order of three bristles. Provisionally we may conclude that the genes selected for in H 28 differ appreciably from those selected for in H 22.

The experiment is being continued.

Mettler, L. E. and J. J. Nagle. North Carolina State University, Raleigh, N.C.

Corroboratory evidence for the concept of the sympatric origin of isolating mechanisms.

Drosophila arizonensis and D. mojavensis are two species found in the southwestern part of the United States and in northwestern Mexico. The central range of arizonensis includes the Central Gulf Coast, Foothills of Sonora, Plains of Sonora, and southern parts of the Arizona Upland and Lower Colorado Valley regions of the Sonoran Desert. Drosophila mojavensis consists of two morphologically and cytologically distinct races. Race A is limited to the Mojave Desert and Race B is found in the Central Gulf Coast, Arizona Upland, and Lower Colorado Valley regions of the Sonoran Desert. Race A of mojavensis is allopatric to arizonensis, while Race B is sympatric with arizonensis over a large portion of its range. Both species are members of the Mulleri subgroup of the Repleta group and they are known to hybridize and produce some fertile offspring (Patterson and Stone, 1952). Drosophila arizonensis is morphologically and cytologically distinguishable from either race of mojavensis (species-specific paracentric inversion differences occur in three of the haploid set of six chromosomes).

Four cage populations were initiated for the purpose of studying inter-specific competition and possible secondary intergradation. Cages I and II were initiated with equal proportions of males and females of arizonensis and Race A of mojavensis, and Cages III and IV were initiated in the same manner using arizonensis and Race B of mojavensis. The composition of each population was determined by a cytological examination (salivary-gland chromosomes) of larvae developing from egg samples taken every thirty days (approximating generations). Parental, $F_1$ hybrid, and post-$F_1$ recombinant karyotypic combinations could be ascertained for the three chromosomes having species-specific arrangements.

The data obtained from these populations conform to the hypothesis that reproductive isolation is stronger in areas where two species are sympatric, as compared to the degree of isolation between allopatric forms of the same species, which is expected under the concept that isolating mechanisms are intensified through natural selection, as originally proposed by A. R. Wallace (Dobzhansky, 1940).

It is evident from Table 1 that the degree of reproductive isolation is much stronger between the naturally sympatric forms than between the allopatric forms (the relative degree of reproductive isolation being inversely proportional to the incidence of interspecific recombinant types). Reproductive isolation between arizonensis and Race B of mojavensis is apparently complete in nature; no hybrids have been found among individuals sampled from several areas of Sonora, Mexico where the two species are known to be sympatric (samples collected by Dr. W. B. Heed). Although the above data suggest that introgression or intergradation might occur between arizonensis and Race A of mojavensis, there is ample reason to suspect that these forms are truly allopatric.

Table 1. Percentages of interspecific recombinant types from hybridization between allopatric and sympatric populations of the same two species.

| Sample Number | Percentage of Recombinant Types | | | |
|---|---|---|---|---|
| Cage No. | I | II | III | IV |
| 1 | 30.0 | 85.0 | 0.0 | 0.0 |
| 2 | 51.0 | 91.0 | 0.0 | 0.0 |
| 3 | 72.0 | 89.0 | 1.7 | 1.7 |
| 4 | 85.0 | 92.0 | 0.0 | 0.8 |
| 5 | 88.0 | 88.0 | 0.8 | 0.0 |
| 6 | 86.0 | 82.0 | 5.0 | 2.5 |
| 7 | - | - | 2.5 | 3.3 |

Poulson, D. F. Yale University, New Haven, Connecticut. Further cases of maternal SR in Drosophila species.

When examined in this laboratory two cases of SR found in Brazil by C. Malogolowkin proved to be spirochete associated. One was in a strain of D. willistoni from Recife referred to as Recife DI, SR 32. The other was in a strain of D. paulistorum from Belem referred to as Belem SR. Both strains were maintained at Yale for several years, but have recently proved difficult to keep. These bring to six the number of known spirochete associated cases of SR in the willistoni species group. As reported earlier there is no evidence of spirochete involvement in the SR lines of bifasciata from Italy and Japan. The same appears to be true of a new case of SR in D. robusta found in a line from Florida by H. L. Carson who has kindly provided materials for study. To date all examined cases of SR in the willistoni species group involve the presence of spirochetes while those in other species groups have given no evidence of such involvement. However, artificially transferred SR from members of the willistoni group can be maintained in very different species such as melanogaster, pseudoobscura, bifasciata, virilis, hydei, and robusta with varying levels of success depending on strain of spirochete and strain of host.

Ursprung, H. The Johns Hopkins University, Baltimore, Maryland. In Vitro hybridization of Drosophila alcohol dehydrogenase.

Isozymes of alcohol dehydrogenase (ADH) in Drosophila have recently been found independently in three laboratories (Nature 204:906, 1964; Science 149:80, 1965; J. Exp. Zool., In press). Two types of homozygous strains were found, I and II, each containing three ADH isozymes. The two strains differ in the electrophoretic mobility of at least one isozyme. A hybrid fly, III, contains seven ADH isozymes: the four parental forms and three hybrid molecules. These results are consistent with the assumption that Drosophila ADH is a dimer.

We have now succeeded in producing the same hybrid molecules in vitro. Flies of types I and II were extracted in 6M guanidine hydrochloride and the extracts combined. No ADH activity was detected in these extracts after agar gel electrophoresis and staining in a mixture routinely used for the demonstration of ADH. This inactivation is reversible however. When the combined extracts are dialized against dilute buffer, electrophoresed, and stained, seven bands are seen, corresponding in electrophoretic mobility to the seven bands of a hybrid fly.

An investigation of the mechanism(s) involved in this in vitro hybridization is in progress. Recovery of bands in the hybridization experiment is favored by β-mercaptoethanol. Guanidinium hydrochloride treatment is not the only condition following which hybridization will occur. Prolonged dialysis of a homozygous fly extract against buffer can result in the formation of two hybrid bands, each intermediate between two parental forms. This finding suggests that ADH isozymes do not necessarily reflect the presence of two polypeptide subunits. Rather, it appears possible that the multiple forms of ADH in homozygous flies are brought about by dimerization of two physical chemical variants of one only polypeptide subunit. This assumption is in agreement with the genetic evidence that the isozyme pattern difference of the two homozygous strains is inherited in a monofactorial fashion.

Courtright, J. B. The Johns Hopkins University, Baltimore, Maryland. Electrophoretic analysis of xanthine dehydrogenase mutants.

The observation that xanthine dehydrogenase (XDH) reacts with a number of different substrates (Genetics 46:1455, 1963) has been interpreted to mean that the enzyme is multivalent, has a broad substrate specificity, or represents a cluster of isozymes which may or may not share common subunits or co-factors. Specifically, the reactivity of $ry^2$ mutant extracts with pyridoxal suggests enzymatic activity in the absence of a $ry^+$ factor.

We have combined agar gel electrophoresis (J. Expt. Zool., in press) and dehydrogenase

staining using various substrates in order to assign catalytic reactions to various proteins present in the gels more precisely. The results are listed in the Table.

| Stock | Substrate in staining mixture | Number of bands in electropherograms |
|---|---|---|
| OreR*) | xanthine, hypoxanthine, 2-amino, 4-hydroxpteridine | 2 (one migrating to the anode (A) (one migrating to the cathode (C) |
| $ry^2$ | " | 1 (C) |
| ma-1 | " | 0 |
| ma-1;ry | " | 0 |
| OreR*) | benzaldehyde, pyridoxal, benzyl alcohol | 1 (C, identical in migration to C above) |
| $ry^2$ | " | 1 (C) " |
| ma-1 | " | 0 |
| ma-1;ry | " | 0 |

*) A different wild type stock, Bethylie, is an electrophoretic variant in the sense that the mobilities of both bands are altered.

Clearly, both $ry^2$ and + contain a cathodally migrating "pyridoxal" band which is absent in ma-1. Its absence in ma-1 is probably due to ma-1 itself, since sc cv dx v ma-1 male progeny resulting from crossing over in sc cv dx v f/ma-1 females lack the band also, whereas ++++f males do contain the band. Extracts of $ry^2$ show the band also if XDH substrates are used, at least at pH 9. At pH 8, the pyridoxal band stains very poorly. This may account for the fact that fluorometric assays for XDH, which routinely are carried out at pH 8, fail to detect more than trace amounts of activity in $ry^2$ extracts.

The pyridoxal reaction present in + and $ry^2$ is not associated with the XDH molecule, which migrates to the anode, but with a molecule of quite different charges. Both bands stain poorly in an lxd stock (kindly supplied by Dr. E. C. Keller). The results are consistent with the assumption that both $ry^2$ and ma-1 are structural genes, and that lxd has a regulatory function.

Schwinck, Ilse. University of Connecticut. Storrs, Connecticut. Experimental induction of additional drosopterin formation in the eyes of various Drosophila mutants.

The amount of drosopterins in the eyes of rosy (ry) and maroon-like (ma-1) mutant flies can be increased (Z. f. Naturforschung 20b:322, 1965) up to levels found in non-autonomous transplantations by incubation of "free pupae" on paper moistened with saline-phenylalanine solutions (pupae dissected from puparium 10-20 hours before eclosion). Tyrosine, dopa and equimolar concentrations of leucine, iso-leucine and alanine were not effective in the induction of this wild type phenocopy eye color. Also, a number of cofactors of phenylalanine oxidation (NAD, NADP, $NADPH_2$, folic acid, tetrahydrofolic acid, 6,7-dimethyl-5,6,7,8,tetrahydropterin) did not enhance drosopterin systhesis in the "free pupae" incubation assay. Implantation of crystals or powder of the amino acids and cofactors cited into the thorax of 3-4 day old pupae without removal of the puparium confirmed the results of the "free pupae" incubation. In contrast to the organ transplantation studies, xanthine dehydrogenase activity is not enhanced in the phenylalanine-induced drosopterin phenocopy with rosy and maroon-like pupae. Furthermore, a similar phenylalanine dependent enhancement of drosopterin synthesis was observed for mutants with normally reduced drosopterin quantities and normal isoxanthopterin accumulation (claret, ca and pink, p), again indicating a non-involvement of xanthine dehydrogenase in the phenylalanine-dependent drosopterin phenocopy mechanism.

Herforth, R. and N. Westphal. University of Nebraska, Lincoln. Observations on the frequency of carbon dioxide sensitivity in a natural population of Drosophilid flies.

Drosophilids were collected daily at a single site in Lincoln, Nebraska, from June to October, 1965. Many of these wild-caught flies were tested for sensitivity to carbon dioxide by submitting them to the standard treatment of 15 minutes of pure $CO_2$ at 14°C. Those not awake 15 minutes after removal from $CO_2$ were considered sensitive.

Sensitivity was found regularly only in D. melanogaster, D. affinis males, and D. affinis subgroup females. The monthly frequency of sensitives in each of these groups is given in Table 1.

Table 1. Monthly frequency of $CO_2$ sensitivity in D. melanogaster, D. affinis males and D. affinis subgroup females.

| | D. melanogaster | | D. affinis males | | D. affinis subgroup females | |
|---|---|---|---|---|---|---|
| Month | S/N | %S | S/N | %S | S/N | %S |
| June | 3/85 | 3.5 | 32/126 | 25.4 | 9/44 | 20.5 |
| July | 12/829 | 1.4 | 91/477 | 19.1 | 39/184 | 21.2 |
| August | 31/1735 | 1.7 | 1/19 | 5.3 | 1/7 | 14.3 |
| September | 38/1738 | 2.2 | 2/3 | 66.7 | 0/6 | 0 |
| October | 17/1083 | 1.6 | | | | |
| Totals | 101/5470 | 1.8 | 126/625 | 20.2 | 49/241 | 20.2 |

One of 44 melanica and 2 of 223 Aulacigaster leucopeza tested did not recover. An attempt to induce sensitivity in melanogaster by inoculation of an extract from the two sensitive Aulacigaster was unsuccessful. Small numbers of other Drosophila species were tested, but no sensitivity was observed. These species, followed by the number of flies tested, were as follows: putrida (25), buskii (18), robusta (18), algonquin males (12), hydei (12), quinaria (3), macrospina (3), transversa (1), and funebris (1).

The percentages of sensitivity obtained for the entire five month period agree well with those obtained by D. L. Williamson (1959, 1961). He found that 1.6% of 6301 melanogaster collected in Lincoln during August and September of 1957 were $CO_2$ sensitive. Percentages of sensitive affinis collected in Lincoln were 24% during June-August, 1957; 25% in June, 1958; and 19% in June, 1959. Apparently the frequency of sensitivity in this population has remained fairly stable, 1 to 2% in melanogaster and between 19 and 25% in affinis.

It also appears that when large samples of flies were tested over a long period of time, as in melanogaster from July to October, very little seasonal variation occurred. This may indicate that the virus present in these flies is temperature-resistant, since a temperature of 30°C results in the apparent destruction of temperature-sensitive viral particles in the oogonia of sensitive females (L'Héritier, 1958). Temperatures in Lincoln surpass 30°C quite frequently during July and August.

Six melanogaster, 39 affinis males, and 15 affinis subgroup females classified as sensitive subsequently recovered from the exposure to $CO_2$. Four affinis females which recovered 30 minutes to one hour after exposure were isolated in vials containing food and allowed to lay eggs. The progeny of these were collected and tested with $CO_2$. Three of these females produced both resistant and sensitive offspring, indicating that they were probably nonstabilized. The offspring from the other female exhibited symptoms similar to those seen in D. melanogaster flies having the gene Dly for delayed recovery to $CO_2$ (McCrady and Sulerud, 1964). These included a recovery time of 1/2 to 2 hours when tested at 14°C, uncoordinated movements during and after the recovery period, and very slow recovery or lethality when tested at 30°C. A strain of these flies has been established, and the delayed recovery response has been expressed in all flies tested through three generations. Crosses are underway to determine whether this characteristic in D. affinis is also due to a chromosomal factor.

References: L'Héritier, Ph., 1958. Adv. Virus Res. 5:195.
McCrady, W. B. and Sulerud, R. L., 1964. Genetics 50:509.
Williamson, D. L., 1959. Ph.D. Thesis, Univ. of Nebraska.
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Perreault, W. J., H. Gay and B. P. Kaufmann
University of Michigan, Ann Arbor. Base composition of DNA in heterochromatin of Drosophila melanogaster.

In defining the biochemical properties of the heterochromatic (heteropycnotic) portions of the chromosomes of D. melanogaster, we have undertaken an analysis of the base composition of DNAs in XO, XX, XY, and XXY flies. The amount of heterochromatin/total chromatin ranges from about 21.2% for XO males to 31.0% for XXY females. To determine the GC content of each of the types listed above, DNA was extracted from 5-10 grams of flies by the method of Mead. Perchloric acid hydrolysates were chromatographed on Whatman #1 paper in an isopropanol-HCl system. Relative amounts of free bases were calculated from optical density measurements of the eluted spots. The results obtained indicate that the base composition of the extracted DNA is not markedly different among the karyotypes studied. It appears, therefore, that the DNA of heterochromatin is not greatly different from that of euchromatin with respect to base composition, even though small changes in GC content lie beyond the resolving power of the methods used. Since our data were extensive - involving several repetitions in the analysis of each karyotype - they indicate that any possible difference in base composition between euchromatin and heterochromatin among these karyotypes could not be greater than 10%. (This work was supported by N.I.H. Grant GM-10499.)

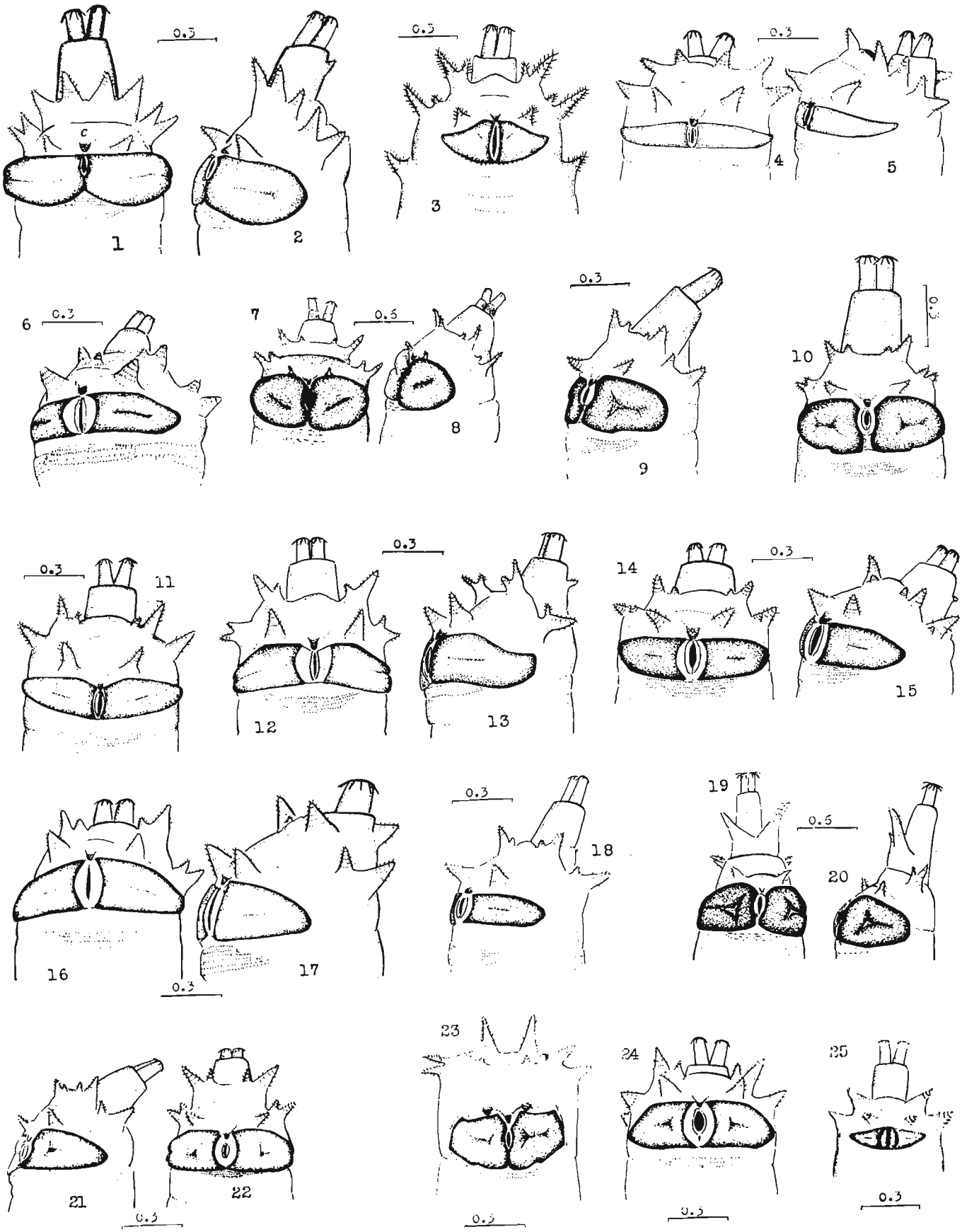
Yalvac, S. Atatürk University, Erzerum, Turkey. Variation in the larval anal organ in various Drosophilidae.

The existence of a larval anal organ, which darkens and becomes conspicuous with several reagents, has been reported by various authors (e.g. Stark, 1918; Wheeler, 1947; Gloor, 1949). The osmoregulatory function of this organ was reported by Gloor and Chen (1950), who used silver nitrate to make the anal organ darken. Waddington (1959) showed that the size of this structure varies in D. melanogaster reared in different salt concentrations.

The expectation that interspecific differences in the form of the anal organ might be useful taxonomically prompted the investigation of this structure in laboratory stocks in a number of species of Drosophila and related genera. Third instar larvae taken from stock bottles (corn meal, molasses, agar medium, with Tegosept) were exposed to 70% alcohol, which causes progressive darkening of the whole larva, during which the anal organ becomes clearly visible. A small amount of Carnoy's Solution was added to prevent wrinkling. At a suitable stage of darkening (varying with species) camera lucida drawings were prepared to illustrate the structure. (This work was done during 1957-59 in the Department of Zoology of the University of Nebraska. The author is indebted to Dr. Marshall Wheeler of the University of Texas for most of the stocks.)

Twenty species of Drosophila and one each of Chymomyza, Scaptomyza, and Zaprionus were investigated. Closely related species were generally not very different from each other as to form of the anal organ, and one might doubt whether environmentally caused variation might sometimes override such slight interspecific differences as were observed. On the other hand, members of different genera and subgenera and of species groups within the subgenus Drosophila did show substantial differences. An account of this variation was presented at the XI International Congress of Genetics. However, illustrations of representatives of more or less distinct types are presented here to make then available to Drosophila workers.

Figures: (1-2) D. victoria (Pholadoris), ventral and lateral views; (3) D. busckii (Drosophila); (4-5) D. duncani (Hirto drosophila); (6) D. melanogaster (Sophophore); (7-8) D. americana (Drosophila, virilis gp.); (9-10) D. hydei (Dros. repleta gp.); (11) D. tripunctata (Dros.); (12-13) D. immigrans (Dros.); (14-15) D. funebris (Dros.); (16-17) D. pallidipennis (Dros.); (18) D. guarani (Dros.); (19-20) D. robusta (Dros.); (21-22) D. putrida (Dros., testacea gp.); (23) Chymomyza procnemis; (24) Zaprionus vittiger; (25) Scaptomyza disticna.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Spofford, Janice B. University of Chicago, Illinois. Variegation in progeny of mothers homozygous or heterozygous for rearrangement.

Hessler (1961 Genetics 46:463-484) and Spofford (1958 Proc. 10th Int. Cong. Genet. 2:270) have both recorded that offspring receiving the $w^m$ allele from a mother homozygous for the rearrangement $Dp(1:3)w^{m\ 264.58a}$ have more eye pigment and are thus less extremely variegated than offspring of the same genotype who received their $w^m$ allele from a mother heterozygous for the Dp-bearing and a structurally normal third chromosome. This work preceded the isolation and characterization of the closely-linked Su-V (Suppressor-of-Variegation) locus, the variegation-enhancing + allele of which was present in some of the third chromosomes in these studies while the variegation-suppressing Su-V allele was present in the others. Because of the strong maternal as well as direct effect on the extent of variegation attributable to this locus, it seemed desirable to repeat the test of maternal effect of homozygosity versus heterozygosity of the duplication itself in flies of known Su-V genotype.

Accordingly, four sets of mothers were prepared from the following crosses, each initiated as 10 or more pair matings:

1) y w/Y;+ Dp ♀ x $Y^L$ w y•$Y^S$ $y^+$/Y;+ Dp ♂ ⟶ y w/Y;(+ Dp/+ Dp or + Dp/+ +) ♀

2) y w/Y;+ Dp ♀ x y w/Y;Su-V Dp ♂ ⟶ y w/Y;(+ Dp/Su-V Dp or + Dp/Su-V + or + +/Su-V Dp) ♀

3) y w/y w;Su-V Dp ♀ x $Y^L$ w y•$Y^S$ $y^+$/Y;+ Dp ♂ ⟶ y w/$Y^L$ w y•$Y^S$ $y^+$;(Su-V Dp/+ Dp or Su-V Dp/+ + or Su-V +/+ Dp) ♀

4) y w/y w;Su-V Dp ♀ x y w/Y;Su-V Dp ♂ ⟶ y w/y w;(Su-V Dp/Su-V Dp or Su-V Dp/Su-V +) ♀

Several variegated daughters from each pair mating were themselves pair-mated with y w/Y;+ + ♂♂, all at the same time. Data from progeny of Dp/Dp and Dp/+ sisters were analyzed. Note that, except for Dp constitution, the sisters whose progenies were compared had identical sex chromosome and Su-V locus genotypes. Each pair of parents was transferred to fresh Carpenter's medium after seven days, all cultures being kept at 24°C.

Both sons and daughters were scored for presence or absence of eye pigment. A random sample of no more than 10 pigmented flies of each sex from each culture was then graded as to approximate area of eyes pigmented, on a scale ranging from 0 for only one pigmented facet on either side to 200 for full pigmentation in both eyes. The results are summarized in the following table:

| | Offspring (all Dp/+) | | Dp/Dp mother | Dp/+ mother | Probability of difference[a] |
|---|---|---|---|---|---|
| y w/Y;+/+ | y w/Y ♀ | av. grade | 99.3 (84)[b] | 80.7 (116) | > .05 |
| | | % $w^m$ | 97.2% (141) | 50.5% (301) | > .05 |
| | y w/Y ♂ | av. grade | 11.1 (45) | 9.3 (80) | > .05 |
| | | % $w^m$ | 60.3% (131) | 31.4% (334) | > .05 |
| y w/Y;+/Su-V | y w/Y ♀ | av. grade | 160.0 (143) | 177.0 (190) | > .05 |
| | | % $w^m$ | 100% (244) | 50.3% (435) | > .05 |
| | y w/Y ♂ | av. grade | 52.6 (108) | 77.3 (161) | > .05 |
| | | % $w^m$ | 57.5% (266)**[c] | 49.6% (417) | ≪ .01 |
| y w/XYw;Su-V/+ | y w/y w ♀ | av. grade | 30.9 (112) | 36.7 (68) | > .05 |
| | | % $w^m$ | 90.6% (212)** | 44.2% (174) | > .05 |
| | y w/Y ♂ | av. grade | 65.0 (109) | 52.2 (58) | > .05 |
| | | % $w^m$ | 95.6% (183) | 38.7% (191) | ≪ .01 |
| y w/y w;Su-V/Su-V | y w/y w ♀ | av. grade | 63.4 (67) | 77.2 (48) | > .05 |
| | | % $w^m$ | 100% (137) | 47.6% (145) | > .05 |
| | y w/Y ♂ | av. grade | 66.7 (58) | 87.1 (29) | > .05 |
| | | % $w^m$ | 100% (94) | 49.5% (101) | > .05 |

[a] Note that the expected % $w^m$ in progeny of Dp/+ mothers is half of the % $w^m$ in progeny of Dp/Dp mothers. Grade differences were tested both by paired-comparison t-tests and one-way analyses of variance.

[b] In parentheses are the total numbers on which the values are based.

(c) Heterogeneity between sets derived from different initial pair matings is indicated by ** if at the 1% level of significance, * if at the 5% level.

Clearly, offspring of Dp/Dp mothers are not consistently more often or more heavily pigmented than are Dp/+ offspring of Dp/+ mothers. In the two instances in which the compared values differ significantly, the differences are opposite in sense. The low penetrance of $w^m$ in sons of y w/Y,+ Dp/Su-V Dp mothers is ascribable to a small number of unusually prolific sibships among a larger number of less fertile groups with high penetrance. The low penetrance in sons of Dp/+ mothers who were y w/$Y^S$ w y$^+$Y$^L$y$^+$;Su-V/+ may have resulted from a preponderance of Su-V +/+ Dp among the mothers employed. Otherwise, the data are easily and satisfactorily interpreted in terms of the known maternal and direct effects of both extra Y-heterochromatin and genotype at the Su-V locus.

It seems reasonable that the earlier data interpreted as evidence for a maternal effect of homozygosity versus heterozygosity of the rearrangement may also be explained as due to segregation at the Su-V locus.

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Grace, D. University of California, Los Angeles. Preliminary localization of ICR 100 induced dumpy lethals.

Three phenotypically distinct dumpy lethals, olv, ol, lv, have been tentatively localized at two specific subloci (Carlson, 1959; Southin and Carlson, 1961). These alleles express extreme phenotypes when heterozygous with the $ov^1$ mutant. The olv mutant shows a strong wing effect, oblique (o) and a thoracic effect, vortex (v), which causes pronounced disruption of the bristle pattern and thoracic pits or eruptions. The ol mutant is expressed as a strong oblique wing effect and lv is expressed as a strong vortex effect. The olv allele has been mapped between $o^2$ and $cm^2$. The lethals ol and lv are located between $cm^2$ and $ov^1$. They have not been separated as they are lethal when combined in the trans configuration.

Preliminary localization of dumpy lethals induced by ICR 100, a quinacrine mustard, suggests that these alleles all map within the particular subloci previously determined (table 1). The position of these alleles was determined by a "four-point" test with respect to one dumpy allele $cm^2$ or $ov^1$ and two outside markers echinoid, ed, at 11.0 and clot cl, at 16.5. Dumpy maps at 13.0.

Tests to determine the definite location are being carried out with other alleles of the dumpy series. The crossovers which have been confirmed lend support to the theory that the dumpy complex can be separated into discrete regions which affect a predictable dumpy phenotype.

Table I: Localization of dumpy lethals.

| $P_1$ Female | $P_1$ Male | Verified Single Crossover | Total Progeny | Curly | Map-Order |
|---|---|---|---|---|---|
| $lv^{78}_{35}$/ed $ov^1_1$ cl | ed $olv^{57}_{57}$ cl/Cy | 1 | 57,815 | 41,398 | $lv^{78}_{35}$ - $ov^1_1$ |
| $lv_{88}$/ed $ov_2$ cl | ed $olv_{57}$ cl/Cy | 2 | 41,210 | 29,784 | $lv_2$ - $ov_{88}$ |
| $ol_{109}$/ed $cm^2_2$ cl | ed $olv_{57}$ cl/Cy | 1 | 74,210 | 56,320 | $cm^2_2$ - $ol_{109}$ |
| $ol^{69}_{109}$/ed $cm_2$ cl | ed $olv_{57}$ cl/Cy | 1 | 32,968 | 27,485 | $cm^2$ - $ol^{69}_{109}$ |
| $olv^{69}$/ed $cm^2$ cl | ed $olv^{57}$ cl/Cy | 4 | 49.373 | 36,720 | $olv^{69}$ - $cm^2$ |

Recombination results of ICR induced dumpy lethals:

lv = thoracic vortices, homozygous lethal
ol = oblique wings, homozygous lethal
olv = oblique wings, thoracic vortices, homozygous lethal
$cm^2$ = thoracic comma

Duke, Edward J. University of North Carolina, Chapel Hill, N.C. Comparison of third-instar larval lymph protein content in seven species of Drosophila.

Recently (Hubby and Throckmorton, 1965) a study has been made, using vertical acrylamide electrophoresis, of the evolutionary relationships between adult soluble proteins within the virilis group of Drosophila. Presented here is an inter-species survey of late third-instar larval lymph protein content, as shown by starch-gel electrophoresis, of the following seven species of the genus Drosophila: melanogaster, simulans, immigrans, hydei, virilis, funebris, and subobscura. In addition, pooled samples of melanogaster, funebris and subobscura species caught in the wild in various parts of Ireland, were examined for lymph protein content.

The results of the survey are shown in fig. 1. As expected, melanogaster and simulans had very similar patterns, with the complete absence of fraction 5 in the latter, constituting the major difference between the two. This protein band was found to be present in pooled samples of all twenty-six laboratory and wild stocks of melanogaster studied. It is possible that fractions 13 and 14 in melanogaster are very similar to fractions 3 and 13 in simulans (fig. 1) except that the electrophoretic mobilities are slightly altered. The protein patterns obtained for the other five species varied quite significantly from the above two species and from each other. Drosophila immigrans exhibited the least number of fractions having only two main bands not counting the "front" (fig. 1). The other four species all exhibited approximately six to nine fractions of differing concentration and composition.

The protein patterns exhibited by cultures of melanogaster, funebris, and subobscura started from individuals caught in different parts of Ireland, were in close agreement with those of the laboratory bred stocks, and only minor differences were observed. In the case of funebris for instance, protein fraction C of the laboratory stock (see fig. 1) was found to be split into two in the wild stocks. A survey of indivudual larvae has shown that the wild stock contained a mixture of individuals having the single and double band phenotypes.

The degree of difference and similarity between the larval lymph protein patterns of the different species can be correlated, to some extent, with their chromosomal arrangement. Those species with a primitive six chromosome pair configuration differ significantly in lymph protein pattern from immigrans, similans and melanogaster which have four pairs. D. simulans and D. melanogaster are more evolved than D. immigrans and exhibit much more diversification of protein fractions. There is, however, quite a close resemblance between the pattern obtained from the Pacific 7 strain of D. melanogaster (see Duke, 1965) and that of D. immigrans. Four protein fractions of the D. immigrans pattern have similar electrophoretic mobility to fractions exhibited by D. melanogaster (fig. 1). By comparison, the lymph protein patterns from the four species containing six chromosome pairs are significantly different from those already described. D. subobscura, although having only two protein bands of exactly similar mobility to melanogaster fractions, has four others of very close mobility (see fig. 1). Four fractions in the lymph of D. hydei resemble those of D. melanogaster in electrophoretic mobilities. It may be significant that D. hydei alone of these four species contains the more evolved V-shaped chromosome. The patterns of D. virilis and D. funebris are least like that of D. melanogaster. Generally speaking, therefore, the patterns of lymph protein content of the seven species studied, corresponded with the evolutionary trends within the genus as indicated by their characteristic chromosome configurations. These electrophoretic data should be firstly considered as an extra taxonomic aid in the systematics of Drosophila, and secondly as a basis for future work in a more refined biochemical approach to problems of evolution.

References: Duke, E. J. (1965). Further studies on the inheritance of lymph proteins in Drosophila. Gen. Res. Camb. (in press).
Hubby, J. L. and Throckmorton, L. H. 1965. Protein differences in Drosophila, II. Comparitive species genetics and evolutionary problems. Gen. 52:203-215

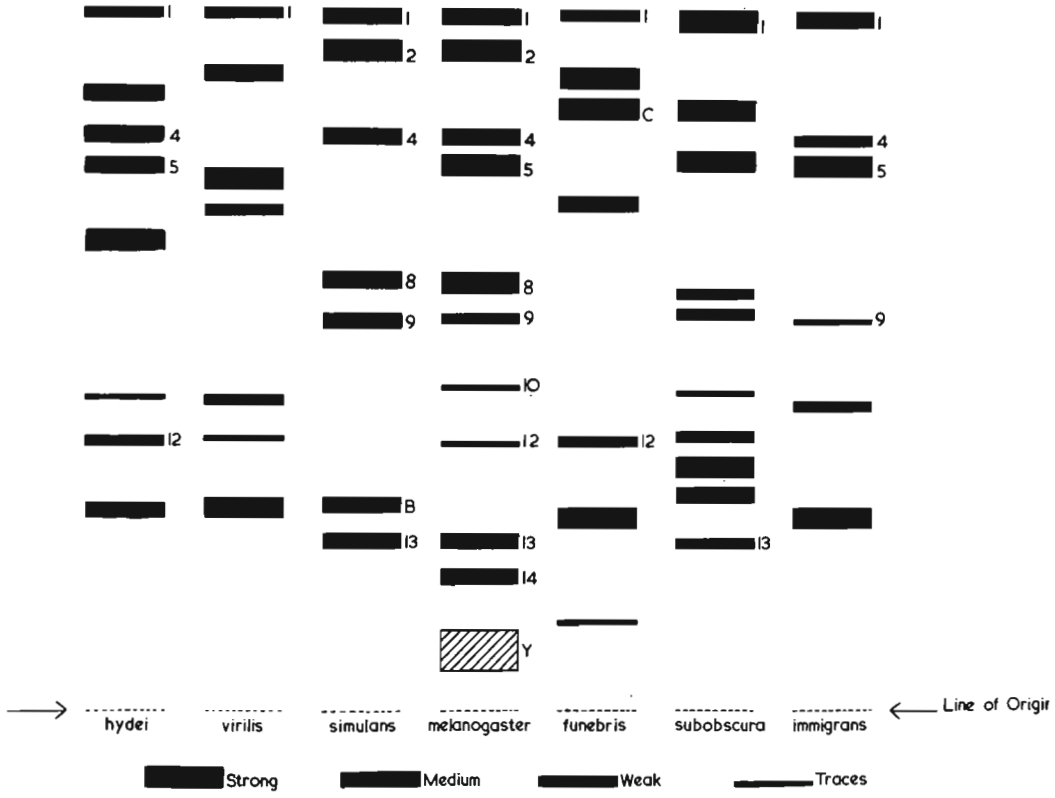



Fig. 1

Druger, M. Syracuse University, New York. The distribution of genetic potential for wing venation abnormalities in a natural population of D. pseudoobscura.

Egg samples from 25 strains derived from single females captured at Pinon Flats, Mount San Jacinto, California, were placed at 16°C and at 25°C for development. In each strain more than 1000 flies were sampled over a five generation period beginning with the $F_3$. A variety of defects were detected, including missing posterior crossvein (cve), missing longitudinal vein (lv) and extra venation (ev). The potential for producing these defects is not restricted to only a few strains but is widespread throughout the population.

Table 1: Distribution of wing venation defects

| No. of Strains | lv | cve | ev |
|---|---|---|---|
| 6 | + | + | + |
| 1 | + | + | 0 |
| 6 | 0 | + | + |
| 3 | + | 0 | + |
| 0 | + | 0 | 0 |
| 1 | 0 | + | 0 |
| 7 | 0 | 0 | + |
| 1 | 0 | 0 | 0 |

Purdom, C. E. and K. F. Dyer. M.R.C. Radiobiological Research Unit, Harwell, England. Spontaneous Mutation Rates.

Spontaneous mutation frequencies from Drosophila males reflect the response of the entire cycle of spermatogenesis to whatever processes are involved. Brood patterns will not reflect any variation in the sensitivity of germ cells, as in the case of induced mutation, but should reveal the rate at which mutations arise spontaneously in the spermatogonial stem cells. Thus, it may be assumed that the difference between sperm from old as opposed to young males lies only in the length of time the cells existed as spermatogonia, and that any difference in mutation frequency may be related to this period of spermatogonial life.

During the past four years much information has been collected on IInd chromosome mutation frequencies from 3- and 21-day old male flies. The data which are shown in Table 1, show that mutation frequency is significantly higher in offspring from the older males ($X^2 = 6.00$ $p = 0.014$). If mutation rate was constant at all germ cell stages the mutation frequency from 21-day old males should be from 2 to 2.5 times the frequency from 3-day old males. This is clearly inconsistent with the data and it must be concluded that mutation rate is not constant and that the stem cells are less sensitive than the later meiotic or maturation stages. This is similar to the pattern discovered for induced mutation and raises the possibility that spontaneous mutation during spermatogenesis mirrors the differential sensitivity pattern observed for radiation induced mutations.

Further experiments were conducted to determine the spontaneous mutation rate in stored sperm in an attempt to compare this with the spermatogonial rate. Male flies derived from an $F_1$ from two inbred lines were allowed to inseminate Cy/Bll females. These were sampled for mutations by the standard Cy/Bll technique either immediately after insemination or after various periods of storage at 10°C. The summed data of a number of experiments are shown in Table 2.

Contrary to expectation, there was no significant evidence of an increase in mutation frequency with ageing of the male. A significant increase in mutation frequency was observed, however, following storage of spermatozoa. The data of all experiments were fitted to the model: $D(y) = \alpha + \theta_1 x_1 + \theta_2 x_2$ where y is the mutation frequency and $x_1$ and $x_2$ the age of the male and duration of sperm storage respectively. The mean value for $\theta_1$, the assumed mutation rate for spermatogonia, was $0.034 \pm 0.027$% mutations per week, which does not differ significantly from zero. However, individual values showed heterogeneity between experiments and one possible explanation is that a low but positive mutation rate in spermatogonia was obscured in some experiments by exceptionally high mutation frequencies in spermatozoa from early ejaculates. Some evidence does exist for this high initial frequency (1, 2,).

Values for $\theta_2$, the mutation rate in stored spermatozoa, showed no evidence for heterogeneity and gave a mean value of $0.040 \pm 0.015$% mutations per week. This is much lower than the figure quoted by Muller (3) for sex-linked recessive lethals (0.06% per week) when due allowance is made for the greater length of the IInd chromosome. It is also not significantly different from the mean value for $\theta_1$.

Neither the mutation rate calculated for spermatogonia, nor that for mature spermatozoa was adequate to explain the initial (Brood I unstored) mutation frequency. This suggests that some intermediate germ cell stage is particularly sensitive to the processes involved in spontaneous mutation. Alternatively, the high initial rate may be due to a high incidence of "partial" or mosaic damage amongst spontaneous mutations. Recessive lethal mutations arising in this way would only be revealed after a further operation during which segregation would occur. Further tests on the stored spermatozoa groups suggested that the rate of origin of "partial" damage was about equal to that for "complete" lethals - this was still inadequate to explain the initial high mutation frequency.

References: 1. Lamy, R., J. Genet., 48:223-236, 1947
2. Ives, P. T., Genetics, 48:981-996, 1963
3. Muller, H. J., Proc. 2nd U. N. Int. Conf. Peaceful Used of Atomic Energy, 22: Geneva, 313-320, 1958

Table 1. IInd chromosome recessive lethal mutations from young and old Drosophila males mated in a 3-day brood sequence

| Age of male (days) | 3 | 21 |
|---|---|---|
| Number of tests | 10,434 | 8,370 |
| Number of lethals | 34 | 48 |
| % ± S.E. | 0.326 ± 0.056 | 0.573 ± 0.083 |

Table 2. IInd chromosome recessive lethal mutation of spontaneous origin in $F_1$ hybrid Drosophila

| Brood | | Sperm storage in weeks | | | |
|---|---|---|---|---|---|
| | | 0 | 4 | 6 | 8 |
| I | Tests | 13,017 | 4,543 | 927 | 3,750 |
| | lethals | 76 | 36 | 11 | 34 |
| | % | 0.58 | 0.79 | 1.19 | 0.91 |
| VI | Tests | 7,450 | 1,072 | 2,493 | 1,564 |
| | lethals | 45 | 9 | 25 | 15 |
| | % | 0.61 | 0.84 | 1.00 | 0.96 |
| XI | Tests | 1,829 | - | 277 | 1,124 |
| | lethals | 12 | - | 3 | 4 |
| | % | 0.66 | - | 1.08 | 0.34 |

Friedman, L. D. and W. C. Kiriazis. Hiram College, Ohio. Chemically induced viability mutants in D. melanogaster.

Studies are in progress on the relative frequency of chemically induced sex-linked lethal and detrimental mutations and their effect on the viability of D. melanogaster. Tests were made on 3925 X-chromosomes from Basc and Canton-S strains that were treated with the monofunctional alkylating agent ICR 100. The treated males were injected with 0.1% ICR 100 in 0.4% saline. Parents were discarded after 3 days so that the effects measured were on mature sperm. The experimental design and analysis is the same as used previously for similar studies with X-rays (Friedman, 1964, Genetics 49:689-699).

Estimates were made on the proportions of complete lethals and the genetic load of lethals and detrimentals induced.

(1) The complete sex-linked lethal frequency induced by this compound in our experiments has been on the average of about 4.5%. There is no significant difference between the lethal rates induced in the + and Basc chromosomes. This differs from the results obtained with X-rays.

(2) The ratio of the genetic load from non-lethal detrimental mutants to that from lethals was .390. The load is computed as the product of the frequency and the average effect on viability. It is a much higher value than any effect of the same kind that has been established for X-ray. This indicates a much higher detrimental effect in relation to lethals that has been induced by the chemical mutagen in comparison to the effect caused by X-ray.

Further studies are in progress including the determination of the induced mosaic lethal frequency. (This work is supported by U.S. Public Health Service Grant GM 11354.)

Khan, A. H. University of Cambridge, England. The mutagenic effect of N-mitroso-3-methylaminosulpholane.

Adult feeding (for 24 hours) of starved one-day-old males with a freshly-prepared solution containing 5% glucose, and N-nitroso-3-methylaminosulpholane (NMAS), is found to produce a significant increase in sex-linked recessive lethal mutations on Drosophila spermatozoa (sampled by mating treated males individually to two virgin Muller-5 females for 3 days).

An increase in the concentration of NMAS from 0.1% to 0.5% does not increase the yield of sex-linked recessive lethals (Table 1). An indication for a delay in the mutagenic effect of NMAS is seen in Table 1, where 6.5% of the tested non-lethal F2 cultures show F1 lethal-mosaicism after the 0.5% NMAS treatment, compared with 3.6% from the control; the corresponding F3 lethal frequencies are 5.9% for NMAS, and 0.36% for the control.

Table 1: Complete and mosaic sex-linked recessive lethal frequencies in Drosophila males after adult feeding treatments with N-nitroso-3-methylaminosulpholane.

| | Concentration (%) | Control | 0.1 | 0.5 |
|---|---|---|---|---|
| | Duration of treatment (hrs.) | 24 | 24 | 24 |
| | Survival (%) | 100 | 100 | 100 |
| | No. males examined | 56 | 69 | 65 |
| $F_2$ | Average no. chromosomes examined/male | 9 | 6 | 11 |
| | No. chromosomes examined | 510 | 433 | 614 |
| | No. lethal chromosomes | 1 | 16 (from 15 ♂) | 21 (from 21 ♂) |
| | Complete lethals (%) | 0.2 | 3.8 | 3.4 |
| | No. non-lethal $F_2$ cultures examined | 55 (arising from 55 ♂♂) | - | 62 (arising from 62 ♂♂) |
| | Average no. females examined/ non-lethal $F_2$ culture | 10 | - | 11 |
| | No. non-lethal $F_2$ cultures yielding at least one lethal in $F_3$ set | 2 | - | 4 |
| $F_3$ | Cultures showing mosaicism (%) | 3.6 | - | 6.5 |
| | Total no. $F_2$ females examined | 547 | - | 563 |
| | No. lethal-bearing $F_2$ females | 2 | - | 33 |
| | Lethals in $F_3$ (%) | 0.36 | | 5.9 |

Gersh, E. Sutton, University of Pennsylvania. Centromere of chromosome 3 in D. melanogaster located to right of ri.

Females of constitution $w^e/w^e$; Dp (1; 3) 264-58a/ri $p^p$ were mated with $w^e$/Y; ri $p^p$/ri $p^p$ males. The Dp is a piece of X including the w locus, and its insertion is known to be in 3L between in and the centromere. It causes a white-mottled ($w^m$) phenotype. $w^e$ $p^p$ eyes are directly distinguishable from $w^e$, and $w^e$ $w^m$ $p^p$ flies can be distinguished from $w^e$ $w^m$, usually directly, always by progeny-testing.

If the order of the 3 heterozygous markers were ri Dp $p^p$, the above cross would yield $w^e$; +++ and $w^e$; ri $w^m$ $p^p$ flies only as rare double crossovers (expected frequency < 1/10,000), while $w^e$; $p^p$ and $w^e$; ri $w^m$ flies would appear as a result of single exchange in region 1. If the order were Dp ri $p^p$, the reverse results would be obtained.

In a total of 1262 progeny, the following phenotypes were found in addition to the parental types:

| | | |
|---|---|---|
| $w^e$; $w^m$ $p^p$, 3 | $w^e$; ri $w^m$, 6 | $w^e$ 17 |
| $w^e$; ri , 4 | $w^e$; $p^p$ , 6 | |

All of the 6 $w^e$; ri $w^m$ flies had some wild-type pigmentation on an eosin background, and were clearly not $p^p$. Four of the 6 $w^e$; $p^p$ flies were obtained as virgins and progeny-tested by mating with $w^e$; ri $p^p$: 3 proved to be genetically $w^e$; $w^m$ $p^p$ (offspring had peach mottling on a $w^e$; $p^p$ background); the fourth gave no mottled offspring and was, therefore, genuinely $w^e$; $p^p$. Six of the $w^e$ flies were obtained as virgins and similarly progeny-tested: all of them produced mottled offspring and were, therefore, non-crossovers.

Thus, only two pairs of complementary crossover types were identified, and these were of the kinds expected as single crossovers if the order of markers were ri Dp $p^p$. We can, therefore, assume that ri is to the left of the Dp, and knowing that the centromere is to the right of the Dp, we can assert that it is also to be right of ri.

Work supported by PHS Grant GM 11379-02.

B. Leigh, State University, Leiden, The Netherlands. An unusual mosaic.

One day old $X^{C2}$, y B/$sc^8$·Y males were irradiated and mature sperm were sampled by mating them to y $sc^{S1}$ In49 $sc^8$; bw; st $p^p$ females. Exceptional $F_1$, y ♂♂ were tested for fertility by backcrossing them to females from the maternal stock. In one fertile cross the $F_2$ consisted of 45 $y^+$ ♂♂, 21 y ♀♀, and 23 $y^+$ ♀♀. On further testing it was found that the non-yellow females carried the $y^+$ marker on chromosome IV and the $F_2$ males could be divided into two classes, those which carried a $y^+$ marker on the Y chromosome and another $y^+$ on chromosome IV and those which only carried $y^+$ on the Y chromosome.

To explain the presence of two $y^+$ markers in the gonads of the phenotypically y exceptional male, it has been assumed that a chromatid exchange occurred in the male pronucleus. This resulted in the transfer of $y^+$ from one of the Y chromatids to one of the chromatids of chromosome IV. At the first mitotic division one daughter nucleus received the unexchanged $sc^8$·Y chromatid and the exchanged IV,$y^+$ chromatid. The other daughter nucleus received the exchanged non-$y^+$ Y chromatid and the unaltered IV chromatid. The first daughter nucleus later developed into the germ cell line and possibly a portion of the endoderm tissue, while the latter daughter nucleus developed into the entire hypoderm.

Thus, the mosaic $F_1$ male provided evidence for the occurrence of radiation induced chromatid exchange and also indicated that differences arising at the first cleavage division do not necessarily lead to the formation of phenotypically half and half mosaics.

Church, R. and F. W. Robertson. Institute of Animal Genetics, Edinburgh, Scotland.
Biochemical comparisons of growth in selected lines of D. melanogaster.

Ten lines of D. melanogaster, which have been selected for large or small body size, fast or slow development time on different chemically defined media, have been compared with the unselected stock, in terms of their biochemical composition at successive stages of growth. The data include estimations of wet and dry weight, lipids, protein, free amino-acids, RNA and DNA for the egg, several larval stages, pupa and adult. In addition, in the case of the unselected stock, the RNA, derived from successive stages, was fractionated by sucrose gradient centrifugation, while the DNA from adults was characterised in terms of molecular weight, Tm and base ratio. The growth and biochemical data represent the most extensive information of this kind for any insect, while the large differences in size and development time between these lines make them unique material for a comparative biochemical analysis.

The selected lines can be classified into two groups in which: (a) there is a strongly correlated change in body size and duration of development and (b) only body size has changed. This contrast in selection response was reported by Robertson (1963) who interpreted it in terms of alteration or constancy of the "critical size" at which larvae attain the capacity to pupate, even if they are removed from food. It turns out that the distinction based on the presence or absence of correlation between body size and development time is quite fundamental. Where development time does not differ from the controls, the DNA content per individual is constant, while the RNA/DNA ratio varies with respect to the size of the adult. On the other hand, where there are correlated changes in both characters, there are also correlated differences in the DNA content per adult, while the RNA/DNA ratio is comparatively unaltered. This suggests that, in the first case, it is cell size and, in the second, cell number which has been altered by selection.

Reference: Robertson, F. W. (1963) The ecological genetics of growth in Drosophila. 6. The genetic correlation between the duration of the larval period and body size in relation to larval diet. Genet. Res. 4:74.

Chauhan, N. S. and F. W. Robertson. In Institute of Animal Genetics, Edinburgh, Scotland.
Quantitative inheritance of red eye pigment in D. melanogaster.

A general survey has been completed of genetic variation in the quantity of red eye pigment in D. melanogaster. The pigment, which comprises the three drosopterins, is extracted from individual flies which are also scored for eye width and thorax length. There is generally a high genetic and environmental correlation between pigment content and eye size, which can vary, to a considerable extent, independently of thorax length. However, this correlation presents interesting aspects since, although it is very obvious when eye and body size vary, due to differences in larval food supply, comparable variation in size due to temperature is without effect on the pigment content. Comparison of the variance of pigment content in wild populations, inbred lines and the $F_1$ of crosses between inbred lines, suggests that about 40% of the variance, which is independent of eye size, is due to genetic segregation. Replicated selection for high and low pigment content has led to about 60% difference after 8 generations. The response tends to be asymmetrical, since selection is more effective for low than high pigment content. In comparisons of pigment content in three inbred lines, derived from the same population, one has the same pigment content as the original population, in another the content is reduced by 30% and, in the third, by 90%. The 90% reduction is apparently due to a strictly additive combination of the effects of two recessive genes, located on respectively chromosomes II and III. The studies are being extended to a quantitative analysis of associated pteridines in the selected lines, crosses, inbred lines etc.

Remondini, D. J. and G. D. Hanks. University of Utah, Salt Lake City. Location of a second chromosome factor, RD2, as one of the Recovery Disrupter (RD) components.

It was observed in repeated tests that a second chromosome heterokaryotype (RD2/+) produced as high a percentage of females as the homokaryotype (RD2/RD2). Two chromosomes without RD activity were used in the analysis. One carried the markers fes 1t, and the other carried the markers Bl $L^2$. Females which were heterozygous for fes 1t but with RD background were mated to males heterozygous for Bl $L^2$ with RD background and the resulting Bl $L^2$ male progeny were mated to stock females containing fes and 1t (to determine the fes 1t constitution of the male) and to 5 tester females in order to detect the presence of the second chromosome RD factor. Males that carry the RD factor almost invariably give percentage female values greater than 60%. Males were considered adequately tested (hence included in the data) if they produced at least 200 progeny. Pooled results (Table 1) of repeated experiments show a clearcut association of RD activity (defined as 60% females or above) with both the $fes^+$ and $1t^+$ markers ($p < .001$ in each case). Since fes is at 5 map units the location of the RD factor is clearly in the left arm. The location (based on 78 progeny tested) is estimated to be at approximately 32. It is suggested that the factor be named RD2. (Supported by NSF Grant GB-456.)

Table 1. Crossover data from heterozygous females.

| Constitution of Maternally Derived Chromosome | | | No. of Males Tested |
|---|---|---|---|
| + | RD2 | + | 25 |
| fes | + | 1t | 20 |
| + | + | 1t | 7 |
| fes | RD2 | + | 11 |

| Constitution of Maternally Derived Chromosome | | | No. of Males Tested |
|---|---|---|---|
| + | RD2 | 1t | 10 |
| fes | + | + | 2 |
| + | + | + | 1 |
| fes | RD2 | 1t | 2 |

References: Erickson, J. 1965, Genetics 51:555-571
Hanks, G. D. 1964, Genetics 50:123-130

Details of this study may be found in: Remondini, D. J. 1964 "Second Chromosome Studies of a Case of Meiotic Drive in Drosophila melanogaster". M.S. Thesis, University of Utah Library, Salt Lake City, Utah.

Friedman, Lawrence D. Hiram College, Ohio. X-ray induced viability effects in spermatogonial cells.

Previous studies of the relative frequency of X-ray induced sex-linked lethal and detrimental mutations and their effect on viability have been done on mature sperm. Experiments, using basically the same experimental design and analysis as those previously, have also been carried out in relation to the effects on the spermatogonial cells. 1-3 day old males of the Basc and Canton-S strains were irradiated with 6000 r of X-rays which were administered in two equal fractions separated by a 24-hour interval. These males were then mass mated to virgin females for a fifteen day period before being entered into the actual experimental design (Friedman, 1964). A total of 1940 chromosomes were tested in this way. The induced sex-linked lethal frequency averaged 2.9%. This effect in the gonial cells resulting from a dose of 6000 r is comparable to the frequency in mature sperm exposed to 1000 r. The detrimental to lethal load ratio (D:L) was .115 which does not differ significantly from the D:L of .125 previously reported for mature sperm treated at a dose level of 1000 r. (This work was supported in part by a grant from the Hiram College Research Fund.)

Robertson, F. W. Institute of Animal Genetics, Edinburgh, Scotland.
Adaptation and sexual isolation.

Populations of D. melanogaster, derived from the cage Pacific population, have been adapted to a new diet containing the chelating agent, EDTA, which reduces growth and survival with increasing concentration (Steffensen 1957). The adapted strains survive perfectly at levels of EDTA which are lethal for the original population. Chromosome analysis suggests that a number of loci are involved and also reveals striking epistatic interaction. Under optimal conditions, the original population and the populations adapted to EDTA, grow at similar rates, but under crowded, competitive condition, the performance of the foundation population is superior to that of the others. The $F_1$ of crosses is roughly intermediate and therefore does not grow as well as either the original population under crowded conditions, or the EDTA-adapted population on the EDTA medium. Since the relative fitness of foundation and adapted populations is influenced by the diet, we have suitable material for discovering whether sexual isolation will develop, with different levels of gene flow between populations living in two environments to which they are particularly adapted. Accordingly replicate pairs of cages, supplied with either the ordinary medium, (lacking the usual dried yeast component), or the EDTA medium were joined by glass tubes of different diameters. Flies of either the original or an adapted population were introduced into the appropriate cage and the populations have been allowed to run for 30-40 generations. At intervals samples were withdrawn from either cage of each combination to test for performance on alternative diets and also for sexual isolation. Differences in growth on alternative diets became progressively less and cannot now be detected. The rate of approach to this similarity was correlated with the tube diameter, which clearly influenced gene flow. Originally there was no evidence of sexual isolation between the control and the EDTA population, nor has there been the least evidence of such isolation in any of the experimental series. In addition, selection for positive assortative mating, in conditions in which flies of the original and the EDTA-adapted population are given an equal chance of mating, has failed, after 15 generations, to provide any evidence of sexual isolation. Statements occur in the literature to the effect that restricted gene flow between populations of Drosophila adapted to different environments is sufficient to promote effective sexual isolation. These data suggest, however, that such assertions are merely speculative.

Reference: Steffensen, D., 1957, Nature 180:300.

Hackman, R. and S. Lakovaara. University of Helsinki, Finland.
The temperature sensitive period of ommatidium determination in rolled mutants of D. melanogaster.

In a paper dealing with the effect of the rolled (rl) locus in D. melanogaster, one of the authors (Lakovaara 1963, Ann. Acad. Sci. Fenn. A, IV, 73:1-58) established that incubation at temperatures above 18°C has a detrimental effect on size and structure of the compound eye. The size of the eye reached a minimum at 26°C with rl in a hemizygous condition. Making use of this sensitivity to rearing temperature it was attempted to elucidate at what stage of development the mutant rl allele influences eye formation.

In the parent crossing the stocks rl/rl and Cy/Df(2)rl$^{10a}$ lt cn were used. The fertilized females were allowed to lay eggs for a short period in culture bottles. The cultures were incubated at 26°C, but every 24 hours a number of them were transferred to 18°C and left there, allowing the flies to complete their development. When the flies emerged, the rl/Df(2)rl$^{10a}$ lt cn individuals were collected, and the mean basal surface of the eyes in each transfer group determined and plotted graphically as a function of developing time.

The graph so obtained indicated that flies reared at 26°C reach their temperature sensitive period (T.S.P.) 36 hours after hatching from the eggs, and that the T.S.P. ends at 96 hours after hatching, coincident with puparium formation. The greatest sensitivity was found at about 60 hours after hatching, when the flies were in the beginning of their third larval instar. At this stage the cells of the eye disk are apparently starting to differentiate (Bodenstein, D. 1950, Biology of Drosophila, ed. M. Demerec). Accordingly it seems reasonable to suppose that the mutant rl allele acts by a temperature sensitive enzyme or other mediator directly on the differentiation and/or growth of the cells forming the ommatidia.

Doane, W. W. Yale University, New Haven, Connecticut. Disc electrophoresis of α-amylases in individual Drosophila.

A new method was used to analyze amylases separated by disc electrophoresis and derived from individual flies, larvae, or their tissues (see technical note, this issue). Amy alleles representing 8 different banding patterns were studied in homozygotes of D. melanogaster; various laboratory strains of D. hydei and D. nigrohydei were also examined. Relative activities were determined microdensitometrically for the different isozymes separated from a given individual.

Terminology of Amy alleles was made to conform to the system worked out by Kikkawa (1964, Jap. J. Genet. 39:401) for his agar gel studies, even though discrepancies were found. Major bands from each homozygote are indicated by superscripts with "1" the fastest and "6" the slowest migrating band (see Figure 1). A very weak band was found to precede the migration of each major band, rather than to follow as in agar gel studies. Thus, for the Amy alleles in melanogaster, a total of 7 bands were found and labelled from 0 to 6 (instead of 1 to 7, as in agar gels). Kikkawa's $Amy^4$ allele has been called $Amy^{1.4}$ since band "1" always appears and, the younger the fly, the more pronounced it is. Relative activities change during both larval and adult development and show a tendency for greater activity to shift from the faster to the slower migrating major band as age increases. Figure 1 shows the pattern typical of 4-day old adult female homozygotes in melanogaster (A to H), and a mixture of all eight types (I). $Amy^{1.2}$ is a new allele found in $adp^{fs}$ strains; $Amy^{1.3}$ was isolated from an inbred Canton-S strain; $Amy^{4.6}$ came from an $adp^{60}$ line; others were generously provided by Prof. Kikkawa. All strains were made isogenic for Chromosomes I, II and III, and co-isogenic for I and III (Amy locus being on II).

From the effects of various activators (e.g., MaCl: $GaCl_2$) and inhibitors (e.g., EDTA, PCMB, α-amylase inhibitor, glutathione), it is clear that all the bands represent α-amylases. Heterozygotes show additive effects of the allele from each parent, i.e., no hybrid enzymes, indicating that the amylases are monomers. Altering pore size of the gel in which separation occurs does not change the basic banding pattern, merely the over-all rate of migration; thus the isozymes are apparently similar in molecular size, but differ in electric

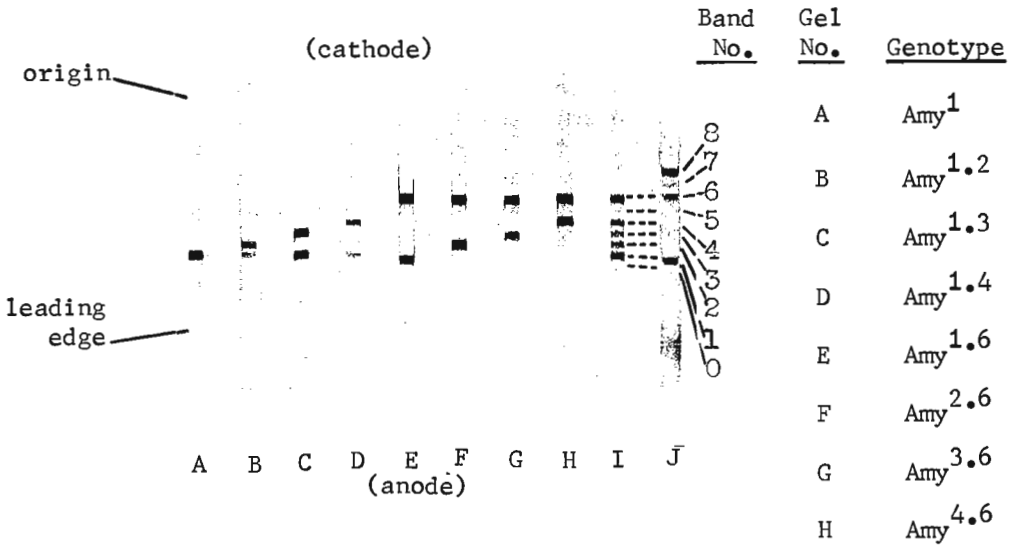



Figure 1. Banding patterns of Amy alleles in 4-day old adult females of D. melanogaster (A-H are individual homozygotes; I is a mixture of all alleles taken at 1/8 total strength of each); J provides a standard of graded activities (see text) from which total activity per fly is determined. Minor bands preceding major ones, e.g., 0 and 5, are barely visible but are recorded microdensitometrically.

change.

The banding pattern of the last gel (J) in Figure 1 results from mixing supernatants of the following sources: $Amy^{1}$ of melanogaster, our strain of nigrohydei, and Zurich and Chile strains of hydei. Each of these strains has only a single major band: "1", "6", "7" and "8", respectively. The supernatants are derived from mass collections of flies and diluted to provide a graded series of known activities. Band "7" is the weakest (equivalent to .0036 uM maltose released/min.), "6" is twice as active, "1" is three times, and "8" four times as active. With these activities as a standard, the total activity, as well as relative, for any given fly can be determined in the other gel columns.

Shiomi, T. Hiroshima University, Japan.
Effects of penicillamine feeding on the growth and radiation induced mutation rates in D. melanogaster.

Penicillamine is 8-mercaptovaline or 8,8-dimethyl cysteine and it is the most characteristic degradation product of the penicillin type antibiotics. Culture media containing 0.5-3.0 mg DL-penicillamine per ml were examined for their effect on the growth and radiation induced mutation rates with Canton-S strain. Contrary to the results of penicillin fed cases, penicillamine had no remarkable delayed effect on the growth rate. At 2 mg or less per ml concentration, it had also no effect on the emergence rate of adult flies, but when culture medium containing 3 mg concentration was employed, the emerged rate was significantly decreased. Sex-ratios (♀/♂) in the progeny produced by the fed males were significantly decreased. Hatchability of eggs fertilized by the sperm of the fed males was significantly reduced.

In the radiation experiments, penicillamine prefeeding effects on the induction of sex-linked recessive lethal mutation were inconsistent with its concentrations. 1 mg group showed similar brood pattern to the control group, and the pattern for 3 mg group was run to opposite direction. Induction of dominant lethals with X-ray irradiation for mature sperms in the inseminated females was reduced by the feeding.

In the case of penicillin feeding experiments, growth rate of flies was prolonged for one day as compared with the normal cultured conditions. On the other hand, the emergence rate was significantly higher in the fed group. The sex-ratio was not changed. Hatchability was not affected or rather increased. X-ray induced sex-linked recessive lethal mutation rates were significantly reduced. However, there were no differences in dominant lethality for sperms irradiated in inseminated females.

Thus the experimental results on the effect of penicillamine feeding seem somewhat different from the results of penicillin fed cases. The picture of contradictory results is one of an intricate network of intermediate factors and their possible interactions which determine the final yield of detectable genetic changes.

Manna, G. K. and K. Chatterjee. University of Kalyani, India.
Ganglionic metaphase chromosomes of D. malerkotliana, Parshad & Paika 1964

The larval ganglionic preparations were made following the technique of E. B. Lewis and Linda Smith Riles (DIS 34) with a modification by us for replacing the solution F of Lewis & Riles with a solution of 2% Gurr's natural orcein, o.25% fast green dissolved in 50% glacial acetic acid and 50% lactic acid (85%) of I. Oster & G. Balaban (DIS 37). Some clear mitotic complement (Fig. 1) contained five pairs of chromosomes: two pairs large, two pairs medium and one pair of small V-shaped chromosomes. The detailed account will be published elsewhere.

Fig. 1

Erk, F. C. and J. H. Sang. Poultry Research Centre, Edinburgh, Scotland.

Allelism of second chromosome melanotic tumor genes.

To test for suspected allelism of second chromosome mutants of diverse origins which produce melanotic tumors, reciprocal pair matings were made in all combinations of wild type (Caribbean, a virtually tumor-free strain), tu bw, tu bw;st su-tu, tu 55g (from Jacobs), tu $B_3$, tu $A_2$, $tu^K$, and Frd. In crosses of the other strains with Frd, a dominant lethal which is phenotypically distinct from the others, half the offspring showed Frd as expected; there was no evidence of allelism with the other loci, and the Frd data are not included in the table below. The remainder of the tumor-initiating genes are essentially recessives, and the suppressor of tu bw (su-tu) behaves as a semi-dominant in most matings.

The larvae resulting from these matings were reared on standard maizemeal-molasses medium seeded with live yeast, and incubated at 25°C. It is well known that penetrance and expressivity of the trait in melanotic tumor strains vary widely, even within highly inbred strains cultured under defined conditions, and depend on temperature, genetic background, and nutrition. In the summary table below, "-" indicates that fewer than 10% of the offspring possessed tumors, "+" that 11-40% were tumorous, and "++" that more than 41% of the flies were tumorous.

Table 1: Tumorous offspring from reciprocal matings between strains with second chromosome loci initiating melanotic tumor formation.

| | FEMALES | | | | | | |
|---|---|---|---|---|---|---|---|
| MALES | wild | tu bw | tu bw;st su-tu | tu 55g | tu $B_3$ | tu $A_2$ | $tu^K$ |
| wild | - | - | - | - | - | - | - |
| tu bw | - | ++ | ++ | ++ | + | - | - |
| tu bw;st su-tu | - | ++ | + | + | - | - | - |
| tu 55g | - | ++ | + | ++ | ++ | - | - |
| tu $B_3$ | - | ++ | + | ++ | ++ | - | - |
| $tu_K A_2$ | - | - | - | - | - | - | - |
| $tu^K$ | - | - | - | - | - | - | - |

Thus it is seen that the second chromosome genes tu bw, tu 55g, and tu $B_3$ behave as if they were allelic, whereas tu $A_2$ clearly is not, a fact demonstrated earlier by Barigozzi and di Pasquale (1956, Ist. Lomb., Rend. Sci., 90:484). It also appears that the suppressor of tu bw in heterozygous condition exerts as great or greater effect on tu 55g and tu $B_3$ as on tu bw. The penetrance of tu $A_2$ is normally quite low, and that of $tu^K$ is even lower when reared on standard media, although its penetrance is increased on media deficient in certain nutrients (Sang and Burnet, 1963, Genetics 48:235).

Since Glassman (1956, DIS 30:116) has already reported allelism between tu bw and tu $e^{11}$, it would seem that a sizable group of second chromosome melanotic tumor-producing loci are probably allelic; the designations of these loci should be standardized to reflect these allelic relationships.

Altenburg, E. and L. S. Browning. University of St. Thomas, Houston, Texas.

Comparative visible mutation rates in the X-chromosome of Drosophila at various stages in oogenesis.

Among approximately 40,580 female progeny of Muller's Vix stock (heterozygous at 13 visible loci in the X chromosome), a total of 77 mutations (45 whole body and 32 mosaic) were recovered at the visible loci under study, or about 1 in 530, after treatment of the female parents with CB 1506 (2-chloroethyl-methanesulfonate) vapor, and about a 20% sex-linked lethal rate from a smaller sample. Most of these mutations were recovered in the first five three-day brooding periods, but none among the relatively few progeny in the first brood, in which most of the oocytes treated in stage 14 would be represented. On the basis of work reported by R. Valencia, we calculate that the mutation rates at the same loci as the above (but in Muller's "jynd" stock) were about 10 times as high after X-ray treatment of stage 14 oocytes (the stage

studied by Valencia) than the rate we recovered after treatment of the earlier stages of oogenesis with an equivalent mutagenic dose of CB 1506. The "equivalence" is based on the lethal rates induced by the agents under study, and on the assumption that the lethal rates induced by radiation of earlier stages in oogenesis (not obtained in our experiments) would be about the same as the rates usually recovered after radiation of the mature sperm cells, though actually they are somewhat lower in the former than the latter case. The visible rates, relative to the lethal, were not widely different after X-ray treatment of stage 14 oocytes, as reported by Valencia, and CB 1506 treatment of the earlier stages, herein considered. The extreme radiosensitivity of stage 14 oocytes has been pointed out by Valencia.

Scharloo, W., M. S. Hoogmoed and A. E. ter Kuile. Universities of Groningen and Leiden, Netherlands. Disruptive and stabilizing selection an a cubitus interruptus mutant.

Scharloo (1964) reported that disruptive selection with random mating, practised on the 4th vein interruption of $ci^{D-G}$, caused a large increase of the genetic variance. Stabilizing selection caused a decrease of both the genetic variance and the environmental variance. In both lines the within fly variance did not change. In a new series of experiments from the same Pacific base population the following selection lines were made:

1. A line maintained under disruptive selection with random mating. The increase in genetic variance was even larger than in the first experiment. Extreme individuals began to overlap with wild type at generation 15. They showed extra venation, probably caused by a plexus allele. At the other side of the frequency distribution individuals with a very short (about 20% present) 4th vein appeared in generation 6. They lacked the 2nd cross vein. Even in the absence of $ci^{D-G}$ the cross-vein defect and an interruption of the 4th vein occurred. This phenotype is dependent on the presence of the right part of the 2nd chromosome distal to c. The within fly variance increased slightly.
2. Two lines under disruptive selection with negative assortative mating (forced mating of high and low extremes). In both lines the genetic variance, the common environmental variance and the within fly variance increased.
3. Two lines under stabilizing selection. In both lines the phenotypic variance decreased as a consequence of a decrease of the genetic variance, but in one line it stayed relatively high throughout the whole experiment (15 generations). In the other line environmental variance and within fly variance decreased as well as genetic variance.

Scharloo, W. and W. Vreezen. Universities of Groningen and Leiden, Netherlands. Correlated responses in 4th and 5th vein selection in Hairless mutants.

Scharloo and Vreezen (DIS 40:63) reported on selection for a large 4th vein interruption caused by Hairless mutants. In these lines selected after introduction of the mutant in the Kaduna and Pacific cage populations, measurements of the 5th vein were also made. The results show:

1. Pacific H selection 1. Only a minor response of the 5th vein.
2. Pacific H selection 2. A strong correlated response of the 5th vein so that after generation 8 practically no 5th vein material was present posterior to the 2nd crossvein, and individuals with a break proximal to the crossvein appeared.
3. Pacific $H^{57c}$ selection. In the first 5 generations both the 4th and 5th vein interruption increased, but after generation 8 the 5th vein increased in length to about its original value.
4. Kaduna $H^{57c}$ selection. 2 lines were selected concurrently for larger 4th vein interruption and larger 5th vein interruption respectively. The correlated responses of the veins not selected for, were very small compared with the direct responses.

Thus different base populations and even different selections from the same base population may show a different pattern of correlated response.

Brosseau, G. E., Jr. University of Iowa, Iowa City, Iowa. Some aspects of ring chromosome behavior in Drosophila.

The somatic behavior of ring chromosomes in corn is well known from the genetic and cytological studies of McClintock (1938). Comparable studies of ring chromosomes in Drosophila are primarily restricted to genetical analysis because of the relatively unfavorable cytological situation found in Drosophila. Rings in corn show mitotic instability which is manifested cytologically by the formation of double bridges, interlocked rings, loss of the ring and changes in ring size. The bridges and interlocks probably result from sister strand crossing over, a single crossover producing a double bridge, 2 crossovers yielding an interlock (sister union as suggested by Hinton, 1959, would produce double bridges but not interlocks). McClintock attributes loss primarily to lagging of the ring chromosome during anaphase. Large rings yield more bridges and changes in size; small rings are more prone to loss by lagging. Changes in size result from breakage of the bridges and fusion of the broken ends. Rings in Drosophila show a greater somatic stability than ring chromosomes in corn. However, both small and large rings in Drosophila show a small frequency of mitotic loss which is manifested by the occurrence of mosaic patches when suitable markers are used. Maternal ageing prior to the introduction of $X^{c2}$ increases the somatic instability of this ring (Brown and Hannah, 1952) and the unusual ring X, $X^{c2}w^{vc}$, shows a high frequency of spontaneous mitotic loss (Hinton, 1955). The few cytological studies which have been done on ring chromosome behavior have not completely elucidated the mechanism of ring loss in Drosophila nor do they permit direct comparisons of ring behavior in corn and Drosophila. The results reported here represent another attempt to study the mitotic behavior of ring chromosomes in Drosophila. While the results are inconclusive, they are presented here for the benefit of others who may be interested in this problem.

The rings studied were the ring X's, $X^{c2}$ and $X^{c2}w^{vc}$, del(1)$X^{c2}$ (an X-ray induced deletion of most of the euchromatin of $X^{c2}$, cytologically it is about 1/2 the size of the ring x) and the ring Y chromosome of Oster, MYR. The $w^{vc}$ stock had been selected for instability just prior to this study. The $X^{c2}$ series was divided into 2 groups, the mothers were aged as virgins for 12 days prior to introducing the ring in one while they were unaged in the other. Larval brain squash preparations were made by dissecting out the brains in hypotonic citrate and staining in lacto-orcein. The preparations were examined using phase contrast. Prophase and metaphase cells were examined for presence of the ring and for changes in size of the ring. Anaphase and telophase were scored for double bridges, interlocked rings, lagging of the rings and any other abnormalities. There was little difficulty in distinguishing between a double bridge and an interlock. No concommitant studies of somatic mosaicism were made. The data obtained are presented in Table 1.

In general, the frequency of abnormal cells in prophase and metaphase was very low. The 6 XO cells in the $w^{vc}$ series were all on a single slide and probably represented an XO sector arising from an early elimination of the ring. Anaphases and telophases were not scored on this slide. Four instances of changes in ring size were recorded, 2 large rings and 2 small rings. Determination of these changes in size was very subjective as a number of factors such as the compactness of coiling of the ring could give erroneous interpretations. Evidence for change in size of the ring must be considered ambiguous. The frequency of mitotic loss of these rings as measured by XO prophase and metaphase figures is apparently very low.

Examination of anaphase and telophase gave more definite indications of ring chromosome instability. $X^{c2}$/Y w dl-49 progeny from unaged mothers yielded more interlocks than double bridges indicating that 2 sister strand exchanges are more frequent than a single exchange, if indeed sister strand exchange is the cause of these configurations. Ageing of the mother increases the frequency of both interlocks and double bridges, indicating a correlation between these configurations and ring chromosome loss. This suggestion is not borne out by the results with $w^{vc}$ or MYR. Although $w^{vc}$ yields a high frequency of somatic loss, the frequency of bridges and interlocks was about the same as $X^{c2}$ except that bridges and interlocks were about equally frequent. These observations do not agree with those of Braver and Blount (1949) or of Hinton (1955). No explanation of this discrepancy is a available at this time. In the present case there does not seem to be a good correlation between bridges, interlocks and somatic loss. This conclusion is supported by the MYR results. MYR is generally a mitotically stable chromosome, no mosaics for $bw^{+}$ (the marker on MYR) being observed in eye tissue. Thus the bridge configurations seen with MYR are

unaccompanied by any detectable loss of the ring. These bridges also mean that sister strand crossing over, or whatever event causes them, occurs in heterochromatin as well as euchromatin.

The data reported here do not lend support to the conclusion that bridges and interlocks frequently lead to ring chromosome loss in larval brain tissue. Of course, the fate of bridges in brain tissue may be quite different from their fate in the rapidly dividing cleavage divisions where most of the losses actually occur. As in corn, the loss may be the result of lagging of the ring in anaphase. A few lagging rings were observed with all of the rings except MYR. These cases could be squashing artifacts although the author feels that the observed lagging chromosomes are bona fide instances of chromosome lagging.

In the last case, del(1)$X^{c2}$, no bridges or interlocks were found. There were 3 instances of lagging of the small ring; in one of these both rings were found at the same pole. The column headed other in table 1 includes instances of stickyness (5), single bridges of undefined origin, but not involving the ring (2), and 4 bridges of undefined nature in the MYR series.

These observations on ring chromosome behavior in Drosophila indicate that these rings act much like the rings in corn. The difference in the observed frequencies of mosaicism in these 2 species is probably due to different fates of the bridges and interlocks. In corn (as in other plants) the growth of the cell wall ruptures the bridge and fusion of the broken ends occurs. In Drosophila there is no cell wall and the bridges may often fail to break. Changes in the size of rings, if they in fact occur, would be evidence for breakage of at least some of the bridges. Hinton (1959) presents cytological evidence that some bridge breakage may occur in cleavage divisions. No clear evidence of bridge breakage was seen in the present study. The fate of cells with either broken or unbroken bridges is uncertain. This condition may cause the death of the cells or perhaps prevents their further division.

The mechanism of ring chromosome loss in Drosophila remains to some extent unclear. Hinton's (1959) finding that one or both ends of a bridge is occasionally not included in the late anaphase or telophase group lead him to conclude that anaphase bridges were the main cause of ring chromosome loss. In the present case, most of the bridges were observed to have both ends at the poles. The few exceptions may have been squashing artifacts; there is no way to be certain. Since loss of ring chromosomes by lagging and by bridge formation are not mutually exclusive events, they may both contribute to loss. It remains to be demonstrated whether one or the other of these events is the principal cause of ring chromosome loss in Drosophila. (Supported by research grant GM-06508 from NIGMS, USPHS)

References: Braver, G. and J. L. Blount, 1949, Rec. Genet. Soc. Amer. 18:78
Brown, S. W. and A. Hannah, 1952, P.N.A.S. 38:687-693
Hinton, C. W., 1955, Genetics 40:951-961; - 1959, Genetics 44:923-931
McClintock, B., 1938, Genetics 23:315-376

Table 1

The frequency of cytologically apparent abnormalities associated with ring chromosomes

| Genotype | Prophase + metaphase: Number of cells | Number XO | Large rings | Small rings | % normal cells |
|---|---|---|---|---|---|
| 1. $X^{c2}$/y w dl-49 ♀ (unaged) | 196 | 0 | 1 | 0 | 99.5 |
| 2. $X^{c2}$/y w dl-49 ♀ (aged) | 143 | 0 | 1 | 0 | 99.3 |
| 3. $X^{c2}w^{vc}$/y w lz dl-49 ♀ | 150 | 6 | 0 | 2 | 94.9 |
| 4. del(1)$X^{c2}$/y:= ♀ | 51 | 1 | 0 | 0 | 98.1 |
| 5. y v/MYR ♂ | 198 | 1 | 0 | 0 | 99.5 |

| Genotype | Number of cells | Anaphase + telophase: Double bridges | Inter-locks | Total bridges | % bridges | Lagging | Other |
|---|---|---|---|---|---|---|---|
| 1. $X^{c2}$ (unaged) | 150 | 1 | 8 | 9 | 6.0 | 1 | 1 |
| 2. $X^{c2}$ (aged) | 161 | 9 | 18 | 27 | 16.8 | 2 | 1 |
| 3. $X^{c2}{}_{w}{}^{vc}$ | 317 | 6 | 4 | 10 | 3.1 | 3 | 5 |
| 4. $del(1)x^{c2}$ | 118 | 0 | 0 | 0 | 0 | 3 | 0 |
| 5. MYR | 225 | 7 | 2 | 9 | 4.0 | 0 | 4 |

Nagle, James J. North Carolina State University, Raleigh. A study of intra- and interspecific polymorphism.

Chromosomal polymorphism has recently been found in populations of Race B of D. mojavensis which occur in Baja, California and Sonora, Mexico (DIS 38:58). This polymorphism involves Chromosome pairs 2 and 3 of the six constituting the karyotype of the species. In Chromosome 2 a simple paracentric inversion distinguishes the Standard (ST-2) from the LaPaz (LP) banding sequence, and a simple paracentric inversion in Chromosome 3 distinguishes the Standard (ST-3) from the Mulege (MU) arrangement. Analyses made from recent collections (courtesy of Dr. W. B. Heed) and from laboratory populations established from previous collections revealed ST-2 to be in low frequency (5-10%), while ST-3 and MU occurred in about equal numbers.

Two cage populations were initiated with equal proportions of males and females of D. mojavensis (Race B) and D. arizonensis, a closely related species. Cytological analyses indicate that mojavensis is replacing arizonensis, although limited hybridization (2-5% recombinant types per generation) is occurring. The intraspecific polymorphism of mojavensis has been maintained over the period of competition with arizonensis. The mean percentage of the second and third chromosome types within mojavensis, based on thirteen samples of each population (over 750 days), is given in Table 1.

Table 1. Mean percentages of the second and third chromosome types of mojavensis.

| Chromosome Number | Banding Sequence | Percentage: Population 1 | Population 2 |
|---|---|---|---|
| 2 | ST-2 | 4.7 | 9.6 |
| 2 | LP | 95.3 | 90.4 |
| 3 | ST-3 | 47.8 | 48.5 |
| 3 | MU | 52.2 | 51.5 |

A third population was initiated with male and female $F_1$ interspecific hybrids. In this case the chromosomes of mojavensis were obligatorily in heterozygous combinations with those of arizonensis at the beginning of the population. Table 2 gives the percentage of mojavensis second and third chromosomes observed in ten samples of the "hybrid" population. The proportions of ST-2 and LP have remained near the equilibrium frequencies observed in the mojavensis stocks, as given above. On the other hand, the percentages of ST-3 and MU deviate significantly from the 50-50 proportions observed in the laboratory populations of mojavensis (Table 1). Specifying no selection, the $F_1$ was expected to consist of equal amounts of ST-3/AR-3 combinations. The large deviation from this expectation, especially in the earlier samples, indicates that the combination ST-3/AR-3 has a much higher adaptive value than MU/AR AR-3. These data support the concept of fitness relativity, as put forth by Levene, Pavlovsky and Dobzhansky (1954, 1958). The intraspecific polymorphism, presumably based on heterosis of the ST-3/MU heterokaryotype, is greatly upset when subjected to a new genetic milieu; a novel chromosomal homologue, AR-3, is superimposed upon the intraspecific polymorphic system through hybridization. A new polymorphic condition is now being approached, seemingly due to ST-3/AR-3 heterokaryotypic superiority, with the apparent elimination of the MU arrangement despite any advantage it has in combination with the ST-3 chromosome.

Table 2. Percentages of mojavensis second and third chromosomes observed in the "hybrid" population.

| Sample Number | Percent Chromosome 2 | | Percent Chromosome 3 | |
|---|---|---|---|---|
| | ST | LP | ST | MU |
| 1 | 6.4 | 93.6 | 80.1 | 19.9 |
| 2 | 6.7 | 93.3 | 89.0 | 11.0 |
| 3 | 5.4 | 94.6 | 84.7 | 15.3 |
| 4 | 4.0 | 96.0 | 87.6 | 12.4 |
| 5 | 4.8 | 95.2 | 90.8 | 9.2 |
| 6 | 3.8 | 96.2 | 90.3 | 9.7 |
| 7 | 4.7 | 95.3 | 92.9 | 7.1 |
| 8 | 2.6 | 97.4 | 88.8 | 11.2 |
| 9 | 5.9 | 94.1 | 91.5 | 8.5 |
| 10 | 4.8 | 95.2 | 96.5 | 3.5 |

Heed, W. B. and R. W. Jensen. University of Arizona. Drosophila ecology of the Senita cactus, Lophocereus schottii.

D. pachea breeds in the rotting stem of senita cactus where it obtains the sterol, schottenol, for growth and reproduction. Other species of desert-adapted Drosophila do not breed in the stem of senita because it contains factors which are toxic to them (Heed and Kircher, Sci., Aug. 1965). The following bioassay is to determine the growth characteristics of Drosophila on the fruit of this unusual plant. Two different strains each (one strain repeated in mojavensis) of five species of Drosophila, which regularly inhabit the Sonoran Desert, were tested for reproductive ability by dividing one vial of mature egg-laying adults from each strain into two. One vial of standard banana media was supplemented with a cube of sterilized fruit and the other with a cuve of sterilized stem. In this way from 40 - 60 flies were tested per vial against their sibs. The number of progeny are recorded in the table.

| | pachea | | pseudoobscura | | arizonensis | | mojavensis | | nigrospiracula | |
|---|---|---|---|---|---|---|---|---|---|---|
| | A | B | A | B | A | B | A | B | A | B |
| Stem | 215 | 183 | 127 | 40 | 74 | 71 | 49 | 41 | 13 | 28 |
| Fruit | 110 | 58 | 161 | 96 | 257 | 255 | 179 | 294 | 37 | 94 |

D. pachea performs better on the stem than the fruit. The four other species perform better on the fruit, one reason being that the parents were killed in the stem vial. The time when 50% of the adults were killed was 7 days for pseudoobscura, 12 days for arizonensis and mojavensis and 25 days for nigrospiracula. Very few parents of any species, including pachea, were dead when they were removed from the fruit vials to avoid overlapping of generations. However, since the larval density was high in both fruit and stem vials in all species, there was also a high degree of larval and/or pupal deaths due to the stem. The low number of progeny from both tests in strain A of nigrospiracula reflects an unexpected unequal sex ratio in favor of the males. All progeny from all tests were fertile (produced larvae) on non-supplemented standard banana media in varying degrees. In the case of pachea the progeny from the fruit vials in one week layed 1/4 to 1/10 as many eggs as equal numbers of progeny from the stem vials, for instance: 73 vs 1,120 and 95 vs 425 eggs.

It is concluded from the performance of pachea that the fruit of senita contains either a very small amount of the sterol, schottenol, or a slightly different sterol. It is known from previous tests that pachea will not complete a full generation on banana food without supplementation either with the stem of senita or with schottenol or closely related sterols. One sterol ($\Delta^{5,7}$ stigmastadien 3-$\beta$-ol) is known to allow growth but not egg production. It may also be concluded that the fruit does not contain toxic factors to any appreciable amount. Both arizonensis and nigrospiracula have been reported associated with the fruit of senita in nature. From the present tests one may confidently predict that pachea does not breed in the fruits of senita in the Sonoran Desert.

Kaplan, W. D. and R. Seecof. City of Hope, Duarte, California. The mutagenic action of Aramite, an acaricide.

Aramite is frequently used in Drosophila laboratories for control of mites. Before embarking upon its use we decided to test it for possible mutagenic action. The results indicate that this acaricide is, indeed, mutagenic.

Fourteen males which developed as larvae on standard Drosophila food containing 0.6% Aramite were mated to 5 M-5 ♀♀ each over a period of three days. From these 14 males 620 F1 cultures were set up, of which 20 were sterile. Twelve lethals were recovered among the 600 chromosomes tested, giving a mutation rate of 2%. The control rate was 0.2%.

Aramite is made by the Naugatuck Chemical Co., Naugatuck, Conn. The active ingredient is 2-(p-tert-butylphenoxy) isopropyl-2-chloroethyl sulfite. (Supported by Public Health Service grant #AI 05038 to R. L. Seecof and #GM 10260 to W. D. Kaplan.)

Seecof, R. and W. D. Kaplan. City of Hope, Duarte, California. The failure of irradiated DNA to produce mutation in Drosophila melanogaster.

Om Parkash reported, Nature 205:312 (1965), that radiated DNA was mutagenic when fed to D. melanogaster. We repeated this experiment, following his reported procedure as closely as possible. We subjected herring sperm DNA (Calbiochem) to 100,000 r of X-rays at 644 r/min at 100 keV, 7 m amp, with filtration equivalent to 0.6 mm of Al. The irradiated DNA was added to a final concentration of 18 mg/ml into a food medium containing sucrose (5%), agar (1%), corn meal (6%), bran (1.5%), and propionic acid to pH 4.5.

In series 1, 1-3 day-old Oregon-R flies (5 pairs) were introduced to treated food in a half-pint bottle and allowed to lay eggs for 12 days at 20°C. Series 2 was the same except that 20 pairs were used and were changed to fresh treated food every 2 days. Adult Oregon-R males, offspring of flies fed upon treated food were mated, each to five M-5 females, for detection of sex-linked lethals.

Series 1 repeats the technique used by Parkash. Series 2 was designed to distinguish between mutations induced in the X-chromosome of the adult females feeding upon treated food for 12 days, and effects upon larval germ cells. Table 1 summarizes the data and shows that the rate of mutation was not elevated above the control rate which is at about 0.2% for our stock. A high sterility characterized the $F_1$ matings. This, however, is attributable to the males of the Muller-5 stock in use at that time, rather than to an effect induced by the irradiated DNA upon the Ore-R chromosomes. (Supported by Public Health Service grant #AI 05038 to R. L. Seecof and #GM 10260 to W. D. Kaplan.)

Table 1

| | Number $F_1$ matings | % sterile | No. chromosomes tested | Number lethals | % lethal |
|---|---|---|---|---|---|
| | | | Series 1 | | |
| | 1045 | 31.3 | 718 | 0 | -- |
| | | | Series 2 | | |
| Day 1-2 | 1050 | 27.7 | 757 | 0 | -- |
| 3-4 | 1049 | 8.2 | 959 | 2 | 0.20 |
| 5-6 | 1051 | 10.6 | 939 | 1 | 0.10 |
| 7-8 | 1095 | 6.9 | 1019 | 2 | 0.19 |
| 9-10 | 1055 | 8.0 | 970 | 1 | 0.10 |
| 11-12 | 1050 | 9.0 | 956 | 1 | 0.10 |

Röhrborn, G. Institut für Anthropologie und Humangenetik, Heidelberg, Germany.

Mutagenic N-Lost-Cyclophosphamides.

The mutagenicity of the following substances has been tested on the Berlin wild stock of D. melanogaster by means of the Basc method: 1. the phosphortriamide B 801; 2. the phosphordiamidomonoester B 518 (Cytoxan), and 3. the monoamidodiester B 525 (see table 1).

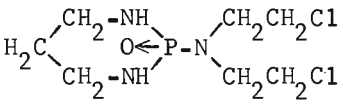
All drugs were applied by feeding on glass filter dishes as aquaeous solutions. The mutagenic activity declined in the following manner: B 801>B 518≧B 525. The differences in mutagenicity between B 801 and the other substances were especially evident in mature spermatozoa (brood I).

In all broods the mutagenic activity of both B 801 and B 518 was higher than that of B 518, although the differences between B 518 and B 525 were not statistically significant. The mutagenic events reported in this short communication were mainly restricted to post-meiotic stages of spermatogenesis.

In this group of compounds the mutagenic activity was correlated with the chemical reactivity.

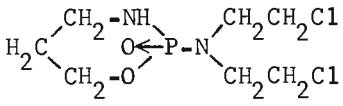
In contrast to the mutagenicity, the toxicity of the drugs in Drosophila declined in the sequence B 525>B 518>B 801. Furthermore, the three cyclophosphamides exerted sterilizing effects without significant correlations with the rates of recessive lethals or to their toxicity.

Table 1: Recessive sex-linked lethal mutations



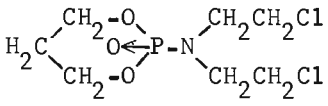
B 801

N,N-bis-(β-chloroethyl)-N',N''-propylenephosphoric-acidtriamide



B 518

N,N-bis-(β-chloreothyl)-N',O-propylenphosphoric-acidesterdiamide



B 525

N,N-bis-(β-chloroethyl)-O,O'-phosphoricacid-diestermonoamide

| brood | Chrom. n | lethals n | % | Chrom. n | lethals n | % | Chrom. n | lethals n | % |
|---|---|---|---|---|---|---|---|---|---|
| | | | | a) Concentration: $10^{-2}$ M | | | | | |
| I | 1249 | 14 | 1.1 | 1095 | 8 | 0.73 | 628 | 3 | 0.48 |
| II | 834 | 16 | 1.9 | 573 | 7 | 1.2 | 311 | 3 | 0.96 |
| III | 555 | 9 | 1.6 | 296 | 7 | 2.4 | 129 | 1 | 0.78 |
| I-III | 2638 | 39 | 1.5 | 1964 | 22 | 1.1 | 1068 | 7 | 0.66 |
| | | | | b) Concentration: $2 \times 10^{-2}$ M | | | | | |
| I | 1310 | 50 | 3.8 | 1037 | 9 | 0.87 | 224 | 1 | 0.45 |
| II | 511 | 18 | 3.5 | 424 | 15 | 3.5 | 82 | 1 | 1.2 |
| III | 75 | 4 | 5.3 | 82 | 4 | 4.9 | - | - | - |
| I-III | 1896 | 72 | 3.8 | 1543 | 28 | 1.8 | 306 | 2 | 0.65 |

duration of pairing: 3 days
Control: o.1±0.03%

Scholefield, J. and D. T. Suzuki. University of British Columbia, Vancouver, Canada. A test system for studies on the mechanism of reverse mutation.

Demerec (PNAS 48:1696, 1962) proposed that reverse mutations might arise by an unequal crossover following a mistake in pairing at the molecular level. Demerec (Genetics 48:1519, 1963) later found this hypothesis would not explain the "selfer" phenomenon in Salmonella but Magni (PNAS 50:975, 1963) proposed a similar mechanism in yeast. He found a high reversion rate of a homozygous allele associated with an exchange of outside markers that was greatly depressed when the allele was hemizygous in a deficiency heterozygote. Baylor et al. (Genetics 52:539, 1965) with phage T2 and Strigini (Genetics 52:759, 1965) with phage T4 have suggested a similar mechanism.

In order to determine whether reversion by unequal crossing over might occur in Drosophila melanogaster, studies were made of Notch mutants. Since both "point" and deficiency mutants are known at this locus, reversion rates in point mutant homozygotes and point/deficiency heterozygotes could be compared. The point mutant tested was $N^{40}$ and the deficiency was $N^{8}$. Welshons' (Genetics 47:743, 1962) selector system was used to kill almost all Notch offspring in the following crosses:

1 + $w^a$ $N^{40}$ rb/Y $w^a$ $N^{40}$ +;Cy,Dp,$bw^v$/+ ♀ X $w^a$ $fa^{no}$ spl/Y;Cy/Pm ♂

2 $w^a$ $N^{40}$ rb/+ $N^{8}$ +;Cy,Dp,$bw^v$/+ ♀ X $w^a$ $fa^{no}$ spl/Y;Cy/Pm ♂

In one series of experiments, females were radiated with 4000 rads of γ-rays.

15 females and 10 males were mated in quarter pint bottles and 30 bottles per tray. The flies were transferred through 2 or 3 six day broods. All bottles were checked daily for offspring from the 10th to the 20th day of the culture. Any possible revertants were testcrossed to $w^a$ $fa^{no}$ spl rb flies. The number of matings is summarized in the following table.

| Brood Number | Non-irradiated | | Irradiated | |
|---|---|---|---|---|
| | $N^{40}/N^{40}$ | $N^{40}/N^{8}$ | $N^{40}/N^{40}$ | $N^{40}/N^{8}$ |
| 1 | 6 trays | 11 trays | 12 1/2 trays | 7 trays |
| 2 | 10 trays | 11 trays | 12 1/2 trays | 7 trays |
| 3 | 2 trays | 5 trays | 1 tray | -- |
| Total | 18 trays | 27 trays | 26 trays | 14 trays |
| Gametes Sampled | 180,000 | 270,000 | 108,250 | 56,700 |

The number of gametes tested was estimated by crossing test females to Oregon-R males and counting the number of offspring produced per bottle, the estimate being based on the sum of half the number of females and all males. No revertants were found in an estimated 450,000 gametes in the non-irradiated and 165,000 gametes in the irradiated series.

While the selector system is relatively efficient, the task of setting up sufficient numbers of crosses to yield large numbers of test females and males proved too great. Since it is quite possible that Notch point mutants are of the "shift" type, the system described should be feasible where facilities and technical help are abundant. (This research was supported by NRC grant A-1764.)

Oshima, C. and T. K. Watanabe. National Institute of Genetics, Misima, Japan. Persistence of some recessive lethal genes in natural populations of D. melanogaster.

Many lethal chromosomes (the second chromosome) were isolated from different male flies collected simultaneously from natural populations located at Kofu and Katsunuma locality in Yamanashi Prefecture in October 1963 and 1964. A total of 16,086 crosses were performed diallelically between the lethal - Curly balanced strains.

The results of allelism tests were divided into three parts; two of them represented

crosses within lethal strains extracted at the same time and the third represented crosses between new and old lethal strains extracted in successive years. The results are given in Table 1.

Table 1. Results of allelism tests between lethal genes isolated from the Kofu and Katsunuma populations in 1963 and 1964.

| Year | 1963(Middle of Oct.) | | | | 1964(late in Oct.) | | | |
|---|---|---|---|---|---|---|---|---|
| Population | Kofu | Katsunuma | Between pops. | Whole loc. | Kofu | Katsunuma | Between pops. | Whole loc. |
| No. of lethal chr. | 61 | 53 | 114 | 114 | 31 | 43 | 74 | 74 |
| No. of crosses | 1830 | 1378 | 3233 | 6441 | 465 | 903 | 1333 | 2701 |
| No. of allelic c. | 60 | 30 | 80 | 170 | 18 | 33 | 55 | 106 |
| Allelic rate (%) | 3.28 | 2.18 | 2.47 | 2.64 | 3.87 | 3.65 | 4.13 | 3.92 |

| | | |
|---|---|---|
| No. of lethal chr. | 97 | 72 |
| No. of crosses | 6984 | |
| No. of allelic crosses | 185 | |
| Allelic rate (%) | 2.65 | |

The Kofu and Katsunuma populations are about thirteen kilometers apart from each other, and vineyards occupy the large part of the intervening area. These populations of D. melanogaster were very large. The allelic rate between lethals isolated from the different populations was relatively high as compared with those within the populations. The allelic rate between new and old lethals isolated in the successive years was also high. These results could be due to persistence of some common lethal genes distributed in both natural populations.

These common lethal genes were found frequently as shown in Table 2, and most of them were located with a paracentric inversion C or B on a chromosome.

Table 2. Frequent lethal genes isolated from the Kofu and Katsunuma populations and their linked inversion.

| Symbol of lethal gene | Frequency of appearance and their linked inversion 1963 | | 1964 | |
|---|---|---|---|---|
| 1201 | 13 | + or In(2R)C | 6 | + or In(2R)C |
| 1202 | 8 | + or In(2R)C | 6 | + or In(2R)C |
| 1203 | 6 | + | 3 | + or In(2R)C |
| 1204* | 6 | + | 7 | + or In(2R)C |
| 1207* | 4 | + or In(2L)B | 2 | + |
| 1208 | 3 | + | 1 | + |

\+ standard chromosome (no inversion)
* these lethal genes had been found in 1959.

The frequency of these persistent lethal genes was about 35 per cent of the total number of isolated lethal genes. Two lethals, symboled 1201 and 1202, were found to be located individually on different chromosomes in 1963 and also in 1964, but these lethal genes were found to be located together on the same chromosome in 1964.

The viability of heterozygotes for these persistent lethal chromosomes and the various kinds of chromosomes (normal, subvital, semi-lethal and lethal) was estimated by using Cy-Pm technique. For the latter, about ten chromosomes were taken randomly from ones of each class. The results are represented in Table 3.

Table 3. Relative viabilities of normal and persistent lethal heterozygotes.

| | Mating | | | | Pooled basis | |
|---|---|---|---|---|---|---|
| | N | SV | SL | L' | No. of counted flies | Relative viability |
| N | N/N | N/SV | N/SL | N/L' | 103,035 | 1.0537 ± 0.00929 |
| L | L/N | L/SV | L/SL | L/L' | 161,218 | 1.0585 ± 0.00748 |
| LL | LL/N | LL/SV | LL/SL | LL/L' | 52,625 | 1.0618 ± 0.01304 |

The viability of Cy/Pm fly = 1.0000
N:normal SV:subvital SL:semi-lethal L':lethal chromosomes

The viabilities of flies having a single or double lethal chromosome in heterozygous state were slightly higher than that of heterozygotes for a normal chromosome. Although their increases are not significant statistically, it can be said that these persistent lethal chromosomes would not manifest any deleterious effect in combining with various kinds of chromosome. As long as a lethal gene is associated with an epistatic gene complex, including a heterotic inversion, it would be exempted from natural selection.

Ménsua, J. L. University of Barcelona, Spain. Y chromosome effect on interocellar bristles in D. melanogaster.

Wolsky (1958) has pointed out the possibility that a polygenic system is involved in the genetic control of interocellar bristles in D. melanogaster, as generally happens with many of the quantitative characters, and that some genes are situated on the Y chromosome. This last point was deduced by making reciprocal crosses between two D. melanogaster strains, with different averages of interocellar bristles, and finding on $F_1$ an intermediate average among the daughters, whereas the sons had an average more similar to their sires in each cross.

In one experiment carried out with a D. melanogaster wild strain from Prat de Llobregat (Barcelona), kept at 17°C in a population-box for 8 months, a clear-cut effect of the Y chromosome on these bristles was found. The experimental procedure was as follows: 80 pairs of flies were crossed at random, 35 eggs of each pair were put in each vial in order to avoid over-crowding. The temperature was set at 17° ± 0.5°C during the experiment. From $F_1$ 4 males and 4 females of each family were counted, and the offspring-parent regression and the partials regressions daughter-dam, daughter-sire, son-dam and son-sire were calculated, revealing the following results:

| | | | |
|---|---|---|---|
| Offspring-Parent | b = 0.389 ± 0.079 | t = 4.924 | P < 0.001 |
| Son-Dam | b = 0.083 ± 0.091 | t = 0.912 | P ≃ 0.3 |
| Son-Sire | b = 0.307 ± 0.063 | t = 4.873 | P < 0.001 |
| Daughter-Dam | b = 0.179 ± 0.092 | t = 1.945 | P ≃ 0.05 * |
| Daughter-Sire | b = 0.216 ± 0.070 | t = 3.085 | P ≃ 0.005 |

* In spite of the fact that the significance level for 0.05 is t = 1.99, we can consider this regression significant.

We can see, on one hand, that the son-dam regression coefficient is not significant, but on the other, the son-sire regression is very significant. Therefore we can conclude that the Y chromosome, which is the only factor received by the sons from the sire, and not from the dam, is the responsible one of these differences between both regressions and that in the Y chromosome where is polygenic activity. But also there is polygenic activity on interocellar bristles on the other chromosomes because the regressions between daughter-dam and daughter-sire are also significant.

In order to see if there was a Y chromosome effect on abdominal and sternopleural bristles, the partial regressions in these bristles were calculated, but no effect at all was found.

References: Wolsky, A., 1958. The formation of interocellar bristles in D.m. Proc. Calcutta Zool. Soc., 11:1-7

(This work was supported by a grant from National Education Ministry of Spain (P.I.O.). I am in debt for advice and suggestions to Dr. A. Prevosti.)

Halfer, C., M. Piccinelli and T. L. Torri.
University of Milan, Italy. Melanotic formations and their development in the stock Freckled of Drosophila melanogaster.

Some histological researches have been carried out on the new tumorous stock Freckled (Frd, 102, 2nd chromosome) of Drosophila melanogaster, characterized by peculiar formations.

Two different tumoral manifestations have been found in this stock, each with its own mechanism of formation: i) an early tumoral manifestation (larval stage with a low and rather inconstant incidence (up to 16.0%), involving the haemolymph cells, and there, similar to the usual tumoral stocks); ii) a late manifestation typical of Freckled, with 100.0% penetrance, characterized by melanotic masses all over the whole body (fig. 1), present only in the pupal stage and in the adult, which involved single fat and pericardial cells.

The action of Freckled depends on temperature (25°) and on certain unidentified substances in living yeast and leads to the synthesis of polyindole in the fat cells (R. Nicolaus, personal communication).

In the fat cells melanization starts about 30 hours from the onset of pupation; at the beginning it is confined to the area of cell nucleus, later spreads to the cytoplasm reaching its height between 93 and 120 hours. Melanization appears first in the thorax, then in the head and in the abdomen.

The pericardial cells undergo also melanization (Fig. 2), especially in the old flies, but do not seem to produce melanin themselves, but to absorb melanotic products from the fat cells through a process of phagocytosis (Bairati Jr., personal communication).

The Freckled phenomenon resembles that described by Jones and Lewis (Biol. Bull., 112:220, 1957) as red cells (r c, 26.0±, 2nd chromosome).

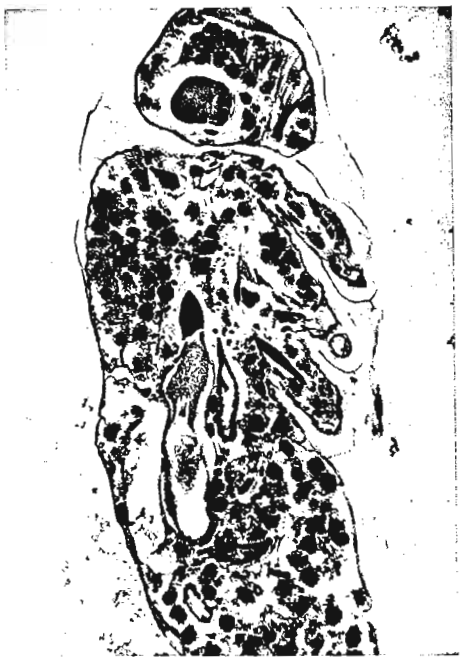


Fig. 1: Frd : pupa, sagittal section. Unstained (x 28)

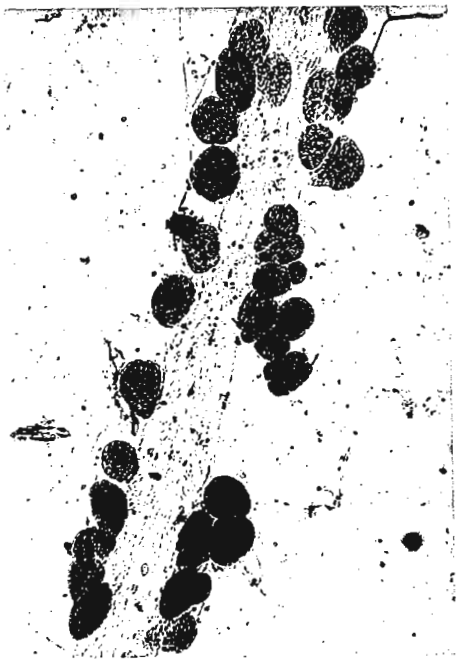


Fig. 2: Frd : pericardial cells of adult fly. Unstained (x 80)

Watson, W. A. F. State University, Leiden, Netherlands. Repair of premutational damage in spermatocytes as sampled from Drosophila pupae.

Earlier work (Sobels 1965, Mut. Res. 2: 168-191) showed that post-treatment with $O_2$, as compared to post-treatment with $N_2$ favors repair of genetic damage induced by irradiation under anoxia in spermatids and spermatocytes sampled from adult flies. Attempts to show that this repair occurred when pupae were irradiated, were unsuccessful when the same experimental procedure was followed. The results reported here show that when 24 hour pupae are pre-treated for 6 hours with $N_2$, irradiated in $N_2$ with 2500R X-rays, and post-treated with either $N_2$ or $O_2$ for two hours, then in the first one-day brood there is a consistent and significant decrease in mutation frequency (as measured by recessive lethals in a ring-X chromosome) after post-treatment with $O_2$. The results are given in Table 1. They show that the similar results obtained from earlier broods of adult flies did not originate from artefacts in the sampling technique, and support Sobels' conclusion that there is a repair system operating at this stage of development.

Table 1: Frequencies of recessive sex-linked lethals induced by 2500 R in one one-day brood from 24 hour male pupae of the genetic constitution $X^{C2}y$ B/sc$^8$,Y after post-treatment with N2 or O2.

| Expt. No. | Post-treatment | No. chromosomes tested | % lethals |
|---|---|---|---|
| 1 | $N_2$ | 487 | 6.37 |
| | $O_2$ | 466 | 3.64 |
| 2 | $N_2$ | 304 | 6.25 |
| | $O_2$ | 371 | 4.31 |
| 3 | $N_2$ | 924 | 7.25 |
| | $O_2$ | 836 | 5.26 |
| 4 | $N_2$ | 614 | 6.35 |
| | $O_2$ | 562 | 4.80 |

Total chromosomes tested = 4564
$P < 0.006$ (two-sided test) using combination of 2 x 2 contingency tables

(Research carried out within the frame of the Association between Euratom and the University of Leiden, contract Nr. 052-64-1 BIAN, and supported by the Institute for Radiopathology and Radiation Protection, and the Health Research Organization T.N.O.)

Mayeda, K. Wayne State University, Detroit, Michigan. Study of penetrance of the tu-h phenotype.

In the course of studying the penetrance of the tu-h phenotype in the tu-h stock maintained at this laboratory, the effect of parental age was investigated. Single pair matings of tu-h female by tu-h male were made and left in the vials for twenty-four hours. The female was then separated from the male and transferred to new vials every twenty-four hours for 14 consecutive days. The male was given a new virgin female every twenty-four hours for 14 consecutive days, the females being transferred to new vials every twenty-four hours as before. The penetrance of the trait was measured in the offspring and is presented in Table 1.

The results of these experiments indicate that there is a correlation between penetrance of the trait and the age of the female. Average penetrance in the offspring of twenty-four hours old females is 67% when all ages of males are combined. As it can be seen from the table, the penetrance gradually increases as the female becomes older. However, there seems to be no correlation between paternal age and penetrance. Further investigations are being conducted to determine if the increase in penetrance in the offspring of older females is due to lack of competition for food in the larval stages.

Table 1. Percent penetrance of the tu-h phenotype for various ages of parents.

| | Age in days | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 |
| Female Parents (1) | 67 | 62 | 67 | 65 | 70 | 73 | 76 | 75 | 76 | 76 | 81 | 84 | 79 | 77 | 94 |
| Male Parents (2) | 71 | 76 | 78 | 79 | 72 | 68 | 60 | 77 | 62 | 74 | 65 | 65 | 67 | 66 | 76 |

(1) For all ages of male (2) For all ages of female

Ling, Lee-Nien L., M. Horikawa, and A. S. Fox. University of Wisconsin, Madison. Aggregation of dissociated Drosophila embryonic cells.

A method for the in vitro culture of Drosophila embryonic cells was recently established by Horikawa and Fox (1965). By using this culture method together with the rotary shaker method for the formation of aggregates devised by Moscona (1961) it is now possible to establish the optimal conditions for the formation of aggregates by Drosophila embryonic cells and to characterize some of their properties.

Embryonic cells were obtained from eggs of a wild stock of Drosophila melanogaster (Oregon R-EL2). Eggs were collected after 6 hours of oviposition. The optimal conditions for the formation of aggregates were as follows: dissociation of the eggs by gentle homogenization, suspension of the embryonic cells together with the yolk material in H-5 culture medium supplemented with 10% newborn calf serum, and rotation of the primary cell suspension at 60 rpm at 30°C for 24 hours.

Two main types of aggregates were observed at the bottom of the culture dishes after 24 hours of shaking; (a) large, more or less spherical aggregates containing both the large and small cell types described by Horikawa and Fox and (b) small, irregularly shaped aggregates which seemed to consist primarily of the small cell type.

Factors that are detrimental to cells such as prolonged or high speed centrifugation and irradiation with ultraviolet resulted in a corresponding decrease in the ability of the treated cells to form aggregates. Cells grown in stationary cultures prior to shaking showed a decreasing ability to form aggregates as the length of stationary culture time increased.

Histological preparations were made of aggregates after 24 hours of shaking followed by 0, 6, 7, and 19 days of stationary culture. Signs of internal organization and cellular differentiation were observed within the aggregates, becoming more evident as the age of the aggregates increased. Although a number of new cell types could be recognized, arranged in characteristic manner, they could not be identified with certainty. (Supported by USPHS Grant No. GM 11777.)

Ménsua, J. L. University of Barcelona, Spain. Antagonistic temperature effect on average number of bristles in D. melanogaster.

Until the present, as far as we know, all workers in Drosophila have considered as true the fact that a temperature elevation in culture corresponds to a falling off in the average number of bristles. Previous tests were carried out on macrochaetae and sternopleural microchaetae (ex. gr. Plunkett, 1927; Beardmore, 1956 - quoted in Thoday, 1958).

The results reported here are an advance of a work, which is being carried out at present on temperature effect on bristles number in D. melanogaster. They prove that different bristle sets behave in different ways in respect to temperature. Three bristle sets were choosen - two of them very common in population research - lying on the three parts of the body: interocellar bristles on the head, both sides of sternopleural bristles on the thorax and abdominal bristles of 4th and 5th segment on the abdomen. An Oregon-R strain and a wild strain from Prat de Llobregat (Barcelona) were used, both kept at 17°C in population

boxes. Eggs from these boxes were re-collected and 90 eggs were put in each bottle in order to avoid over-crowding. Two sets of bottles (5 each one) from each strain were cultivated at 17° ± 0.5°C and 25° ± 0.5°C respectively. A replica at a different time with Prat strain was made (Prat B). 22 males and 22 females of each bottle were counted. The results were as follows:

| | | 17°C | 25°C | d(17°-25°) | P ≤ |
|---|---|---|---|---|---|
| ABDOMINAL BRISTLES | | | | | |
| Prat A | ♀♀ | 45.04 ± 0.25 | 45.57 ± 0.34 | -0.53 ± o.42 | 0.2 |
| | ♂♂ | 35.90 ± 0.21 | 37.39 ± 0.34 | -1.49 ± o.40 | 0.001 |
| Prat B | ♀♀ | 44.46 ± 0.33 | 46.04 ± 0.35 | -1.58 ± 0.48 | 0.001 |
| | ♂♂ | 36.05 ± 0.29 | 36.65 ± 0.34 | -0.60 ± 0.45 | 0.2 |
| Oregon-R | ♀♀ | 46.17 ± 0.29 | 47.91 ± 0.35 | -1.74 ± 0.45 | 0.001 |
| | ♂♂ | 37.08 ± 0.29 | 39.08 ± 0.26 | -2.00 ± 0.39 | 0.001 |
| STERNOPLEURAL BRISTLES | | | | | |
| Prat A | ♀♀ | 20.53 ± 0.12 | 18.36 ± 0.22 | +2.17 ± 0.25 | 0.001 |
| | ♂♂ | 19.48 ± 0.13 | 17.92 ± 0.24 | +1,56 ± 0.27 | 0.001 |
| Prat B | ♀♀ | 20.08 ± 0.17 | 18.69 ± 0.20 | +1.39 ± 0.26 | 0.001 |
| | ♂♂ | 19.41 ± 0.16 | 17.74 ± 0.20 | +1.67 ± 0.26 | 0.001 |
| Oregon-R | ♀♀ | 21.06 ± 0.14 | 19.73 ± 0.17 | +1.33 ± 0.22 | 0.001 |
| | ♂♂ | 20.39 ± 0.13 | 18.72 ± 0.15 | +1.67 ± 0.20 | 0.001 |
| INTEROCELLAR BRISTLES | | | | | |
| Prat A | ♀♀ | 7.20 ± 0.06 | 7.92 ± 0.10 | -0.72 ± 0.10 | 0.001 |
| | ♂♂ | 6.95 ± 0.07 | 7.37 ± 0.12 | -0.42 ± 0.14 | 0.005 |
| Prat B | ♀♀ | 7.22 ± 0.10 | 7.82 ± 0.10 | -0.60 ± 0.14 | 0.001 |
| | ♂♂ | 7.10 ± 0.10 | 7.39 ± 0.10 | -0.29 ± 0.14 | 0.05 |
| Oregon-R | ♀♀ | 7.23 ± 0.09 | 7.69 ± 0.08 | -0.46 ± 0.12 | 0.001 |
| | ♂♂ | 7.22 ± 0.09 | 7.41 ± 0.08 | -0.19 ± 0.12 | 0.1 |

Note: In Prat A (at 17°C) 220 flies of each sex were counted, instead of 110.

As we can see, on one hand, the sternopleural bristles behave as was known, but on the other hand abdominal and interocellar bristles increase their averages when temperature increases. The differences between bristles averages in both temperatures (d) are significant, except in three cases, but in all cases the differences are negative.

At present the work is being followed up to see what happens when flies are cultivated at 12° and at 29°C, and with temperature shocks, and the possibility that those antagonistic differences in bristles averages would be correlated with these three points: 1) Speed differences in growth at both temperatures, 2) Differences in time formation of thorax and abdomen hypoderm - at 25°C the thorax hypoderm is completed 27 hours before abdomen hypoderm (Bodenstein, 1950) - and 3) The possibility that some morphogenetical substance for bristles was diffused in a different manner during bristle formation because of the temperature and speed in growth differences.

References: Bodenstein, D. 1950. The postembryonic development of Drosophila. Biology of Drosophila, Chap. IV:275-367. New York, John Wiles & Sons, Inc.

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(This work was supported by a grant from National Education Ministry of Spain (P.I.O.). I am in debt for advice and suggestions to Dr. A. Prevosti.)

Dolfini, S., A. Gottardi and G. Rezzonico Raimondi. University of Milan, Italy.

First results on changes of chromosome number in cells of D. malenogaster cultured in vitro.

In order to explain the changes of chromosome number that occur in vitro, embryonic cells of the Varese wild strain of D. melanogaster, which could be a favorable material for these investigations, have been cultured, according to the technique described by Horikawa and Fox (1964) (fig. 1).

Chromosome countings on squashed cells after 12, 18, 21, 24, 48, 72, 96, 120 and 168 hours of culture, have shown that at the first analysis (12 hours) some heteroploid cells are already present. The frequency of abnormal mitoses increases during the subsequent hours of culture (Table 1).

Table 1. Percentage of metaphases showing various chromosome numbers after in vitro culture of Drosophila melanogaster embryonic cells.

| Hours of Culture | Number of chromosomes | | | | | | | | | | | | | No. metaphases analyzed |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | |
| 12 | - | - | - | 3. | 88.1 | 5.2 | 3.0 | 0.7 | - | - | - | - | - | 135 |
| 18 | 1.6 | - | 1.6 | 6.3 | 71.9 | 7.8 | 4.7 | - | - | 1.6 | - | 4.7 | - | 64 |
| 21 | - | - | 1.7 | 10.3 | 79.3 | 1.7 | 3.4 | - | - | 1.7 | - | - | 1.7 | 58 |
| 24 | - | 1.4 | 2.7 | 8.1 | 70.3 | 10.8 | 4.1 | 1.4 | 1.4 | - | - | - | - | 74 |
| 48 | - | 1.6 | 1.6 | 8.2 | 70.5 | 6.6 | 3.3 | 6.6 | 1.6 | - | - | - | - | 61 |
| 72 | - | 12.3 | 4.6 | 29.2 | 24.6 | 15.4 | 7.7 | - | - | 3.1 | 3.1 | - | - | 65 |
| 96 | - | 5.0 | 11.7 | 26.7 | 20.0 | 15.0 | 6.7 | 5.0 | 6.7 | - | - | 1.7 | 1.7 | 60 |
| 120 | - | 10.0 | 3.3 | 38.3 | 20.0 | 20.0 | - | - | 1.7 | 3.3 | 3.3 | - | - | 60 |
| 168 | 2.0 | 4.0 | 16.0 | 24.0 | 22.0 | 20.0 | 4.0 | 2.0 | 4.0 | - | 2.0 | - | - | 50 |

Two periods of striking increase in heteroploidy have been noted, the first between 12 and 18 hours and the second between 48 and 72 hours. Tetraploid cells were virtually absent during the first hours of culture.

The first and fourth pairs of chromosomes are most frequently involved (86.4%) in these chromosomal variations (fig. 2). The data, we have found, seem to indicate that heteroploidy results from non-disjunctions and/or other mitotic errors and not from a primary polyploidization followed by chromosomal losses.

Fig. 1

Fig. 2

Fig. 1: Normal metaphase after 24 hours of culture.
Fig. 2: Abnormal metaphase showing five elements of the first pair (48 hours of culture).

Goldin, Herbert. University of Oregon, Eugene. Oxygen uptake in two Minutes of D. melanogaster.

Since all heterozygous Minute larvae, regardless of their locus, exhibit delayed growth and development, as compared to normal sibs, and since investigations by Farnsworth (J. Expt. Zool. 157:345-352, 1964) have indicated abnormalities in cellular respiration and protein synthesis of $M(2)1^2$, it was felt that respiratory studies of $M(2)1^2$ and M(3)w heterozygous as well as homozygous larvae might prove fruitful in further elucidating the Minute effect.

Larvae were collected according to the method of Farnsworth (DIS 37:139, 1963). Controls (+/ltd and +/ca) were tested for $O_2$ consumption at 2, 24, 48, 72 and 90 hours of development. Since Minute homozygotes do not grow appreciably after hatching, a control of approximately the same size was considered more appropriate than one of similar age. Therefore, +/+ larvae, two hours post hatching were used as controls for the Minute homozygotes.

Heterozygote Minutes ($M(2)1^2$/ltd and M(3)w/ca) were obtained by mating ltd/ltd (ca/ca) virgin females with $M(2)1^2$/ltd (M(3)w/ca) males. The resultant larvae could be readily distinguished at 48 hours of development, on the basis of Malphigian tubule color, the Minute heterozygotes having the characteristic yellow pigmentation and ltd/ltd or ca/ca an absence of pigmentation. The heterozygotes were tested for $O_2$ consumption at 48, 72, and 96 hours of development.

Homozygous Minutes live for about 50-60 hours and can be readily distinguished from their wild type sibs in culture at 24 hours of development on a size basis. These were tested for respiration at 24 and 48 hours of development.

The larvae were removed from a yeastless culture medium directly to a micro-respirometer. The apparatus was a modification of that described by Thimann, Yocum and Hackett (BBA 53:239-257, 1954) and consisted of a 5 ml vial with a moistened piece of filter paper on the bottom. In the mouth of the vial was inserted a one-hole rubber stopper, through which was placed a 0.2 ml pipette, calibrated to 0.001 ml (one microliter). A small piece of cotton wick saturated with 10 N KOH served to absorb $CO_2$ and was suspended in the chamber on an insect pin inserted in the rubber stopper. The entire unit was immersed in a water bath, the temperature of which was effectively regulated at 25°C by means of a heating unit, a stirring attachment, and a copper coil through which cold water could be run for cooling. As noted above, the KOH filter served as a trap for $CO_2$ given off by the larvae during respiration, and thus when the oxygen in the vial was used, water was drawn into the pipette. Oxygen uptake readings, in microliters, could thus be taken directly by noting the initial and final levels of water in the pipette.

After the larvae had been transfered to the vial, the whole unit was assembled and placed in a water bath for a 15 minute equilibration period, after which readings were taken at 10 minute intervals for 30 minutes. A blank respirometer was always inserted in the bath to serve as a thermobarometer to compensate for any changes in barometric pressure.

The results were recorded as the mean value of oxygen uptake in microliters of oxygen per larva per hour, as shown in Table I, and represent a minimum of five experiments from two different cultures on different experimental days for each genotype and age.

The data show that the control respiratory rate steadily increases to 48 hours post hatching. At 72 hours of development, the rate has quadrupled that of the 48 hour stage. At 90 hours, just prior to pupation, there is only a slight increase in respiration compared to 72 hour larvae.

In the case of the Minute heterozygotes, there is no significant difference in $O_2$ consumption, when compared to controls, at 48 hours after hatching. At 72 hours, however, the $M(2)1^2$/ltd respiratory rate is only half that of its +/ltd control and $O_2$ consumption is even further depressed in M(3)w/ca, when compared to its +/ca control. At 96 hours of development, the Minute heterozygotes show a slight increase in respiration compared to 72 hour larvae but, still do not approach the value obtained for 90 hour controls.

In the Minute homozygotes, $O_2$ consumption of the 24 hour larva was slightly lower than that of the two hour wild type control. $Minute(2)1^2/M(2)1^2$ larvae had somewhat increased their respiratory rate at 48 hours but, M(3)w homozygotes, although still apparently living, had a respiratory rate which was undetectable by the apparatus.

The data thus presented, have indicated another facet of the $M(2)1^2$ and $M(2)1^2$ and M(3)w phenotype, i.e., a lowered respiratory rate, which is time specific during larval development, occuring some time between 48 and 72 hours after hatching.

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Table 1: Effect of genotype on $O_2$ uptake in ul $O_2$/larva/hour ± SD

| genotype | age (hrs., post hatching) | | | | | |
|---|---|---|---|---|---|---|
| | 2 | 24 | 48 | 72 | 90 | 96 |
| +/+ | .265±.05 | | | | | |
| +/ltd$_2$ | .29 ±.15 | .926±.37 | 1.82± .74 | 7.96±1.4 | 8.75±3.03 | |
| M(2)l$^2$/ltd | | | 1.77±1.07 | 3.99±1.8 | | 5.83±1.79 |
| M(2)l$^2$/M(2)l$^2$ | | .195±.048 | .25± .09 | | | |
| +/ca | | .603±.37 | 2.16± .88 | 8.32±1.91 | 9.71±1.71 | |
| M(3)w/ca | | | 2.16± .5 | 3.24±1.1 | | 4.3 ±1.35 |
| M(3)w/M(3)w | | .132±.12 | ~0 | | | |

Sharma, R. P. Indian Agricultural Research Institute, New Delhi, India. Radio-sensitization of Drosophila melanogaster by N-Ethylmalemide.

It has been demonstrated by Bridges (1960) in E. coli and Sharma (1965) in Vicia faba, that N-Ethylmalemide possesses radiosensitizing ability. A preliminary report on the radiosensitizing effect of this chemical in Drosophila is presented here.

2.5 ml of 100 uM solution (pH7) of N-Ethylmalemide was mixed with 2.5 ml of basic medium, comprised of agar (3%), yeast (10%), glucose (10%), propionic acid (0.4%) and water (100 ml), to get 50 uM concentration of the chemical. Freshly laid Drosophila eggs (Oregon-K) were transferred to this medium and allowed to develop up to adult stage. The newly emerged males were collected and kept for two days. One batch was kept as such, whereas the other batch was irradiated with 2400 r of X-rays. The males collected from the normal medium were irradiated with the same radiation dose to serve as control for the chemical-radiation combination treatment. The males were crossed with M-5 virgin females at the rate of one male and three females. The sex-linked recessive lethals were scored in $F_2$.

From the data (Table 1) it is seen that the combination treatment of chemical and radiation shows about 2-fold increase (5.4%) in the frequency of sex-linked recessive lethals over radiation (2.8%). The chemical alone is not able to produce any mutation. The possible explanation for such radiosensitizing effect produced by X-Ethylmalemide may be due to its ability to combine and inhibit the sulphydryl groups.

Table 1

| Treatments | Chemical dose | Radiation dose | No. of Chromosomes tested | % of sex-linked recessive lethal |
|---|---|---|---|---|
| N-Ethylmalemide | 50 uM | - | 695 | - |
| X-rays | - | 2400 r | 634 | 2.8 |
| N-Ethylmalemide + X-rays | 50 uM | 2400 r | 646 | 5.4 |

References: Bridges, C. B. (1960). Sensitization of E. coli to radiation by N-Ethylmalemide. Nature 188:415.
Sharma, R. P. (1965). The radiosensitizing effect of N-Ethylmalemide on Vicia faba. Curr. Sci. (In press).

Ehrman, Lee* and Diether Sperlich.** The Rockefeller Institute. XXY Drosophila paulistorum ♀♀.

At least three different kinds of hybrid sterility occur within the superspecies Drosophila paulistorum. This superspecies consists of six races or incipient species; hybrids between the races are fertile as ♀♀ but sterile as ♂♂ (Dobzhansky and Spassky, 1959). The hybrid ♀♀ can be backcrossed to ♂♂ of the parental races, and the backcross progenies consist again of fertile daughters and sterile sons. The sterility of the backcross ♂♂ depends upon the genetic constitution of their mothers; all the sons of a ♀ carrying any mixture of the chromosomes of the parental races are sterile, even if some of these sons themselves carry only the chromosomes of a single race (Ehrman, 1960). This is, then, an instance of genic sterility operating through a maternal effect, the genes responsible being distributed in all three pairs of the chromosomes which the species possesses. Evidently, the sterility of the $F_1$ ♂♂ is due to a different mechanism, since $F_1$ hybrids are descendants of pure rather than hybrid mothers.

A third kind of sterility has been reported (Ehrman, 1963), so far in only a single cross, between strains from Mesitas and those from Santa Marta, Colombia. Both the Mesitas and Santa Marta strains belong to the Transitional race of D. paulistorum. The cross Mesitas ♀ x Santa Marta ♂ gives fertile hybrids of both sexes, but the male progeny of the reciprocal cross is sterile. The hybrid ♀♀ can be backcrossed to males of either parental strain; the ♂ progenies of these backcrosses are sterile if they carry the Y chromosome of Mesitas in the Santa Marta cytoplasm, or the Y chromosome of the Santa Marta strains with the cytoplasm of Mesitas.

With this new evidence in mind, an attempt was made to acquire Drosophila paulistorum ♀♀ carrying a Y chromosome (metacentric and indistinguishable from the X chromosome at metaphase, in this species); experiments were planned in which the fertility or sterility of interracial hybrid males would be ascertained when they had received their Y chromosome from their mothers. ♂♂ of the Lancetilla, Honduras strain of the Centro-American race were given a dosage of 3000r at one time and three separate stocks were easily established in which the ♂♂ carried a Y-II chromosome (carrying a dominant marker gene) translocation. Occasional nondisjunction at meiosis would then produce the desired individual ♀♀ carrying a Y chromosome. However, we could never get them to breed despite constant care. One such ♀ laid eggs, none of which hatched.

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Anderson, Wyatt W. Rockefeller Institute. Frequency of spontaneous wing vein abnormalities in experimental populations of Drosophila pseudoobscura.

Many of the studies of polygenic variability, classical as well as recent, have utilized abnormalities of wing venation in Drosophila. They have shown that alleles which modify the normal wing venation are widespread, although individually rare. By recombination and segregation alone, combinations of alleles necessary for various wing vein abnormalities are expected to occur in low frequencies.

In a study of body size in six experimental cage populations of Drosophila pseudoobscura, 3,326 wings were examined. The six cages were all begun from offspring of crosses among forty strains isolated at Mather, California; Bryce National Park, Utah; Ferron, Utah; and Gunnison, Colorado. The cages were maintained for six years before these measurements. One wing was examined from each fly. As shown in Table 1, the low frequencies of aberrant venation patterns which are expected, do occur.

Table 1

| Type of Wing Vein Abnormality | Number Observed |
|---|---|
| posterior cross vein absent or reduced | 4 |
| anterior cross vein forked | 2 |
| posterior cross vein forked | 3 |
| Total aberrancies | 9 |
| Total flies examined | 3,326 |
| Frequency spontaneous wing vein abnormalities | 0.28% |

Kang, Y. S. and C. C. Lee. Seoul National University, Korea. The frequency of reciprocal translocation in D. melanogaster irradiated with 500r of X-rays.

The determination of the frequency of reciprocal translocation in D. melanogaster, Seoul strain males irradiated with 500r of X-rays was carried out in the present study. The reciprocal translocation was observed among Y, 2nd and 3rd chromosomes.

The frequency was determined at 3 intervals and during spermatogenesis after irradiation.

In 1963, Ives observed different frequencies in several Oregon-R heterozygotes which were irradiated with the same dosages of X-rays. The other works of various authors were represented in the total frequency of the translocation.

Drosophila melanogaster (Seoul strain) males and vg:se mutant females were used for the materials in the present experiment.

The results are summarized in tables 1, 2 and 3.

Table 1. The frequency of reciprocal translocations in control group

| Time eggs were laid after mating (days) | Time mating was made after hatching (days) 1 - 2 No.of tests | No. of trans.% | 3 - 4 No.of tests | No. of trans.% | 5 - 6 No.of tests | No. of trans.% | 7 - 8 No.of tests | No. of trans.% | 9 - 10 No.of tests | No. of trans.% |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 - 2 | 280 | A 0 B 0 C 0 Tot. 0 0.000 | 270 | A 0 B 0 C 0 Tot. 0 0.000 | 310 | A 0 B 0 C 1 Tot. 1 0.322 | 255 | A 1 B 0 C 0 Tot. 1 0.392 | 365 | A 0 B 0 C 0 Tot. 0 0.000 |
| 3 - 4 | 200 | A 0 B 1 C 0 Tot. 1 0.500 | 210 | A 0 B 1 C 0 Tot. 1 0.476 | 290 | A 0 B 0 C 0 Tot. 0 0.000 | 250 | A 0 B 0 C 0 Tot. 0 0.000 | 338 | A 0 B 2 C 0 Tot. 2 0.591 |
| 5 - 6 | 315 | A 0 B 1 C 0 Tot. 1 0.314 | 180 | A 0 B 1 C 0 Tot. 1 0.555 | 350 | A 1 B 0 C 0 Tot. 1 0.285 | 304 | A 0 B 1 C 0 Tot. 1 0.329 | 365 | A 0 B 1 C 0 Tot. 1 0.277 |
| Grand tot. | 795 | 2 0.251 | 660 | 2 0.203 | 950 | 2 0.210 | 809 | 2 0.247 | 1068 | 3 0.280 |

A: Translocation between Y and 2nd chromosomes.
B: Translocation between Y and 3rd chromosomes.
C: Translocation between 2nd and 3rd chromosomes.

Table 2. The frequency of reciprocal translocations at interval and during spermatogenesis after irradiation (500r of X-rays).

| Time eggs were laid after mating (days) | Time mating was made after irradiation (days) 1 - 2 No. of tests | No. of trans. | % | 3 - 4 No. of tests | No. of trans. | % | 5 - 6 No. of tests | No. of trans. | % |
|---|---|---|---|---|---|---|---|---|---|
| 1 - 2 | 570 | A 0<br>B 2<br>C 1<br>Tot. 3 | 0.526 | 325 | A 0<br>B 2<br>C 1<br>Tot. 3 | 0.923 | 475 | A 2<br>B 4<br>C 0<br>Tot. 6 | 1.263 |
| 3 - 4 | 228 | A 0<br>B 1<br>C 0<br>Tot. 1 | 0.438 | 310 | A 0<br>B 2<br>C 1<br>Tot. 3 | 0.967 | 250 | A 2<br>B 5<br>C 0<br>Tot. 7 | 2.800 |
| 5 - 6 | 196 | A 0<br>B 1<br>C 0<br>Tot. 1 | 0.510 | 509 | A 2<br>B 3<br>C 0<br>Tot. 5 | 0.999 | 285 | A 1<br>B 2<br>C 0<br>Tot. 3 | 1.052 |
| Grand total | 994 | 5 | 0.503 | 1144 | 11 | 0.965 | 1010 | 16 | 1.594 |

A: Translocation between Y and 2nd chromosomes.
B: Translocation between Y and 3rd chromosomes.
C: Translocation between 2nd and 3rd chromosomes.

Table 3. Comparison of the translocation frequency among Y, 2nd and 3rd chromosomes in control and irradiated group.

| Chromosomes | Control No. of trans. | Control % | 500r No. of trans. | 500r % |
|---|---|---|---|---|
| Y : 2 | 2 | 18.181 | 7 | 21.875 |
| Y : 3 | 8 | 72.727 | 22 | 68.750 |
| 2 : 3 | 1 | 9.090 | 3 | 9.343 |

The mean frequency of reciprocal translocations appearing in the control group was 0.256% in D. melanogaster Seoul strain. At three intervals tested, no significant difference in the frequency was observed.

The frequency of reciprocal translocations during spermatogenesis showed 0.503% on 1-2 days and 1.594% on 5-6 days. It seems that the rate on 5-6 days was three times that on 1-2 days.

The comparison of the translocation frequency among Y, 2nd and 3rd chromosomes showed that the Y and 3rd chromosomes were involved most frequently.

Milkman, Roger D. Syracuse University, New York. cve polygenes in laboratory mutant stocks.

Combinations of genes producing the cve phenotype have arisen in the progeny of crosses among the following laboratory strains: Ore R, y ct ras f, od sy, dp cn bw, and ey. Inbreeding and intrachromosomal recombination increase the numbers of cve flies, as is shown in the table. All matings were 4 x 4 except that inbred lines began with pair matings. In each inbred sample, 100 flies from each of 10 replicate lines were examined. The table shows the number of samples with at least one cve fly per thousand (at 18°C) and the percentage of cve flies. Comparison results are given for flies collected in nature (previously reported). Data for the inbred $F_6$ are not quite complete. Here the increased percentage of cve flies was found in spite of a decrease in incidence in the progeny of the major contributing line, od sy X y ct ras f, which contained 58 cve flies in the mass $F_6$ and 46 in the inbred $F_6$. Rapid response to selection for cve in the od sy X y ct ras f mass progeny is seen from the penetrance after 4 generations: males, 23%; females, 70%. Penetrance is much lower at 25°.

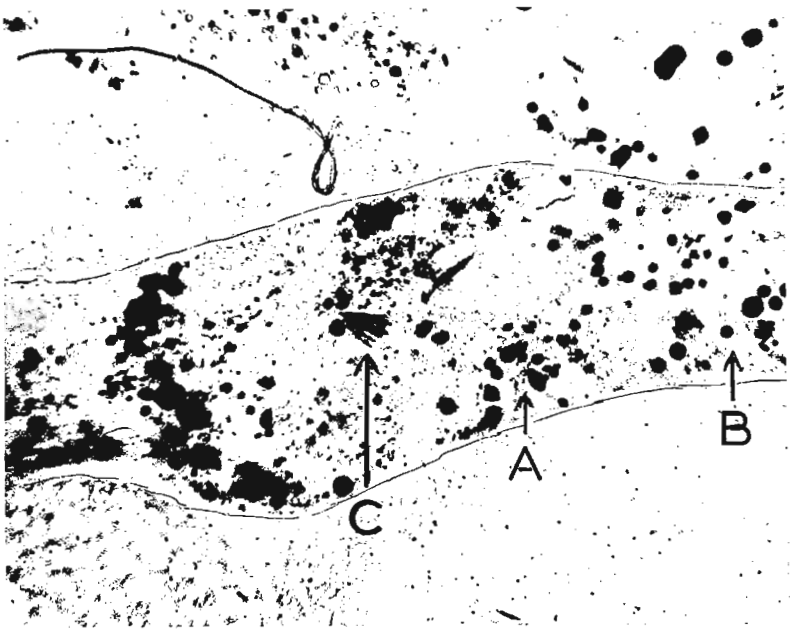
| | No. of Samples | | % cve |
|---|---|---|---|
| | Total | With cve | |
| Parental stocks | 6 | 3 | 0.07 |
| Parental inbred $F_2$ | 6 | 3 | 0.07 |
| $F_1$ | 15 | 2 | 0.01 |
| Mass $F_2$ | 15 | 4 | 0.06 |
| Inbred $F_2$ | 15 | 6 | 0.19 |
| Mass $F_6$ | 15 | 7 | 0.49 |
| Inbred $F_6$ | 15 | 12 | 0.65 (est.) |
| $F_2$'s from wild inseminated females | 70 | 54 | 0.70 |

Traut, H. Universität Münster, Germany. Pre-existing Y-suppressed lethals in Drosophila melanogaster.

Individual In49 v $sn^{x2}$ B males were crossed with X•Y females (genotype $Y^S$•X InEN y v•$Y^L$). $F_1$ virgins were mated singly to $Y^S$•X InEN y $y^{x2}$•$Y^L$ males (no free Y). The $F_2$ In49 v $sn^{x2}$ B males carry no Y. If this class of males is missing this may be due either to an "orthodox" lethal or to a lethal, the expression of which is suppressed by the Y-chromosome. In order to distinguish between these two possibilities, $F_2$ virgins carrying the In49 v $sn^{x2}$ B chromosome were mated to males with a free Y. If in the progeny of these females the In49 v $sn^{x2}$ B male class was missing again, an orthodox lethal was present, if not, a Y-suppressed lethal. The progeny of each P male has been scored separately. The Y-suppressed lethals found by us were not distributed randomly over the total offspring but could be traced back - even through several broods - to only a few P males. Therefore, these Y-suppressed lethals pre-existed already in the P males, from which they were derived; i.e. they did not arise as new mutations in the germ cells of these males. From 78 P males tested, 6 carried a pre-existing Y-suppressed lethal (7.7%). This high accumulation of Y-suppressed lethals seems to have been enabled by the fact that the In49 v $sn^{x2}$ B males were kept in the stock cultures with X•X Y females. Thus spontaneously arising Y-suppressed sex-linked lethals are conserved instead of being eliminated through homozygosity of the X-chromosome. Since these experiments were not especially designed to detect Y-suppressed lethals but for another purpose, the nature of these lethals has so far not been analyzed. (Work supported by USAEC grant A T 11-1-195 to Dr. H. J. Muller and co-workers.)

Clancy, C. W., Ann Sullivan, and Wm. H. Vandling. University of Oregon. Ommochrome-like pigment in the Malpighian tubule cells of the eye color mutant, cho.

The Malpighian tubules of the recessive, X-linked, eye color mutant, chocolate (cho) are strikingly colored by a purplish to brown pigment(s) located in the cytoplasm of the tubule cells (See DIS 29:75 for original report and description of $cho^1$ and $cho^2$). The accompanying photograph shows the several forms in which the pigment exists in a single cell. The letter (A) designates the irregularly shaped, dark purple granules, (B) the spherules of various size ranging in color from pale yellow to dark brown, and (C) the large, purple, fan-shaped crystals.




Since the original report states that the color of the tubules is "bright yellow like +," it was at first imagined that the trait might be due to some infectious agent contaminating our strains. Genetic tests eliminated this possibility and defined the tubule trait as a pleiotropic effect (additional phene) of the mutation.

A developmental study by one of us (A. S.) determined that about 40% of late first instar larvae of $cho^1$ are separable from wild type by the bright orange color of their tubules. By the latter part of the second instar, all of the cho larvae have tubules containing the anomalous pigment elements mentioned above, and are easily separated from wild type. The peak development of the pigment seems to occur during the mid-pupal period.

Quantitative fluorometry of the pteridine components of the tubules reveals no significant differences from wild type, but genetic and solubility tests assign the anomalous pigment to the ommochromes. An attempt to induce formation of the pigment in the tubules of the compound, $cho^1v$, by injecting ommochrome precursors, kynurenine, and hydroxy-kynurenine, into late third instar larvae was unsuccessful, although the bright-orange eye color of the hosts was transformed to cho indicating effectiveness of the solutions in producing (as expected) ommochrome pigment in the eye tissues. Similarly, transplantation of cho v tubules into wild type gave a negative result, as did an experiment in which ovaries were allowed to develop in wild type and the tubules of the recovered cho v offspring studied microscopically.

Systematic comparisons with respect to the pteridine pigments of the eyes by means of paper chromatography and fluorometry (W. H. V.) enable one to distinguish $cho^1$ from $cho^2$, the latter having about three times as much of the drosopterin complex as the former. Both mutant alleles have reduced amounts of the drosopterins and of iso-xanthopterin as compared to wild type, and both accumulate excess amounts of the Hb pterins.

In retrospective summary, it appears that the cho mutants mimic the third chromosome recessive, "red" (red Malpighian tubules, 3 - 55.5±) reported by Oster in DIS 28:77, and investigated by Aslaksen and Hadorn in 1957 (Archiv J. Klaus-Stiftung 32:464).

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Marien, Daniel. Queens College. Drosophilidae from the northern Netherlands.

Collections were made between September, 1963 and August, 1964 in the vicinity of Groningen in the north of Holland. The flies were attracted to large containers of decaying fruit, usually banana, but including plums, apples, tomatoes, grapefruit, and oranges as available locally. Since the flies were trapped incidental to collecting samples of D. subobscura for experimental purposes, and since but one collecting technique was employed, the list of species is no doubt incomplete. Although some are from several nearby localities, and the coastal island of Schiermonnikoog, the majority of flies were trapped in the "Hortus de Wolf," a small, mixed woodland near the Genetical Institute of the State University at Groningen. Most of the species found there, however, are not kept in stock at the Institute and only a very few specimens of one D. melanogaster mutant, being used in experiments from March, 1964, were recovered in the "Hortus," and only after that time.

Sobels, Vlijm, and Lever (1954, Arch. Néerlandaises Zool., 10:357-374) have published on the distribution of Drosophila in the Netherlands, but they had no material from Groningen or the northern part of the country. Other than Parascaptomyza and Chymomyza, no species not listed by Sobels et al. were found in the present survey. Four species recorded by them are absent from the Groningen collection, but they are either very uncommon (D. rufifrons, D. cameraria, D. polychaeta) or of very restricted ecology (D. macularis). Their identifications of D. bifasciata and D. helvetica are probably erroneous; their records of the former are most likely referable to D. obscura and of the latter to D. silvestris. Some differences were noted between our two investigations, particularly my finding that D. obscura was much more common than D. subobscura, that D. limbata and D. littoralis were not at all rare, and that D. deflexa occurred in woodland. Some of these differences may be due to differences in extent, intensity, and time of collecting.

Frequency and distribution by month of drosophilid species in the northern Netherlands in 1963-1964

| Species | Month | ♀ | ♂ | Total |
|---|---|---|---|---|
| Chymomyza costata | May-July | 0 | 3 | 3 |
| Parascaptomyza disticha | July-Oct | 3 | 3 | 9[a] |
| D. deflexa | June-July | 8 | 9 | 17 |
| D. busckii | June-Nov | 43 | 47 | 90 |
| D. melanogaster | April-Oct | 259 | 319 | 578 |
| D. simulans | June | - | 2 | 2 |
| D. obscura | April-Nov | 398 | 771 | 1169 |
| D. silvestris | May-Nov | 44 | 124 | 168 |
| D. tristis | July, Aug, Oct | 5 | 13 | 18 |
| D. ambigua | June-Oct | 16 | 18 | 34 |
| D. subobscura | April-Nov | 276 | 325 | 601 |
| D. transversa | April, Oct | 1 | 2 | 3 |
| D. phalerata | June-Oct | 26 | 37 | 63 |
| D. kuntzei | June-Oct | 3 | 16 | 19 |
| D. limbata | Apr, July, Oct | 20 | 18 | 38 |
| D. littoralis | June-Aug | 6 | 9 | 15 |
| D. testacea | May-June | 2 | 2 | 4 |
| D. funebris | April-Nov | 115 | 272 | 387 |
| D. hydei | July-Oct | 49 | 51 | 100 |
| D. immigrans | June-Nov | 58 | 203 | 261 |
| D. fenestrarum | Sept | 1 | 0 | 1 |
| | | | | 3580 |

[a]Including 3 unsexed individuals.

Suzuki, D. T. University of British Columbia, Vancouver, Canada. The effects of mitomycin C on crossing over in Drosophila melanogaster males.

It has been demonstrated that the antibiotic, mitomycin C (MC) increases mitotic crossing over in Ustilago and Saccharomyces (Holliday, Genetics 50:323, 1964), in öogonia of Drosophila (Suzuki, Genetics 51:635, 1965) and somatic cells of Drosophila (E. B. Lewis - personal communication). It was felt that crossing over in spermatogonia of Drosophila males might be induced by injection of MC.

Males, 24 to 48 hours after eclosion, were injected in the gonadal area with an MC (100 µg/ml) or saline (0.7N) solution. Each male was mated separately with four virgin females and transferred, without etherization, to fresh vials with four virgins at two-day intervals for sixteen days. Each set of inseminated females was transferred to fresh vials twice at four-day intervals to ensure maximal recovery of treated cells. The markers used were ru, h, st, $p^p$, ss, $e^s$ on chromosome 3. The results are shown in the following table:

| | | BROOD NUMBER | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
| MC | Total | 2,822 | 5,306 | 5,995 | 7,308 | 6,646 | 5,513 | 2,649 | 2,145 |
| | aver/male | 108.5 | 212.2 | 239.8 | 292.3 | 265.8 | 220.5 | 120.4 | 126.2 |
| | Crossovers | 0 | 0 | 0 | 1* | 0 | 0 | 0 | 0 |
| NaCl | Total | 2,929 | 3,629 | 3.160 | 2,835 | 2,724 | 2,296 | 1,742 | 715 |
| | aver/male | 195.3 | 241.9 | 210.7 | 189.0 | 227.0 | 176.6 | 134.0 | 71.5 |
| | Crossovers | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

* in the region ss-$e^s$

The absence of effect of MC at a concentration known to be mutagenic in Drosophila males (Mukherjee, Genetics 51:947, 1965) and recombinogenic in females (Suzuki, 1965) might indicate that the nature of induced gonial exchanges in males is different from that in females. (This research was supported by N.R.C. grant A-1764 and USAEC grant AT(45-1)-1924.)

Di Pasquale, A. and L. Zambruni. University of Milan, Italy. Localization of the "brown spots" character of Drosophila melanogaster.

As previous investigations have shown, the inheritance of the brown spots character (whose phenotypical manifestation appears in the females only after copulation, see DIS 37), is bound to the 2nd chromosome (see DIS 33-34). A new investigation has been brought about to find its localization in the chromosome. Using as markers b (48,5), cn (57,5), vg (67,0), experiments were made with the aim of revealing cases of recombination with bsp; to avoid errors of classification, since bsp has not a 100% penetrance, it was preferred to use another method which permitted the recovery of each chromosome, recombined or not, in isogenic condition and therefore in stable lines. As bsp females mated with males of different stocks show typical spots with significant differences according to the genotype of the copulating male, the classification of the individual lines (i.e. of the single chromosomes) as bsp or + was done on the basis of observations made on females mated with bsp males.

88 chromosomes were analyzed: according to the results obtained, bsp must be located ± 7 units to the left of b, being the frequencies of recombination 7.9% ± 2.87 bsp-b, 19% ± 4.10 bsp-cn, 46.0% ± 5.12 bsp-vg.

Differences in the frequency of brown spots manifestation have been found in lines of different genotype which can be explained with the presence of modifiers also located on the 2nd chromosome.

Lee, Taek Jun. Chungang University, Seoul, Korea. Analysis of heterosis in populations of D. auraria.

Korean populations of D. auraria A race are dimorphic with respect to the pigmentation of the abdominal tergites of female. By genetic analysis of these wild flies, the relation, d/d>d/D>D/D has been disclosed as regards the relative frequencies of the genotypes in natural populations.

Homozygotes' dark form (D/D) and homozygotes' light form (d/d) were mixed together in a definite initial ratio in population cages. Approximately one year later, the frequencies of the light and dark forms reached an equilibrium, the light one being usually more frequent than the dark one. This indicates that the heterozygotes' dark form possesses the highest adaptive value. In order to discover the reason for this adaptive advantage of the heterozygotes, comparisons were made among these forms with respect to the physiological traits of the bearers. Fecundity, longevity of adult and viability at various stages of development were the chief traits chosen for study. All experiments were carried out at 25°C using boiled yeast medium for measurement. As shown in the following tables, heterozygotes' dark were superior to both homozygotes' light and dark in the fecundity and egg hatching rate, although the differences are scarcely significant.

Fecundity; average number of eggs per day (two pairs in one vial)

| D/D | d/D | d/d |
|---|---|---|
| 36.96 ± 2.51 | 40.97 ± 2.83 | 36.91 ± 2.42 |

Longevity; average length of life in days

| | | | |
|---|---|---|---|
| D/D | ♀ 41.63 ± 1.68<br>♂ 38.29 ± 2.01 | Total | 39.89 ± 1.36 |
| d/D | ♀ 38.53 ± 0.46<br>♂ 39.73 ± 0.78 | Total | 39.00 ± 0.42 |
| d/d | ♀ 36.09 ± 2.11<br>♂ 42.36 ± 1.76 | Total | 39.09 ± 1.45 |

Viability;

| | D/D | d/D | d/d |
|---|---|---|---|
| Egg hatching rate (%) | 71.2 | 91.8 | 81.4 |
| Frequency of pupation (%) | 83.7 | 86.1 | 88.8 |
| Frequency of emergence (%) | 67.7 | 83.9 | 82.3 |

Lints, F. A.[1] and E. Zeuthen[2]. Oxygen consumption of the egg in wild and inbred strains of Drosophila melanogaster.

In order to verify a hypothesis correlating negatively the duration of life to the metabolism and more precisely to the rate of oxygen consumption (Lints, 1963), a series of experiments on the oxygen uptake of Drosophila melanogaster was started. Using the gradient-diver method (Løvlie and Zeuthen, 1962) measurements were made on fertilized eggs--from egg-laying to the emergence of the larva--of two strains, Abeele wild and Abeele inbred F99 and F100.

The eggs were collected from parents of different ages; however, male and female of a given experiment were equally old. Neither the weight, nor linear measurements of the egg was taken. Oxygen consumption was read twice an hour during the entire development of the egg, except for the two or three hours immediately after egg-laying where for technical reasons readings were not possible. The rate of oxygen consumption is expressed in µl/egg/hour. With the 10 to 15 µl divers used, (and for the observed gas consumptions ranging from 2 to 5 x $10^{-2}$ µl/hour) the absolute error of the method is of the order of 6 x $10^{-4}$ µl/hour. All the experiments were run at the temperature of 25°C.

The essential results are as follows: for a mean duration of 19.75±0.56 hours the mean total $O_2$ consumption of the Abeele wild strain is 0.599±0.042 µl, while it has a value of 0.616±0.025 µl for a mean duration of development of 19.80±0.92 hours in the inbred strain. The difference between the consumptions from egg-laying up to emergence was tested by means of the analysis of covariance of total $O_2$ consumption in relation to parental age, and it is not significant (Table 1).

The figure shows the mean rate of $O_2$ consumption for the two strains studied. Each of these curves can be best described by two regressions of rate on time: one starting at egg-laying up to the tenth hour of development; a second starting from the latter point up to the emergence of the larva; i.e., for the last 9 hours of development. The onset of the fairly steep increase in the consumption rates in the second part of the embryogenesis seems to correspond with the first muscular movements which occur around the 10th to 8th hour before emergence (Poulson, 1950); at that time the regression coefficients rise from 0.0026 to 0.0161 for the wild strain, and from 0.0080 to 0.0152 for the inbred one.

The difference between the regression coefficients rate on time for the first ten hours of development is highly significant ($t = 3.2$; $0.001 < P < 0.01$); however, for the same period there is no significant difference for the means. In the second part of embryogenesis the difference between regression coefficients is statistically not significant, and the difference between means gives a t value of 1.5 ($0.1 < P < 0.2$). Indeed, a close examination of the figure shows that, while starting at a somewhat lower level the consumption rate of the inbreds from the fifth hours after egg-laying, and continuously up to emergence is a little higher than that of the wild strain, and this accounts for the small (not significant) difference in total consumption.

The hypothesis relating duration of life to respiration rate postulated a higher $O_2$ consumption for inbreds. The data here supplied neither fully support, nor contradict the hypothesis. Indeed, on the one side, after 20 hours of development the total consumptions are statistically not different although a little higher for the inbreds; but, on the other side, at least in the first ten hours of development, the slopes of the rate curves are significantly different, the increase in rate being much higher for the inbreds. One would need to know the evolution of the rate curves during the next steps in development, i.e., during the larval and pupal stages, and the respiration rates during adulthood. More precisely one should establish whether the small, but regular difference in rate in favor of the inbreds is maintained.

More experiments of the type reported here are being performed with eggs of other strains, taking into account differences in egg size and weight. The experiments will be extended to include later developmental stages (larval, pupal, and imagos). A complete report of the present data will be published in the "Comptes Rendus des Travaux du Laboratoire Carlsberg."

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[2]Biological Institute of the Carlsberg Foundation, 16, Tagensvej, Copenhagen N, Denmark.

Table 1

| Item | Abeele wild | Abeele inbred | Difference t | Difference p |
|---|---|---|---|---|
| Number of observations | 6 | 10 | -- | |
| Duration of development (hours) | 19.75 ±0.56 | 19.80 ±0.92 | -- | |
| Mean total consumption (ul) | 0.599±0.042 | 0.616±0.025 | 0.9* | 0.3-0.5 |

| | Abeele wild | | | Abeele inbred | | | Differences** means | | between regression coeff. | |
|---|---|---|---|---|---|---|---|---|---|---|
| | b | t | p | b | t | p | t | p | t | p |
| Regression: rate on time (10 first hours) | 0.0026 | 2.3 | 0.05-0.10 | 0.0080 | 6.2 | <0.001 | 0.7 | 0.50 | 3.2 | 0.01-0.001 |
| Idem: 9 last hours | 0.0161 | 8.6 | <0.001 | 0.0152 | 10.0 | <0.001 | 1.5 | 0.1-0.2 | 0.3 | -- |
| Regression: total consumption on parental age | 0.0084 | 1.3 | 0.2-0.3 | 0.0018 | 0.3 | -- | --*** | -- | 0.7 | 0.50 |

*Difference tested by means of the covariance analysis of total $O_2$ consumption in relation to parental age.

**Differences tested by means of the analysis of variance.

***See item: difference between mean total consumption.

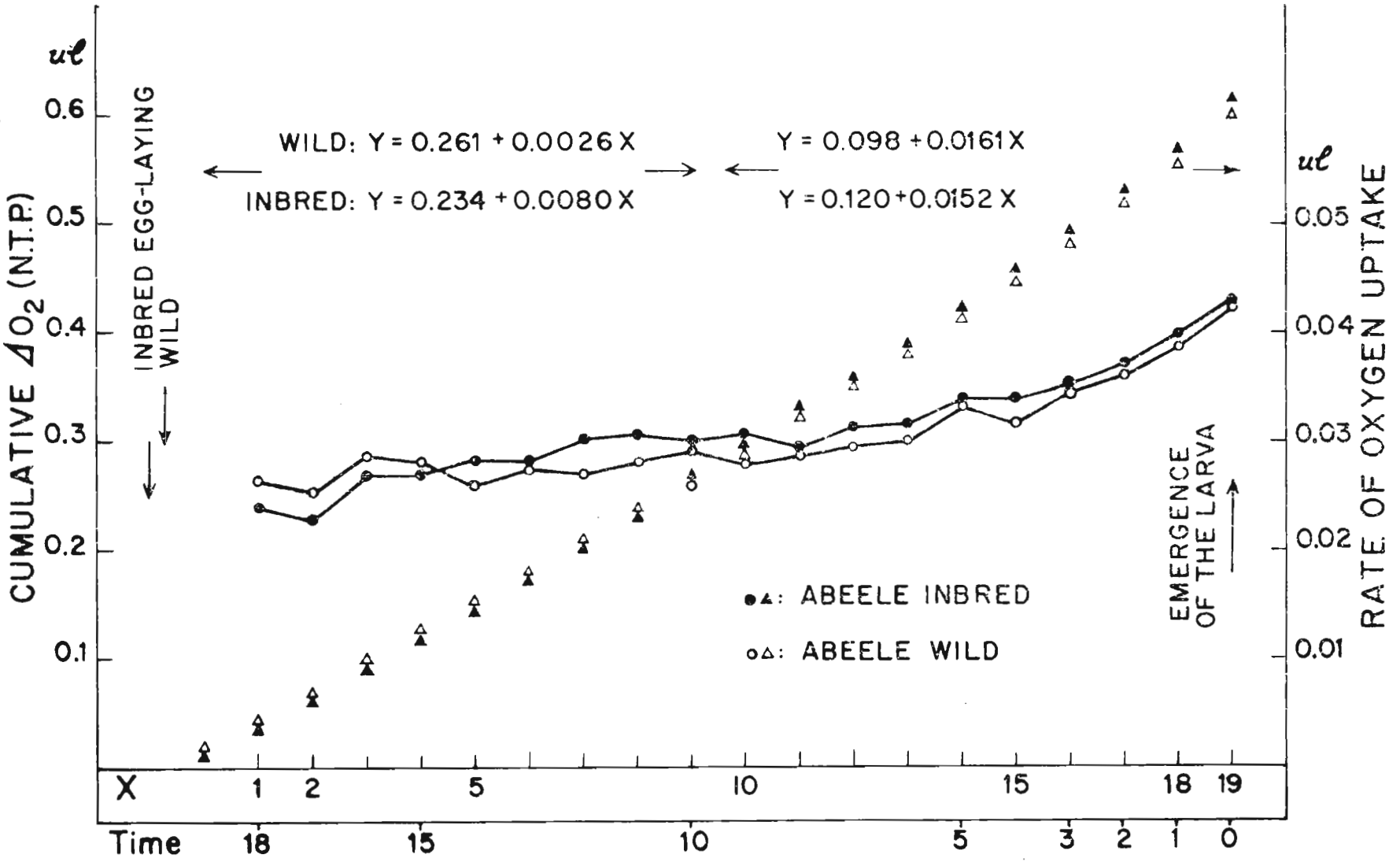


Figure 1.--Curves delineated by circles: The rate of oxygen consumption (right ordinate) is plotted against the observation number (abscissa, upper scale), the time between two observations being exactly one hour. The rate curve shown for each strain is a mean calculated from the several different curves which were observed experimentally, and made to coincide on the time axis at the moment of emergence of the larva. (This is, in fact, the only biologically defined point in time, since Drosophila females can retain the egg in the uterus for a variable period, and hence the exact time of fertilization cannot be determined).

Curves delineated by triangles: Cumulative oxygen uptake ($\Sigma \Delta O_2$ N.T.P.--left ordinate) is plotted against time in hours (abscissa, lower scale), the origin of time being the moment of emergence of the larva. The first point on each cumulative oxygen uptake curve was calculated by means of the appropriate regression formula.

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Khan, A. H. University of Cambridge, England. Tests for hydroxylamine mutagenesis in Drosophila.

The mutagenic effect of hydroxylamine has been tested on mature spermatozoa of Drosophila using an adult feeding method. Treatment was in one-pint bottles, the bottoms of which were covered with a double thickness of filter paper. The filter paper was kept lightly soaked with the hydroxylamine solution (containing 5% glucose) during the period of treatment. Fifty newly-hatched Oregon-K males, were starved for 12 hours, and placed in the treatment bottles for 24 hours during which time the treatment solution was their only source of nourishment.

Treated males were examined by the Muller-5 (Basc) method for the frequency of sex-linked lethal mutations. A single three-day brood was examined by individually mating one treated male to two Muller-5 virgin females.

The chemical was highly toxic, killing all males at a 0.4% treatment. The survival at 0.3, 0.2 and 0.1% concentrations of hydroxylamine was 65.0, 82.5 and 97.5% respectively. The sex-linked lethal results listed in Table 1 show that hydroxylamine is not mutagenic in Drosophila under these conditions.

Table 1.--Sex-linked recessive lethal frequencies in Drosophila males after adult feeding treatments with hydroxylamine.

| Concentration of hydroxylamine (%) | 0.1 | 0.2 | 0.3 |
|---|---|---|---|
| Duration of treatment (Hrs) | 24 | 24 | 24 |
| Survival (%) | 97 | 82 | 65 |
| No. males examined | 45 | 33 | 28 |
| Average no. chromosomes examined/male | 9 | 10 | 13 |
| No. chromosomes examined | 411 | 345 | 364 |
| No. lethals | 0 | 1 | 0 |
| Lethals (%) | 0.0 | 0.28 | 0.0 |

Lee, P. Y. and V. A. Strangio. University of Melbourne. Brood sensitivity to the induction of polygene mutations.

Males from a highly-inbred wild type D. melanogaster stock were irradiated with 500r X-rays and then mated individually to three virgin females from the same stock. Four broods were established from successive matings, each three days in duration. Sternopleural bristles were counted in $F_1$ females only (see Mukai et al., 1963). Although pooled data from all four broods indicate a significantly increased variance of the bristle number distributions in females from the irradiated series, the preliminary results have so far failed to reveal a detectable sensitivity pattern.

Mather, Wharton B. University of Queensland, Australia. New inversions in D. rubida.

Two new complex inversions and one new simple inversion have been detected in flies from New Guinea on chromosome II R--complex G and H and simple I. Relating these to the giant chromosome photographic map for D. rubida (Mather, W. B., 1961, Genetics 46:799) G has limits 6.1 - 15.3, H 7.1 - 14.1, and I 4.7 - 5.5. In addition, a new complex--J on chromosome III has limits 23.5 - 26.0.


Mather, Wharton B. University of Queensland, Australia. D. rubida inversion polymorphism.

It has been shown previously (Mather, 1964) that certain inversions vary significantly in frequency at different times of the year at Port Moresby, New Guinea. It is the purpose of this report to measure inversion frequency in a different geographical region at Bulolo, New Guinea and to compare the inversion frequency patterns in these two populations.

Material was collected from fermenting banana baits at Bulolo in August, 1963 and February, 1964.

The material was analyzed by mating males and despermed females from the wild against a standard strain and scoring seven larvae from each mating against a photographic map (Mather, 1961). Salivary chromosomes were prepared by the method given in Strickberger (1962).

The results are set out in Table 1. It should be noted that when comparisons were made between August and February only certain inversions showed significant differences in frequency. These are indicated.

Particular attention should be drawn to the following results:

1. In chromosome II L inversion A has a significantly higher frequency in August than February in both males and females.

2. In chromosome II R inversion A has a significantly higher frequency in August than in February in females but not in males.

Table 1

| Chromosome II | August 1963 (percent) | | February 1964 (percent) | |
|---|---|---|---|---|
| | ♂ | ♀ | ♂ | ♀ |
| Standard | 0 | 0 | 0 | 1.0 |
| LA | 14.4 | 15.7 | 5.2 | 8.1 |
| RA | 35.6 | 44.3 | 31.0 | 26.3 |
| B | 44.9 | 48.1 | 41.4 | 46.9 |
| C | 0.4 | 1.4 | 0 | 0 |
| D | 99.6 | 98.6 | 100.0 | 98.6 |
| Chromosome III | | | | |
| Standard | 27.1 | 28.6 | 27.9 | 27.6 |
| A | | 0.9 | | |
| B | | 0.9 | | |
| D | 53.4 | 53.5 | 45.8 | 49.0 |
| E | 49.6 | 54.5 | 54.8 | 54.3 |
| H | 12.3 | 12.2 | 13.4 | 13.3 |
| I | 21.6 | 14.6 | 15.2 | 16.2 |
| J | 0 | 0 | 0.3 | 0 |
| Flies scored | 121 | 106 | 146 | 103 |

N. B. Arrows indicate differences significant at the 5% level.

When the range for inversion frequency at Bisianumu, Port Moresby, (Mather, 1964) is compared with that at Bulolo (Table 2) it is found that for all inversions there are different non-overlapping ranges. Bulolo is higher for II LA, II RA, B, D, III D, E, H, I, and J and lower for II RC, and III A and B. Perhaps the most outstanding feature of the Bulolo population is that it is virtually homozygous for the complex inversion II RD.

Thus, on cytological grounds the Bulolo population may be designated a different race from the Port Moresby population. Sexual isolation tests are to be carried out between the two populations.

Table 2

| Chromosome II | Port Moresby Range % | Bulolo Range % |
|---|---|---|
| Standard | 23.5 - 47.2 | 0.0 - 1.0 |
| LA | 0 - 2.9 | 5.2 - 15.7 |
| RA | 5.4 - 10.1 | 26.3 - 44.3 |
| B | 12.7 - 38.2 | 41.4 - 48.1 |
| C | 23.6 - 34.1 | 0 - 1.4 |
| D | 25.9 - 46.1 | 98.6 - 100 |
| F | 0 - 1.0 | - |

Table 2.--continued

| Chromosome III | Port Moresby Range % | Bulolo Range % |
|---|---|---|
| Standard | 52.9 - 72.2 | 27.1 - 28.6 |
| A | 2.3 - 5.6 | 0 - 0.9 |
| B | 1.0 - 6.7 | 0 - 0.9 |
| D | 4.4 - 9.8 | 45.8 - 53.5 |
| E | 22.2 - 41.3 | 49.6 - 54.8 |
| H | 0 - 1.5 | 12.2 - 13.4 |
| I | 0 - 1.1 | 14.6 - 21.6 |
| J | - | 0 - 0.3 |
| Flies scored | 361 | 476 |

Acknowledgements are due to Sheridan Butler, Janice Dines and Rosalyne Spurway for technical assistance and Mr. D. Angus who collected the flies.

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McIntire, Sarah A. and Thomas Gregg. Miami University, Oxford, Ohio. Pteridines and the white locus in D. melanogaster.

It has been shown by Green (Heredity, 1959) and Judd (Genetics, 1964) that the white locus in D. melanogaster is genetically complex in that non-complementary mutations exist in at least five different sites that are separable by crossing over. It has also been shown (Gregg unpublished) that, to some extent, different alleles at the white locus control the presence of different pteridine compounds associated with red pigment in the wild type eye. Since the pteridines are a more direct reflection of the action of the genes that control their presence than the phenotype of the eye itself, it was felt that a study of the pteridines present in heads of mutant individuals carrying different white alleles might clarify the nature of the genetic complexity of the locus.

Pteridine accumulation patterns for thirty-seven white alleles were determined, using chromatographic methods similar to those of Throckmorton (Univ. Texas Publ. 6205), but using a paper in the shape of an Erlenmeyer flask silhouette (Harrison, Hayes, and Chua, Ohio Jour. Sci. in press) for better separation. The results are shown in the table below.

It appears to be impossible to explain the ten patterns in terms of a single genetic block, which would indicate that the locus is polycistronic in nature. But, if the white locus is polycistronic and if the established recombination sites represent different cistrons, it is surprising to find mutations at different sites producing the same pattern of pteridine accumulation. This particular observation is more readily explained if one assumes that the white locus is a single cistron. It is also difficult to explain the results by assuming that there are several cistrons present, any one of which could contain more than one of the established recombination sites. For instance, if pattern II is controlled by a cistron containing sites 1, 2, and 4, then it should also contain 3, but alleles at 3 produce at least two other patterns, III and IV, but not pattern II. The same is true for pattern IV, in that this pattern is produced by alleles at sites 2, 3, and 5, while alleles at site 4 produce other patterns.

However, the difficulties of explaining the lack of correspondence between the recombination sites and the pteridine patterns, assuming a polycistronic locus, appear to be considerably less troublesome than explaining the ten different patterns on the basis of a locus containing a single cistron. Therefore, in spite of the lack of correspondence, the existence of ten distinct patterns appears to be strong evidence for the polycistronic nature of the white locus.

Table 1.--Pteridine Accumulations Controlled by Various Alleles at the White Locus

| | + aRM A58K11 a58112 mR7aH1 $m^4$ $m^4w$ r,dup | Bwx 1* bf 2 $bf^2$ e 4 $e^2$ 4 ch 4 | sat 2 crr a 3 $a^2$ 3 $a^4$ 3 | col 2 sp-w 5 sp $sp\text{-}w^4$ $a^3$ 3 co 3 | bl | cf cp | i h 4 "a"59e15 | w 4 17G2 57 r,def t | 11E4 | $ec^3$ |
|---|---|---|---|---|---|---|---|---|---|---|
| Drosopterins | ++++ | - | (+) | (+) | - | ++ | - | - | (+) | + |
| Isoxanthopterin | +++ | (+) | + | (+) | + | + | - | - | - | - |
| Xanthopterin | +++ | - | (+) | + | + | (+) | - | - | - | - |
| Blue-violet | ++ | - | - | - | (+) | - | - | - | - | - |
| Sepiapteridine | ++ | (+) | ++ | +++ | (+) | ++ | - | - | - | - |
| 2-amino-4-hydroxypteridine | +++ | - | - | - | - | - | - | - | - | (+) |
| Biopterin | +++ | (+) | ++ | +++ | +++ | ++ | (+) | - | - | - |
| | I | II | III | IV | V | VI | VII | VIII | IX | X |

*Recombination site (Judd, Genetics, 1964)

++++ very large amount
+++ large amount
++ moderate amount
+ small amount
(+) trace amount
- none

Ritossa, F. M. and P. Cammarano. Oak Ridge National Laboratory, Tenn. Isolation and properties of ribosomes from D. melanogaster.

D. melanogaster larvae were homogenized in an all-glass apparatus with two volumes of a medium containing 0.05M Tris pH 7.6, 0.025M KCL, 0.005M 2-mercaptoethanol, 0.25M sucrose; when present, $Mg^{++}$ was either 0.1 or 5mM. The homogenate was centrifuged 20 min. at 20,000 x g, and the resulting postmitochondrial supernatant was further centrifuged at 105,000 x g for 90 min. The material sedimenting at 105,000 x g was resuspended in the homogenization medium and immediately used for analysis in a 10%-34% sucrose density gradient. At times, sodium deoxycholate (1.2%) was added to the postmitochondrial supernatant; in this case, the material sedimented at 105,000 x g was resuspended in the above medium and recentrifuged at 105,000 x g for 90 min. Occasionally homogenization was performed by grinding the tissue under liquid nitrogen; the resulting powder was resuspended in the homogenization medium and processed as above. Isolation of the particles in the medium containing 5mM $Mg^{++}$ led to the appearance of a heavy peak of approximately 170 S (fig. 1A). Treatment of the isolated material with amounts of ribonuclease which are known to result in selective breakage of the interribosomal RNA (10 mμg/mg of RNA) in a variety of materials did not alter the sedimentation profile of this peak. The same sedimentation profile was observed when DOC was used during the isolation procedure.

Centrifugation of the same preparations in a sucrose density gradient in the absence of $Mg^{++}$ results in the resolution of a minor protein component uniformly spread throughout the gradient and a sharp peak sedimenting in the region pertaining to particles of sedimentation constant 80-83 (fig. 1A); this peak showed a 280/260 ratio typical of ribonucleoprotein particles (0.54). No evidence existed for the appearance of subunits of the main peak component comparable to the 50 S and 30 S subunits described in other organisms. The extent of these phenomena was not influenced by either changes in the homogenization conditions or by the use

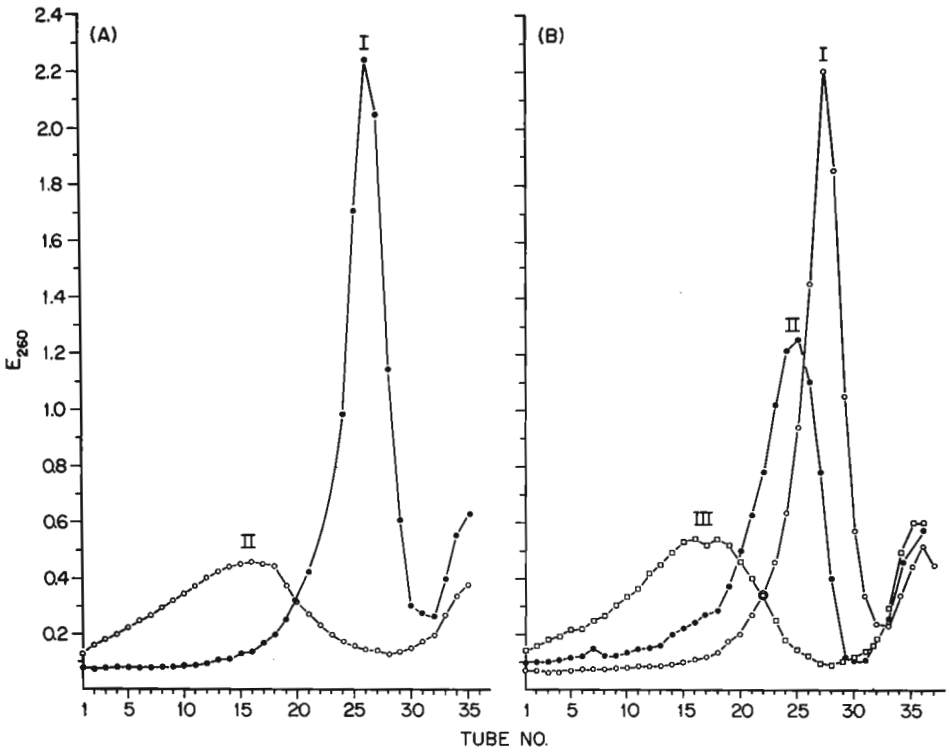



Fig. 1: (A) Sucrose density gradient profiles of ribosomes isolated in the absence of $Mg^{++}$ (peak I) and in the presence of $Mg^{++}$ (5mM) (peak II). Ribosomes obtained in the presence of $Mg^{++}$ 0.1mM were centrifuged in a 10-34% linear sucrose gradient buffered with the isolation medium, except $MgAc_2$ was omitted (peak I). Ribosomes obtained in the presence of $Mg^{++}$ (5mM) were centrifuged in a similar gradient including 5mM $Mg^{++}$ (peak II).
(B) Sucrose density gradient profiles of ribosomes isolated in the absence of $Mg^{++}$ but centrifuged in sucrose density gradient containing either 0.5mM $Mg^{++}$ (peak II) or 5mM $Mg^{++}$ (peak III). Ribosomes represented in peak I were centrifuged in a sucrose density gradient without $Mg^{++}$.

of deoxycholate during the isolation procedure. In addition, replacement of Mg with Ca ions at identical concentration did not alter the picture nor lead to the appearance of polysomal peaks although the contrary has been reported. In other experiments, the particles were isolated in the presence of 0.1mM Mg and analyzed in sucrose density gradients of different Mg concentrations. The increase of Mg content in the sucrose gradient from 0.1 to 5mM led to progressive aggregation of the 80 S particles to form the heavy sedimenting peak seen in particles isolated in the presence of 5mM Mg, showing that partial aggregation of the monomeric particles occurred already in the presence of 0.1mM Mg concentration (fig. 1B). It may be noted that the 80 S component isolated from the corresponding region of the sucrose density gradient failed to reaggregate when subsequently centrifuged in a gradient containing 5mM Mg.

The foregoing results indicate that in larvae of D. melanogaster isolation conditions similar to those extensively employed for isolation of polysomes in a variety of organisms lead to the appearance of only one heavy sedimenting peak, resulting perhaps from the non-specific binding of individual ribosomes with a, so far, undefined protein component in the presence of high magnesium concentration.

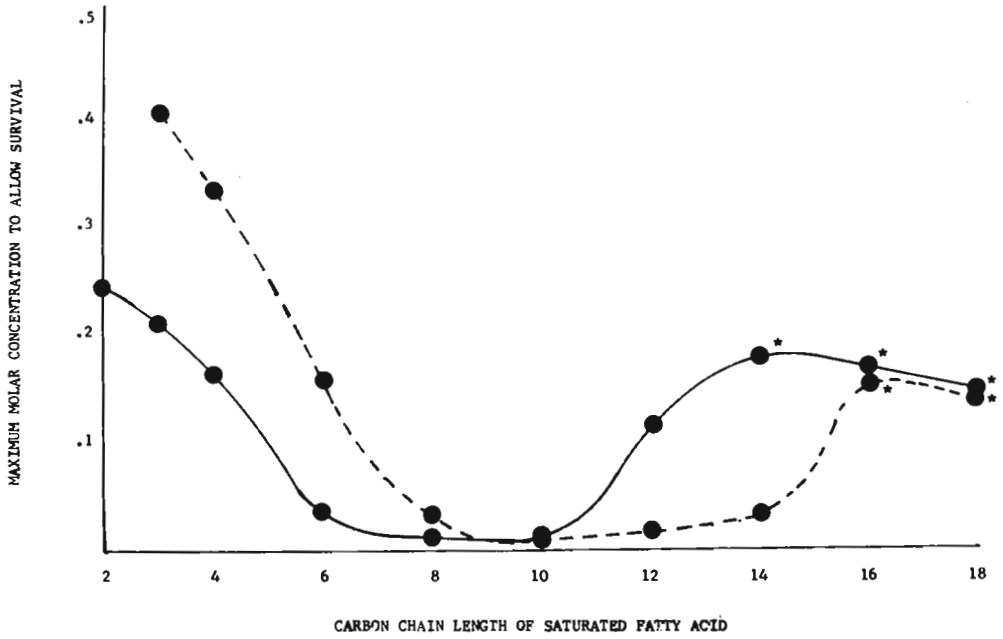
Research carried out at the Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tenn., and jointly sponsored by the Comitato Nazionale Energia Nucleare, Laboratorio di Radiobiologia Animale, Rome, Italy, and by the U. S. Atomic Energy Commission under contract with the Union Carbide Corporation.

Keith, Alec D. University of Oregon.
The effect of exogenous fatty acids on survival in D. melanogaster.

Various saturated fatty acids were added to a cornmeal molasses diet and the effects on the parents or progeny were recorded. The same procedure was repeated using Sang's medium C (J. Exptl. Biol. 33:45, 1956) with good agreement, ruling out any specific effect of the cornmeal molasses medium.

The accompanying graph shows the maximum molar concentration in the media to allow survival. The different effects of fatty acids from their methyl esters are also shown.

Even though both media have a reasonable degree of buffering capacity, the acid strength of short chained fatty acids (less than 6 carbons) was such that the final mixture of medium was usually lowered in pH. This pH change may account for why $C_3$, $C_4$ and $C_6$ methyl esters, having lost their acid groups, give much higher tolerance levels than the corresponding acids. At the other side of the curve, $C_{12}$ and $C_{14}$ acids are solids with low vapor pressures while their esters are liquids with relatively high vapor pressures. It appears that one of these two physical properties is responsible for the difference in tolerance levels, although specific metabolic disturbances by individual molecular species are not ruled out. In both acids and esters, $C_8$ and $C_{10}$ components have very detrimental effects and the adults usually die in less than an hour after exposure. In all cases, with the two media reported, if the adults lived they proved to be fertile and the resulting progeny developed normally.




This graph shows how fatty acids and their methyl esters differentially affect the survival of D. melanogaster. * represents the maximum concentration used (no lethal effect).
----- = methyl esters
_____ = fatty acids

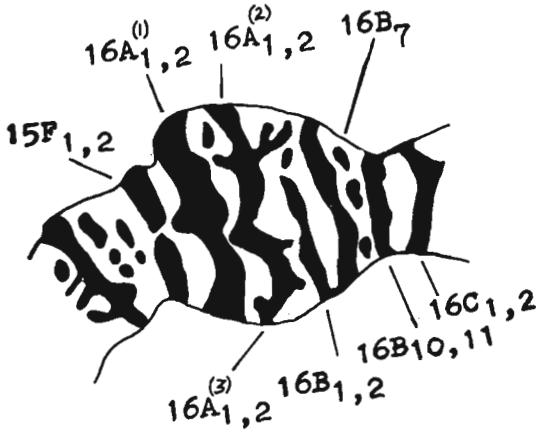
Eichler, V. B. and W. M. Luce. University of Illinois. Cytological observations of the Infrabar-Bar mutant of Drosophila melanogaster.

This report describes the salivary gland chromosome cytology of the Infrabar-Bar double type mutant in Drosophila melanogaster. The stock used was a long inbred forked Infrabar-Bar line established by Luce (1935) from an 18-facet female arising from a mating between inbred forked Infrabar strain and an inbred strain of forked Bar. The Infrabar-Bar line has repeatedly given rise to Infrabar and to Bar.

Hager (1941), in the only report extant on the salivary chromosomes of this particular double type mutant, could find no more than a duplication of the 16A segment accompanied by a swelling or bulging of this region over that found in Bar or Infrabar. Hager's interpretation thus departs from the triplication hypothesis found by others in favorable preparations of the other double type mutants at the Bar locus. In the present study the best preparations suggest an interpretation which reconciles the duplication with the bulging hypothesis of Hager (1941) with the triplication hypothesis of Bridges (1936) and Sutton (1943).

Figure 1 is a photograph of the bulge which is characteristic of the 16A region in salivary gland chromosome preparations. Two sets of bands are immediately apparent by locating the thick bands which represent $16A_{1,2}$. Examination under phase contrast clearly suggests the interpretation indicated in Figure 2. There is a triplication, designated in the figure by the superscripts (1), (2), and (3) of the 16A segment but not in linear order. The second and third segments are somewhat end to end across the chromosome with some overlapping.

This interpretation would explain the apparent increase in the width of the bulge shown here and noted by Hager and also would support the triplication hypothesis of Bridges and Sutton.



Lee, Taek Jun. Chungang University, Seoul, Korea. Drosophila survey in Korea.

Since 1955, a drosophilid survey in Korea has attracted the attention of taxonomists and geneticists, and repeated surveys have been carried out by Chung et al., (1955, 1956, 1958, 1959), Paik and Kim (1957, 1958), Lee (1957, 1958, 1959, 1962), Kang et al., (1958, 1959, 1960, 1962), and Kim (1963).

As the result of studying the above records, approximately 80 species of Drosophilidae occurred in Korea and were known to us.

The author continued the collection of drosophilid flies during a period ranging from September 1956 to November 1963. The collections were made at 54 localities of Korea, resulting in the capture of a total of 94,115 flies involving 65 species belonging to 9 genera as shown in the following table.

Fifty four localities are as follows: Quelpart Is., Yeosu, Hampyong, Mt. Bulkap, Masan, Mt. Chiri, Namwon, Milyang, Mt. Naejang, Jeongeup, Seonyu Is., Mt. Deokyu, Muju, Mt. Palkong, Keumsan, Mt. Daedun, Kimcheon, Yeongdong, Nonsan, Buyeo, Okcheon, Daejeon, Sapsi Is., Mt. Kyelyong, Daecheon, Boryong, Mt. Chilkap, Sangju, Kongju, Mt. Sokli, Hongseong, Cheongju, Yesan, Munkyong, Mt. Undal, Seosan, Asan, Mt. Baekma, Jincheon, Dangjin, Anseong, Hambaek, Icheon, Suwon, Wonju, Samcheok, Incheon, Yongdungpo, Kimpo, Daekwanryung, Mt. Jukyeop, Mt. Ohdai, Mt. Soyo and Mt. Sulak.

| Species | Specimens obtained | Collecting methods or feeding habits |
|---|---|---|
| Stegana sp. from Mt. Sulak | 1 | S |
| Amiota alboguttata | 14 | S, E |
| A. variegata | 1094 | F, S, T, E |
| Leucophenga argentosa | 84 | F, S |
| L. concilia | 33 | F, S |
| L. maculata | 28 | S |
| L. magnipalpis | 32 | S |
| L. ornatipennis | 8 | S |
| L. quinquemaculata | 35 | S |
| L. sp. from Mt. Jukyeop | 1 | S |
| Microdrosophila fuscata | 22 | S |
| M. matsudairai | 769 | F, S |
| M. purpurata | 165 | S |
| M. urashimae | 4 | S |
| Mycodrosophila basalis | 58 | M |
| M. japonica | 5 | M |
| M. koreana | 140 | F, M |
| M. poecilogastra | 30 | M |
| M. shikokuana | 2 | M |
| M. splendida | 7 | M |
| Liodrosophila castanea | 267 | F, S |
| L. sp. from Mt. Kyelyong | 1 | F |
| Scaptomyza apicalis | 64 | S |
| S. graminum | 194 | S |
| Parascaptomyza pallida | 4410 | S |
| Drosophila magnidentata | 2 | S |
| D. raridentata | 13 | S |
| D. histrioides | 194 | F, S |
| D. nokogiri | 14 | F, S |
| D. quadrivittata | 18 | S |
| D. sexvittata | 134 | F, S |
| D. trilineata | 16 | S |
| D. busckii | 574 | F, S, G, T |
| D. coracina | 3443 | F, S, G, T |
| D. puncticeps | 1 | S |
| D. rufifrons | 650 | F, T |
| D. sp. from Hongseong | 1 | S |

| Species | Specimens Obtained | Collecting methods or feeding habits |
|---|---|---|
| D. bifasciata | 315 | F, T |
| D. suzukii | 3759 | F, S, T |
| D. lutea | 209 | F, S, T |
| D. melanogaster | 2584 | F, G |
| D. ficusphila | 5 | F, S |
| D. nipponica | 679 | F, S |
| D. clarofinis | 162 | F, S |
| D. magnipectinata | 8 | F, S |
| D. auraria (A, B, & C race) | 45184 | F, S, G |
| D. sp. from Mt. Sulak | 1 | F |
| D. brachynephros | 2680 | F, S |
| D. angularis | 8174 | F, S |
| D. unispina | 691 | F, S |
| D. takadai | 26 | F |
| D. nigromaculata | 4852 | F, S, G, T |
| D. kuntzei | 38 | F |
| D. sp. from Mt. Soyo | 1 | S |
| D. testacea | 563 | F, S |
| D. bizonata | 4433 | F, S, G, T |
| D. histrio | 875 | F, T |
| D. sternopleuralis | 86 | F, S |
| D. tenuicauda | 24 | S |
| D. immigrans | 116 | F, S, T |
| D. pengi | 1690 | F, T |
| D. virilis | 907 | F, G |
| D. lacertosa | 2734 | F, S, G, T |
| D. cheda | 235 | F, S, T |
| D. sordidula | 556 | F, S, T |
| Total | 94,115 | |

** F(fruits), S(sweeping), G(garbage), T(tree-blood), M(fungi), E(human eye).

Among 65 species of Drosophilidae enlisted here 11 species can be added to the fauna of Korea. They are as follows: Stegana sp. from Mt. Sulak, Leucophenga sp. from Mt. Jukyeop, Microdrosophila fuscata Okada, M. urashimae Okada, M. matsudairai Okada, Liodrosophila sp. from Mt. Kyelyong, D. magnidentata Lee, D. (Paradrosophila)sp. from Hongseong, D. (Sophophora) sp. from Mt. Sulak, D. takadai Lee and D. (Drosophila) sp. from Mt. Soyo. In the collection for this survey, the following species were captured in northern localities more than in southern localities: Microdrosophila purpurata, D. histriodes, D. bifasciata, D. unispina, D. takadai, D. kuntzei, D. testacea, D. histrio and D. tenuicauda. And the following species were found in the high altitudes more than in the low altitudes: D. histriodes, D. bifasciata, D. unispina, D. testacea, D. bizonata and D. histrio. This fact suggests that these habitats are in the high altitudes or northern localities of Korea.

The following species were captured in southern localities more than in northern localities: Microdrosophila matsudairai, Liodrosophila castanea, D. lutea and D. sternopleuralis. This fact suggests that these habitats are in southern localities of Korea. The widely distributed species in Korea are Amiota variegata, Paradrosophila pallida (previously referred to as Parascaptomyza disticha), D. busckii, D. coracina, D. suzukii, D. melanogaster, D. nipponica, D. auraria, D. angularis, D. brachynephros, D. unispina, D. nigromaculata, D. bizonata, D. immigrans, D. virilis, D. lacertosa and D. sordidula. Particularly, D. auraria was most predominant with frequency of 48 percent of the total number of 94,115, and widely distributed in Korea. Out of 65 total species collected 39 species (about 60%) have been obtained at fruit-traps, 49 species (about 75%) by sweeping, 8 species (about 12%) in garbages, 15 species (about 23%) on the tree-bloods, 6 species (about 9%) at fungi and 2 species (about 3%) around human eyes. In addition, the following 6 species such as Amiota variegata (F S T E), D. busckii (F S G T), D. coracina (F S G T), D. nigromaculata (F S G T), D. bizonata (F S G T) and D. lacertosa (F S G T) were captured by the four methods out of six different methods, which shows that those species have widely ranged feeding habits.

Khan, A. H. and T. Alderson. University of Cambridge, England. An attempt to sensitize Drosophila chromosomes to X-irradiation after 5-bromodeoxyuridine "incorporation" into DNA.

Substitution of 5-bromodeoxyuridine (BdU) for thymine in DNA has been shown to enhance the sensitivity of mammalian cells and micro-organisms to X-irradiation.

Using an aseptic and chemically-defined culture medium, the radiosensitivity (for sex-linked recessive lethal mutations) of Drosophila larval spermatogonia has been compared in the presence and in the absence of BdU. (Drosophila does not utilize pyrimidine bases unless they are supplied as the nucleoside). In order to increase the chance of BdU incorporation, larvae were cultured in the presence of the folic acid analogue, aminopterin, which inhibits thymine synthesis; this procedure limits the culturing time to 48 hours, since longer periods impose a folic acid deficiency (by aminopterin) which is not corrected in time for emergence of adult flies. (Folic acid is required for adult emergence).

Oregon-K eggs were collected and sterilized by Sang's method, and spread evenly over a sterile 3 percent agar surface. On emergence the larvae were transferred under aseptic conditions to the media (minus folic acid and RNA) at a density of 100 larvae/25 ml of medium for 48 hours, transferred onto sterile 3 percent agar for X-irradiation, and then onto a normal chemically-defined medium (plus 0.4% RNA) supplemented with additional folic acid (0.02 percent) until emergence of the adult flies. Irradiation was by a Maximar General Electric Machine at a dose of 810 r (delivered at 180 r per minute). Virgin males and females were examined for sex-linked recessive lethals by the Muller-5 (basc) method using a single brood by mating individual males to two virgin females for 3 days, and, in the case of females, by mating individual females to two males for 3 days.

The types of treatment for both male and female larvae are listed in Tables 1 and 2, where larvae are cultured in the presence of either dU (deoxyuridine) or BdU for 48 hours (with and without aminopterin), and followed by irradiation, or without irradiation. None of the sex-linked recessive lethal frequencies differ significantly from one another; there is no evidence for BdU-induced mutagenesis; and only a trivial increase in mutation following X-irradiation of males. There is no evidence for an increase in mutational radiosensitization of the X-chromosome in the presence of BdU, nor is there evidence for BdU incorporation, although the BdU + aminopterin cultures do slow down larval development compared with the dU + aminopterin cultures.

Table 1.--Sex-linked recessive lethal frequencies in Drosophila males induced by X-irradiation (810 r) after larval feeding treatments in the presence of 5-bromodeoxyuridine (BdU), or deoxyuridine (dU).

| | dU | BdU | BdU + X-irradiation | dU + X-irradiation | dU + X-irradiation | BdU + X-irradiation |
|---|---|---|---|---|---|---|
| Concentration of dU and BdU (%) | 0.02 | 0.02 | 0.02 | 0.02 | 0.02 | 0.02 |
| Concentration of Aminopterin (%) | - | - | 0.002 | - | 0.002 | 0.002 |
| Hatchability (%) | 78.3 | 73.8 | 51.1 | 65.5 | 56.3 | 61.6 |
| No. males examined | 74 | 99 | 47 | 94 | 45 | 90 |
| Average no. chromosomes examined/male | 7.8 | 6.4 | 7.9 | 7.3 | 8.2 | 9.5 |
| No. chromosomes examined | 5577 | 636 | 371 | 692 | 371 | 855 |

Table 1.--continued.

| | dU | BdU | BdU + X-irradiation | dU + X-irradiation | dU + X-irradiation | BdU + X-irradiation |
|---|---|---|---|---|---|---|
| No. lethal chromosomes | 1 | 2 | 1 | 4 | 2 | 4 |
| Lethals (%) | 0.17 | 0.31 | 0.26 | 0.57 | 0.53 | 0.46 |

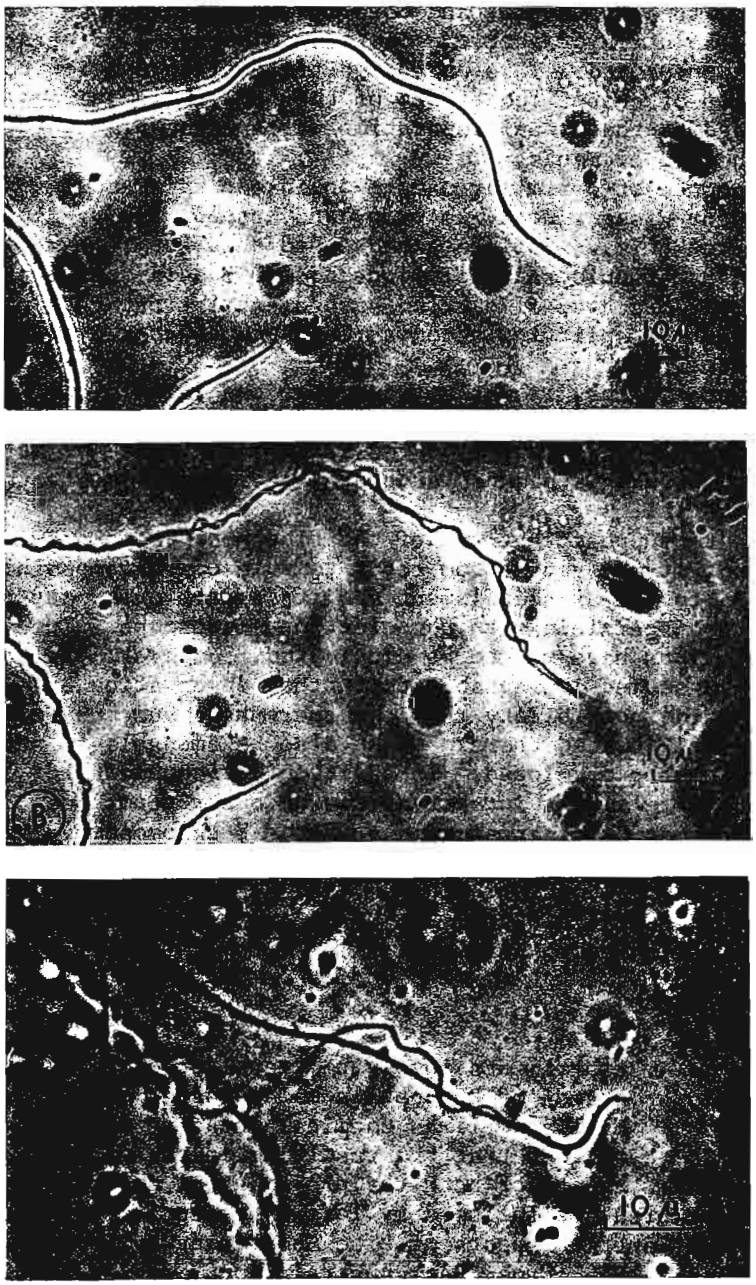
Table 2.--Sex-linked recessive frequencies in Drosophila females induced by X-irradiation (810 r) after larval feeding treatments in the presence of 5-bromodeoxyuridine (BdU) or deoxyuridine (dU).

| Treatment | dU | BdU | BdU + X-irradiation | dU + X-irradiation | dU + X-irradiation | BdU + X-irradiation |
|---|---|---|---|---|---|---|
| Concentration of dU and BdU (%) | 0.02 | 0.02 | 0.02 | 0.02 | 0.02 | 0.02 |
| Concentration of Aminopterin (%) | - | - | 0.002 | - | 0.002 | 0.002 |
| Hatchability (%) | 78.3 | 73.8 | 51.1 | 65.5 | 56.3 | 61.6 |
| No. females examined | 40 | 47 | 55 | 55 | 50 | 45 |
| Average no. chromosomes examined/female | 6.8 | 6.4 | 4 | 5.8 | 7.3 | 8.6 |
| No. chromosomes examined | 273 | 301 | 220 | 320 | 365 | 389 |
| No. lethal chromosomes | 1 | 1 | 0 | 0 | 0 | 1 |
| Lethals (%) | 0.36 | 0.33 | 0.0 | 0.0 | 0.0 | 0.25 |

Oster, I. I., J. Duffy and R. Binnard.
The Institute for Cancer Research. Observations on a piece of tail.

During the course of counting the number of spermatozoa utilized in successive matings by males of Drosophila melanogaster in connection with experiments on radiation sensitivity, we found that two structural elements could be recognized in the spermatozoon's tail following fixation. Hitherto, observations by others (Cooper, K. W., 1950, Biology of Drosophila; Yanders, A. F., and J. P. Perras, 1960, DIS; Kaplan, W. D., et al., 1962, DIS; Lefevre, G. Jr. and U. B. Jonsson, 1962, Genetics) had revealed that Drosophila has the type of sperm usually described for insects--that is, a filiform head, no separately discernible mid-piece, and a tail. In fruit flies, the only unique feature which had been noticed until now was the unusual length of the tail (0.2 mm to 6.6 mm, depending on the species, although the diameter is of the order of 0.2μ). Our experiments involved the removal with watch-maker's forceps of the vagina and uterus from a female immediately after copulation to a slide containing a drop of Drosophila Ringer's solution, teasing

open the uterus which allows the spermatozoa to flow out, permitting the sperm sample to dry slowly in air (which facilitates spreading of the sperm mass), fixing in 25% acetic acid, adding a drop of lactic-acetic orcein with fast green, and squashing with a cover slip. Observations with phase optics of material prepared in this manner revealed spermatozoa in which the tails appeared to be composed of two separable fibers (please see Figure 1).



Several different stocks of Drosophila melanogaster, including Oregon-R (wild type) as well as individuals carrying different recessive and dominant mutations, consistently showed this pattern. One fiber is spiralized, and the other appears to be fairly straight. The two fibers generally seem to be of the same thickness at the head end but the spiralized one gradually becomes thicker as it approaches and finally joins the straight fiber at the tail end of the sperm. Also, the gyres of the spiral appear to be smaller at the anterior end. The degree to which such differences in thickness and spiralization may be due to the fixation is not yet certain. In well-spread preparations the doubleness of the tail can be observed and traced along the entire length of the spermatozoon.

Since it was possible that this effect was the outcome of the method by which the material had been prepared, variations of the original technique were tried. Following the observation that air-drying per se did not have any effect, we were able to develop a fairly simple procedure which yields consistent results. In practice this consists of removing either the testis and/or the seminal vesicles from a male, transferring the organs to a drop of Drosophila Ringer's solution on a slide, gently teasing apart the organs to facilitate separation of the spermatozoa, and covering the sample with a cover-slip. The excess Ringer's solution can then be drawn off by holding a piece of filter paper at one side of the cover-slip while introducing a 25% aqueous solution of acetic acid along the opposite side. As the acid flows over the sperm, their heads become darker and distinctly visible (all observations should be made with phase

Figure 1: Drosophila melanogaster spermatozoa.
A. Live spermatozoa in Drosophila Ringer's solution.
B. The same spermatozoa following treatment with 25% acetic acid. (Photographed at 430X using phase optics).
C. Anterior portion of an acid-treated spermatozoon as seen under oil immersion. (Photographed at 970X using phase optics).
(Note that in A and B a head is located at the lower left and a tail-end can be seen at the upper right of the photographs).

optics) and one fiber appears to pull away from the other, the former becoming convoluted. Weaker solutions of acetic acid produce only a slight separation of the fibers. Twenty-five percent solutions of formic or lactic acid as well as a 1:1 solution of acetic acid and ethyl alcohol act similarly to that of aqueous acetic acid. On the other hand, a 1:3 solution of acetic acid in ethyl alcohol, hydrochloric acid, butyric acid, ammonium hydroxide, and formaldehyde have no effect on the tail. We have also found that the enzyme pronase produces a separation of the fibers of the sperm tail but with this treatment there is only a partial separation (i.e., sections of the tail seem to be affected) and both fibers appear to be non-spiralized. However, application of a 25% solution of acetic acid to such separated fibers causes one element of each pair to become convoluted. This uniform differential reaction to acetic acid indicates that the spiralization of one of the fibers of the tail following immediate acid fixation is not somehow related to the actual separation which takes place, but reveals a structural difference between the two elements.

Among the few previously reported accounts of a similar nature on insect spermatozoa were those made by Ballowitz (1890, Zeit. für Wissen. Zool. Leipzig) and Retzius (1909, Biologische Untersuchungen, Neue Folge XIV, Stockholm). Ballowitz had found that subjecting macerated samples of beetle spermatozoa to hypertonic salt solutions or osmic acid for several days revealed a number of fibers in the tail. We have also tried such drastic treatments with Drosophila, but at best only obtained a partial splitting accompanied by much cross-wise fragmentation of the tails of the spermatozoa.

After numerous observations of the preparations, it was noticed that the tails of Drosophila spermatozoa which had only been in Ringer's solution often exhibited separation along small areas of their length. In these cases neither fiber appeared spiralized. The slight degree with which this occurs makes it evident why it had not attracted the attention of other investigators who had studied non-fixed material.

At the present time it seems probable to suppose that one of the fibers represents an elongated mitochondrion. On the other hand, it may yet be too early to rule out the possibilities that either the two fibers correspond to the axial filament and its sheath or to any (or all) of the groups of bundles which are discernible with the electron microscope. The functional aspects of these structural relations have yet to be elucidated. However, the fact that a portion of the spermatozoon which had seemed impregnable to further observation by visible light has been dissociated enhances the possibility that the sperm head and the orientation and state of the chromosomal material located therein might also be amenable to such observation provided the proper techniques can be developed. In addition, it should be of interest to determine the nature of these tail elements in spermatozoa of other genotypes (particularly in those bearing two Y chromosomes or disarrangements of the Y chromosome), in spermatozoa which had been treated with various mutagens, and in spermatozoa of other species of Drosophila.

This work has been supported by grant AT(30-1)-2618 from the U. S. Atomic Energy Commission and grant CA 06927-02 from the U. S. Public Health Service).

Oster, I. I. The Institute for Cancer Research. Improved strains for detecting somatically-induced damage.

Experiments involving treatment of immature stages of Drosophila melanogaster with physical as well as chemical mutagens led us to suggest that the resulting increase in mortality of both the pre-imaginal and post-imaginal stages was largely due to chromosome breakage followed by loss of essential parts of the genome (Muller, H. J., 1958, Conf. on the Genetic Aspects of Life Shortening by Radiation, Dec. 13 and 14, 1958, Ames, Iowa; Oster, I. I., 1958, Proc. Sec. Austral. Conf. on Rad. Biol.; Oster, I. I., 1959, Science; Oster, I. I., 1960, Science; Oster, I. I., 1960, in Proc. of Conf. on Research on the Radiotherapy of Cancer; Oster, I. I., 1961, The Sec. Int. Conf. of Human Genetics; Oster, I. I. and A. Cicak, 1958, DIS). Several genetical schemes, involving comparisons between males and females, rod-X- and ring-X-bearing males, and normal rod-X-carrying males and attached-X females, were utilized to obtain evidence for this view. Soon thereafter, additional confirmatory data was obtained by Ostertag and Muller (1959, Science), who also extended the work to include comparisons of normal individuals with those heterozygous for small deficiencies (Ostertag, 1963, Zeit. für Vererbungsl.).

Based on our previous results we had developed a standard technique for determining whether any particular agent is capable of causing life-shortening via a genetic mechanism. This had consisted of comparing the mortality rates between two strains of males, one carrying a normal rod-X chromosome and the other a ring-X chromosome, which had been similarly treated during one of the pre-imaginal stages. Although this method proved to be very reliable, we thought that it would be useful to devise an even more sensitive one.

This can now be done by combining a ring-X chromosome with two autosomal deficiencies in the following manner (phenotypes are shown in parenthesis):

$P_1$ $Y/X^{c2}$, y B; $e^{11}$ ♂♂ (y B e) x

Cy,InsO T(2;3)CyD#2 D InsCXF e

$sc^8$·Y/y f:=; ——————ooooo ooooooooooo——— ♀♀ ($y^+$ f Cy D $vg^{-D}$ Ly)

$vg^{-D}$ ooooooooooooooo Ly

$F_1$: Select non-yellow, non-ebony ($y^+$ $e^+$) larvae for treatment. Third instar larvae should be used since ebony (e) cannot be recognized in the younger stages. These will be males of the composition: $sc^8$·Y/$X^{c2}$, y B; ——— ooooooooo which can be recognized because

$vg^{-D}$ Ly

their mouth parts are black ($y^+$) and their spiracle sheaths are normal colored ($e^+$). On the other hand, their sisters will have brown mouth parts and their brothers which do not carry the deficiencies will have darkly-colored spiracle sheaths. Re-combinants with only one of the deficiencies will die during early embryogenesis because of aneuploidy.

Males for the $P_1$ cross can be maintained with attached-X females carrying a normal Y chromosome while $P_1$ females can be kept as a balanced stock with males having the same autosomal constitution. Larvae for comparison can be obtained by crossing males carrying a normal rod-X with females which do not contain the two deficiencies. Otherwise these stocks should carry the same markers in order to avoid the introduction of undesirable viability differences. The translocation, T(2;3)CyD#2, between a second chromosome containing $dp^{txI}$ Cy,InsO pr $cn^2$ and a third chromosome containing ru h D InsCXF e was produced especially for this scheme.

The $F_1$ resulting from this cross offers the advantage of being more sensitive to the induction of somatic damage than individuals containing either only a heterozygous deficiency or a ring-X chromosome. As in the previous methods which we had developed for utilizing the larval stages the present one allows for a more accurate measure of the numbers being actually treated than those involving flies which are not marked suitably. By introducing those few characteristics which can be detected in the immature stages, it is possible to select for treatment and observation only those individuals which possess the desired genotype. This also permits us to study the pattern of damage which may ensue following a particular treatment. In addition, the use of heterozygotes reduces the inviability which may arise due to homozygosis, thereby increasing the background mortality to undesirable (and often unpredictable) levels.

Preliminary tests, using low doses of X rays and gamma rays, have confirmed our expectations for the proposed scheme.

This work has been supported by grant (AT(30-1)-2618) from the U. S. Atomic Energy Commission, grant G 14148 from the U. S. National Science Foundation, and grant CA 06927-02 from the U. S. Public Health Service.

Oshima, C. and T. Watanabe. National Institute of Genetics, Misima, Japan.

The effect of insecticide selection on experimental populations of Drosophila pseudoobscura.

About forty homozygous strains of four kinds of chromosomes, ST, AR, CH and PP, were used in the experiments. These strains, originated from the Mather population in California, had been established by Dobzhansky in 1963.

Heterozygous female flies for ST and AR were crossed with heterozygous male flies for CH and PP. Two initial populations were made with 250 female and 250 male offspring and after two generations (one generation period lasts about 20 days), the salivary gland chromosomes of 150 larvae hatched from sampled eggs were observed for detecting the frequency of each chromosome.

In the $F_2$ generation of both populations, the frequencies of the four kinds of chromosomes should be theoretically equal, i.e. 25 per cent of each. However, the observed frequencies were ST:33.3, AR:26.3, CH:22.7, PP:17.7 per cent in Population I and ST:26.3, AR:28.0, CH:26.3, PP:19.3 per cent in Population II. Further, each population was divided into two populations A and B. Flies in Population A were exposed to insecticide test paper (DDT 1%, 2% or Dieldrin 0.1%) for one hour and transferred into a new cage in each new generation. Flies in Population B were transferred into a new cage without exposure. The test papers and test kits were prepared by WHO in Geneva and sent to us. These populations are being maintained in a constant temperature room (25°C) and the varying frequencies of chromosomes from $F_2$ to $F_{12}$ in each population were observed as shown in Table 1.

Table 1. Changing frequencies of four kinds of chromosomes in DDT or Dieldrin selected and non-selected populations

| | $F_2$ | | | $F_3$ | $F_4$ | $F_5$ | $F_7$ | $F_9$ | $F_{12}$ |
|---|---|---|---|---|---|---|---|---|---|
| Pop. I | ST 33.3<br>AR 26.3<br>CH 22.7<br>PP 17.7 | IA (DDT selected) | ST | 40.3 | 55.0 | 55.3 | 73.3 | 77.0 | 92.7 |
| | | | AR | 18.3 | 23.0 | 24.3 | 15.7 | 18.7 | 6.3 |
| | | | CH | 22.0 | 11.7 | 8.3 | 4.0 | 0.7 | 0.0 |
| | | | PP | 19.3 | 10.3 | 12.0 | 7.0 | 3.7 | 1.0 |
| | | IB (non-selected) | ST | 42.0 | 45.7 | 53.7 | 65.3 | 72.3 | 67.0 |
| | | | AR | 24.7 | 25.7 | 26.3 | 22.0 | 17.0 | 27.0 |
| | | | CH | 18.3 | 17.3 | 10.0 | 7.0 | 6.3 | 4.7 |
| | | | PP | 15.0 | 11.3 | 10.0 | 5.7 | 4.3 | 1.3 |
| Pop. II | ST 26.3<br>AR 28.0<br>CH 26.3<br>PP 19.3 | IIA (DL. selected) | ST | 43.7 | 41.0 | 43.0 | 71.0 | 84.3 | 90.3 |
| | | | AR | 19.7 | 30.0 | 29.7 | 16.7 | 5.7 | 2.0 |
| | | | CH | 15.3 | 9.7 | 10.3 | 4.3 | 6.0 | 4.7 |
| | | | PP | 21.3 | 19.3 | 17.0 | 8.0 | 4.0 | 3.0 |
| | | IIB (non-selected) | ST | 36.3 | 45.7 | 49.3 | 52.3 | 64.3 | 68.7 |
| | | | AR | 21.7 | 29.7 | 27.3 | 30.0 | 25.3 | 26.3 |
| | | | CH | 23.3 | 14.7 | 13.3 | 12.0 | 5.3 | 3.0 |
| | | | PP | 18.7 | 10.0 | 10.0 | 5.7 | 5.0 | 2.0 |

The frequency of ST chromosomes has increased in both selected and non-selected populations, but after $F_7$ generation the increase in the former (IA, IIA) was greater than in the latter (IB, IIB). On the other hand, AR chromosomes showed changes which were opposite to those of ST, especially in the selected populations. The frequencies of both CH and PP chromosomes have gradually decreased. However, their relative frequencies in selected populations were slightly different from those in non-selected populations: PP became more frequent than CH after several treatments, while the CH frequency was found always a little higher than those of PP in non-selected populations.

These results seem to suggest that the remarkable evolutionary changes in natural populations of D. pseudoobscura in California during about twenty years might be due to insecticide selection: ST chromosomes increased and AR chromosomes underwent changes which were the reverse of those in ST. CH chromosomes decreased and became rare and PP chromosomes emerged spectacularly.

This work was supported by a grant from the Japan Society for the Promotion of Science as part of the Japan - U.S. Cooperative Science Program, and when Prof. Th. Dobzhansky came to Japan in September 1964, the experiment was commenced with his advice.

MacIntyre, R. J. and T. R. F. Wright. The Johns Hopkins University. Recombination in FM4/+; SM1/+; $Ubx^{130}$/+ Heterozygotes.

In investigations on the selective value of different Esterase 6 alleles in experimental populations, an attempt was made to construct two stocks which were coisogenic except at the Est 6 locus and an unknown number of genes between hairy (26.0) on the left and thread (43.2) on the right. The balancers employed in the construction of these stocks were: for the X chromosome, FM4 (Mislove and Lewis, DIS 28:77); for Chromosome 2, SM1 (Lewis and Mislove, DIS 27:58); and for Chromosome 3, $Ubx^{130}$ (Lewis, PNAS, U.S. 38:953-961, 1952). In order to detect recombination within the three major chromosomes when they were simultaneously balanced over crossover suppressors, three testcrosses were set up. In the first, FM4/ sc ec cv $ct^6$ v $g^2$ f; SM1/+; $Ubx^{130}$/+ females were mated to sc ec cv $ct^6$ v $g^2$ f males, and their progeny were examined for recombinations between the X Chromosome markers. Likewise, offspring from two other matings, FM4/+; SM1/ al dp b pr cn c px sp; $Ubx^{130}$/+ females x al dp b pr cn c px sp males and FM4/+; SM1/+; $Ubx^{130}$/ ru h th st cu sr $e^s$ Pr ca females x ru h th st cu sr $e^s$ + ca males were checked for crossing over on Chromosomes 2 and 3 respectively.

In the testcross involving the Chromosome 2 markers, recombinations between al and dp, and between px and sp could not be detected since the SM1 chromosome contains mutant alleles at the al and sp loci. Of 507 chromosomes examined, none showed any recombinations of the markers between dp and px. Apparently, crossover suppression of Chromosome 2 by the SM1 balancer is complete, even when the two other major chromosomes are each heterozygous for several inversions.

Examination of Tables 1 and 2 reveals that this is not true of the FM4 and $Ubx^{130}$ balanced chromosomes. Table 1 indicates that about one out of every four X Chromosomes from FM4/+; SM1/+; $Ubx^{130}$/+ females will be a recombinant. If one compares the percentage of crossing-over with the approximate size of each block (as estimated from reference maps of the salivary gland X Chromosomes and the genetic distances between the markers involved), it can be seen that the recombinational events producing viable chromosomes are distributed fairly randomly both between and within the blocks delineated by the breakpoints on the FM4 chromosome. This is not the case in Chromosome 3 (Table 2). Here, almost all the recombinants result from either single crossovers near the end of the right arm or from multiple crossovers, nearly always including a double crossover in block II.

It is easy to see that when the "coisogenic" lines are initiated with 10 to 20 wild-type segregants from the last cross in mating schemes involving the use of the multiply rearranged balancer chromosomes, FM4, SM1, and $Ubx^{130}$, there is a very good chance that cryptic heterozygosity is introduced into the derived stocks. Uncontrolled regions on the genetic map, initially thought to be restricted to Chromosome 3 between hairy and thread, actually included the whole X Chromosome and in Chromosome 3, from the tip of the left arm to the vicinity of the thread locus and the distal one-fifth of the right arm. Furthermore, analysis of the recombinant progeny of the testcross involving markers on Chromosome 3 revealed that of the double-crossovers within block II, slightly over half included the Est 6 locus. Thus, since an Est 6 allele from the $Ubx^{130}$ chromosome may have been introduced into one of the coisogenic stocks, still another possible variable was detected and subsequently had to be taken into consideration when the results of the experimental populations founded by the "coisogenic" stocks were analyzed (see MacIntyre and Wright, Amer. Nat., In press).

Research supported by NSF Grant GB-1822.

Table 1.--Phenotypes of Recombinants

| Block | Region | N | sc + + + + + + | sc ec + + + + + | sc ec cv + + + + | sc ec cv ct + + + | sc ec cv ct v + + | sc ec cv ct v g + | sc + + ct v g f | sc + + + v g f | sc + + + g f | sc + + + + f | sc ec + ct v g f | sc ec + + v g f | sc ec + + + + f | sc ec cv + v g f | sc ec cv + + g f | sc ec cv + + + f | sc ec cv ct + g f | sc ec cv ct + + f | sc ec cv ct v + f | sc + + ct v + + | sc + cv ct + + + | sc + cv ct v + + | sc ec + + v g + | sc ec cv + v + + | sc ec cv + v g + | Total Crossovers by Region |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | 14 | 15 | 13 | 22 | 5 | 8 | 4 | 2 | 1 | 3 | 5 | 3 | 3 | 5 | 3 | 5 | 5 | 10 | 3 | 1 | 1 | 1 | 1 | 1 | 1 | |
| I | sc - 3C | | X | X | | | | | X | X | X | X | | | (X) | | | | | | | X | X | X | | | | 42 |
| II | 3C - ec | | | X | | | | | (X) | (X) | | | | | (X) | | | | | | | | | | | | | 15 |
| | ec - 4EF | | | X | | | | | (X) | (X) | | | | | (X) | | | | | | | | | | | | | 15 |
| III | 4EF - cv | | | (X) | | | | | (X) | (X) | | | X | X | X | | | | | | | | X | X | X | | | 14 |
| | cv - ct | | | | X | | | | X(X) | | | | X | | | X | X | X | | | | X | | | | X | X | 38 |
| | ct - v | | | | | X | | | | X(X) | | | | X | | X | | | X | X | | | X | | X | X | X | 51 |
| | v - 11F | | | (X) | X | X | | | X | X | | | | | X | | X | X | X | X | | X | | X | | | | 69 |
| IV | 11F - g | | | (X) | X | X | X | | X | X | X | | | | X | | | X | | X | X | | | | | X | | 69 |
| | g - f | | | | | | | X | | | | X | | | X(X) | | | X | | X | X | | | | X | | X | 34 |

Summarized data:

| | | | |
|---|---|---|---|
| Number of flies counted: | 541 | Number of recombinant flies: | 135 |
| non crossovers : | 406(75%) | least number of crossovers : | 347 |
| single crossovers : | 27( 5%) | per cent in Block I : | 12% |
| double crossovers : | 28( 5%) | II : | 9% |
| triple crossovers : | 56(10%) | III : | 50% |
| quadruple crossovers: | 24( 4%) | IV : | 30% |

Table 1: Results of the testcross involving X Chromosome markers. "Blocks" refer to sections of the X Chromosome delineated by the breakpoints on the FM4 chromosome (Mislove and Lewis, DIS 28:77). "Regions" refer to sectors within each "block" in which crossovers can occur that result in phenotypically detectable recombinant chromosomes. An "X" in the table denotes a region in which a crossover would have to occur in order to produce a viable chromosome with the indicated phenotype. "(X)" denotes a crossover in another possible, but less probable, series of recombinations to give the observed phenotype, also without involving deficiencies or duplications. The total crossovers per region were calculated omitting the "(X)" series from consideration. N = number of flies. Percentagesgiven in the summarized data are rounded off to the nearest whole number.

Table 2.--Phenotypes of Recombinants

| | | | ru + + + + + + | ru h + + + + + | ru h th st sr + + | ru h th st sr Pr + | ru + th st sr Pr ca | ru h + + + Pr ca | ru + th st sr Pr + | ru + th st sr + + | ru + + st sr Pr + | ru + + + Pr + | Total Crossovers by region |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Block | Region | N | 1 | 2 | 9 | 112 | 55 | 1 | 6 | 3 | 1 | 1 | |
| I | ru - 61AC | | X | X | | | | X | | | | X | 5 |
| II | 61AC - h | | | X | | | X | X | X | X | X | | 68 |
| | h - th | | | X | | | X | X(X) | X | X | | | 67 |
| | th - st | | | | | | | | | | X | | 1 |
| | st - 74 | | | | | | | (X) | | | | | |
| III | 74 - 89DE | | | | | | | | | | | | |
| IV | 89DE - sr | | | | | | | (X) | | | | | |
| | sr - 93B | | | | | | | (X) | | | | | |
| V | 93B - 96A | | | | | | | | | | | | |
| VI | 96A - Pr | | | | X | | | X | | X | | X | 14 |
| | Pr - ca | | | | | X | | | X | | X | X | 120 |

Summarized data:

| | | | | |
|---|---|---|---|---|
| Number of flies counted: | 769 | | Number of Recombinant flies: | 191 |
| non crossovers : | 578 | (75%) | least number of crossovers: | 275 |
| single crossovers : | 122 | (16%) | per cent in Block I : | 2% |
| double crossovers : | 55 | ( 7%) | II : | 50% |
| triple crossovers : | 13 | ( 2%) | VI : | 49% |
| quadruple crossovers : | 1 | - | | |

Table 2: Results of the testcross involving chromosome 3 markers. See Table 1 legend for notations. The "blocks" are delineated on the salivary gland map by the breakpoints on the $Ubx^{130}$ chromosome given in Lewis (PNAS U.S. 38:953-961, 1952).

Lee, Taek Jun. Chungang University, Seoul, Korea. Variations in sex-ratio of wild D. suzukii.

The collections of Drosophilid flies were made at various localities of Korea during a period from 1956 to 1965. D. suzukii was found to be widespread in Korea. A total of 3912 specimens of D. suzukii were captured in seventeen natural populations. The sex-ratio of males was higher than females in most of the collecting localities. The percentage of males in the entire sample of D. suzukii was 67.38. However, a conspicuously higher percentage (82.03%) of females was observed in 295 samples from Muju (Kucheondong) in August of 1962. The sex-ratio of D. suzukii varied with season and altitude. The data showed the percentage of females to increase in the fall. The males were abundant (87.6%) in the low altitude sites, while the females were abundant in the high altitude sites. At the high altitude sites the sex-ratio was approximately 1:1 ratio, based on collecting data from Mt. Kyelyong (827 m. high) in August of 1957. The same fact, abundance of females at high altitude sites, was also observed the next year (August, 1958) when the collections were made again at Mt. Kyelyong. This suggests that in summer the high altitude, lower temperature sites are more favorable for the deposition of eggs by the females of D. suzukii.

Iyengar, Shanta V. Louisiana State University. A reciprocal translocation involving the $Y^{c}$:bw chromosome and a second chromosome of D. melanogaster.

Among the IVth brood progeny of a 15-day old irradiated $Y^{c}$:bw D. melanogaster male and virgin females of the following genotype: y v;S Sp cn bw/Cy cn bw, a "white" eyed exceptional male with yellow body and Curly wings was found, the expected males being yellow vermilion and either S Sp or Cy. The exceptional males could result either from loss of the paternal X chromosome or the entire $Y^{c}$:bw or just the bw segment on it. On being mated to bw ♀♀ this male proved to be fertile and produced in repeated tests only Cy ♀♀ and Cy ♂♂ but no Cy ♀♀ or Cy ♂♂. In the $F_2$ generation Cy and Cy ♀♀ and ♂♂ were produced. The results from this and other genetic tests indicate a reciprocal translocation involving the ring Y and a second chromosome of the treated male presumably in a spermatid or possibly in an earlier germ cell. The breaks that affected the translocation were also responsible for the possible deletion of the bw segment of the $Y^{c}$:bw chromosome or mutation of the bw to the recessive condition. (This work was supported by a research grant from the Greater Baton Rouge and New Orleans Cancer Association.)

Seki, T., Y. Fukushi and H. Kikkawa. Osaka University, Japan. A close relationship between the color of puparium and β-alanine in some species of insects.

It has been found that black puparium mutants of insects such as Bombyx, Drosophila and Musca lack β-alanine in their puparium sheaths (Seki, DIS 36:115, 1962; Fukushi and Seki, Jap. J. Genet. 40:203-208, 1965).

Recently, Fukushi found that the black puparium mutant of Musca becomes brownish when β-alanine is mixed in the larval food in a concentration of about 0.4 M. Such induced brownish (normal type) puparia contain β-alanine, and the content increases as the color of puparium comes near the normal type.

However, an attempt using the ebony mutant of D. virilis gave a negative result (Kikkawa). This may be due to the reason that the ebony mutant of D. virilis seems to be very stable in its expression as compared with that of the black puparium mutant of Musca.

Leahy, Sister Mary Gerald C.S.J. Mount St. Mary's College. Egg deposition in D. melanogaster increased by transplant of male paragonia.

Mated females of D. melanogaster (Canton-S) have a higher oviposition rate than virgins. Both the sperm and paragonial fluid which the female receives at mating are possible sources of a stimulant for oviposition. The relative effect of the two materials was therefore in question. The answer was determined by transplant of each substance into the thorax of two day old virgin females.

The glands and testes were removed into insect saline. A virgin female was etherized, an incision made into the pleural region of the thorax and a single gland inserted. Sections of testis, approximate in size to the gland were transplanted into other virgins. The paragonia and testes were obtained from males which were three days old and had been separated from females for two days. After the operation the flies were placed in individual containers which had media with lamp black. Egg count was made four days after transplant.

A striking rise in egg deposition followed transplant of paragonia. Figure 1 is a summary of five experiments which gave significant evidence that these glands provide an

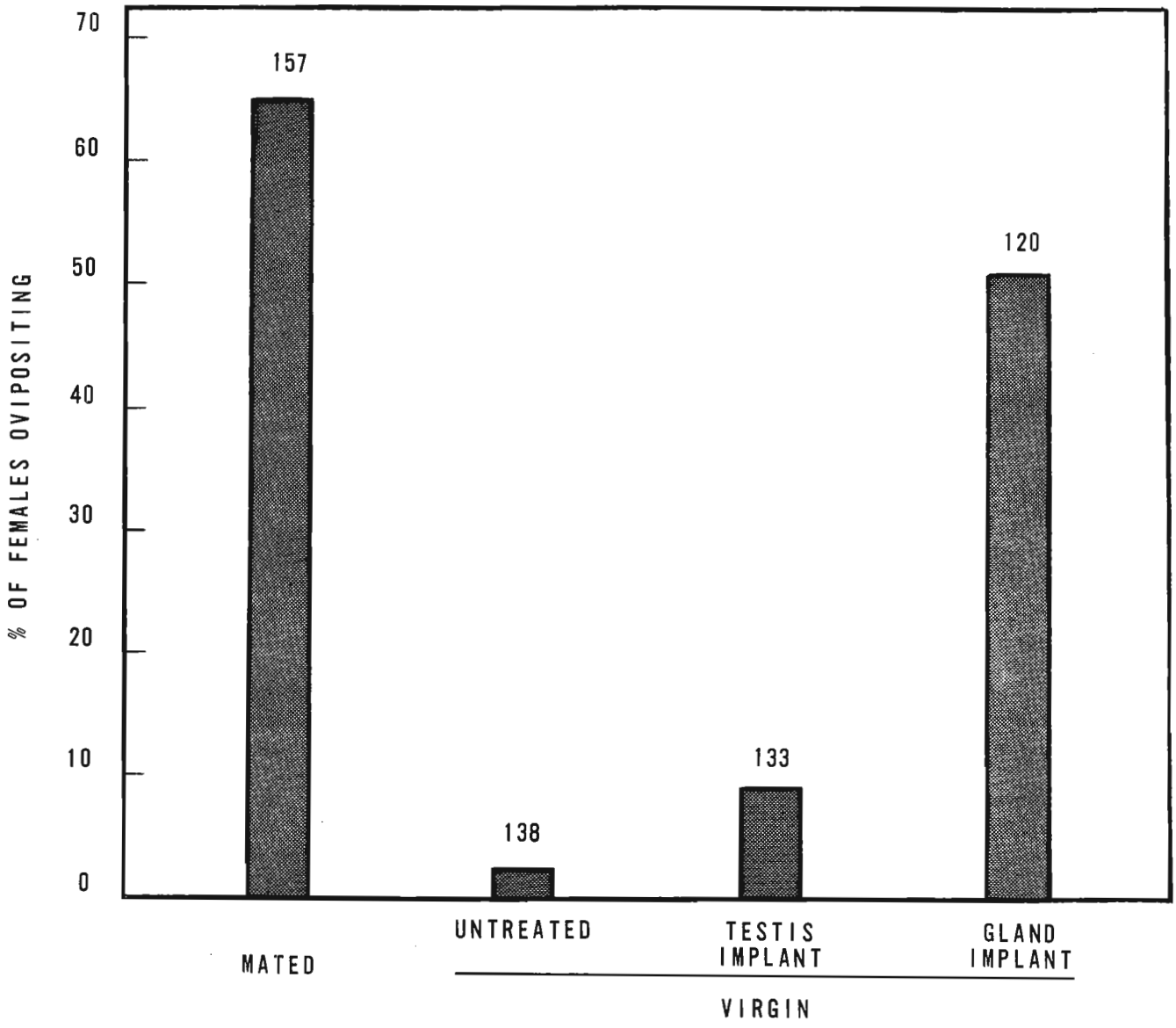



Fig. 1.— Oviposition in females of *Drosophila melanogaster*.

oviposition stimulant received by the female at mating. An average of 11% of the virgin females oviposited when testes were transplanted, whereas an average of 52% oviposited after paragonia transplant.

Non-specific activity of the paragonial substance is indicated in our current investigation of other Diptera. Chemical study of paragonia extract is in progress.

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## The mutagenicity of phenyl-N-lost-derivatives.

A number of bifunctional chloroethyleneimines with different basal constitution have been tested on their mutagenic activity on the Berlin wild stock of Drosophila melanogaster by means of the Basc method in successive three-day brood periods. The drugs were applied orally.

The test substances were of the type R-  -N $\begin{matrix}CH_2CH_2Cl\\CH_2CH_2Cl\end{matrix}$, R being the symbol for different substituents.

The mutagenic activity of the compounds tested declined in the following order of substituents (R): $NH_2 > H \gg OCH_3 \tilde{} CHO > Cl$ = control. The differences in mutation rates were in part restricted to single broods. There was no strict correlation between chemical reactivity and mutagenicity (table 1). The mutation rates increased with prolongation of the feeding period. The mutagenic action of C.B. 1077 (R = CHO; very low reactive) demonstrates that chemical reactivity tests alone are not suitable for predicting whether an alkylating drug is mutagenic or not. The most toxic compound was C.B. 1074, a substance with only a low reactivity. The most effective sterilizing and mutagenic compound was C.B. 1128. The mutagenic action of C.B. 1128 could be depressed by administering l-cysteine simultaneously.

Table 1. The mutagenicity of p-N-bis-(β-chloroethyl)-anilines of the type R-  -N $\begin{matrix}CH_2CH_2Cl\\CH_2CH_2Cl\end{matrix}$

| SUBSTITUENT | n lethals / n test chromosomes = % lethals brood I | II | III | Totals | reactivity |
|---|---|---|---|---|---|
| | a) dose: $10^{-2}$ M; feeding period: 3 days | | | | |
| R=$NH_2$ (C.B. 1128) | t e s t - ♂ ♂ | s t e r i l e | | | very high |
| R=H (C.B. 1074) | a b s o l u t e l y | t o x i c | | | low |
| R=$OCH_3$ (C.B. 1045) | 8/475=1.7% | 3/585=0.51% | 7/703=1.0% | 18/1763=1.0% | high |
| R=CHO (C.B. 1077) | 4/426=0.94% | 3/242=1.2% | 3/328=0.91% | 10/ 998=1.0% | very low |
| R=Cl (C.B. 1053) | -/192=0.00% | -/156=0.00% | 1/259=0.39% | 1/ 607=0.16% | very low |
| | b) dose: $10^{-3}$ M; feeding period: 3 days | | | | |
| R=$NH_2$ (C.B. 1128) | 35/943=3.7% | 18/816=2.2% | 9/791=1.1% | 62/2550=2.4% | very high |
| R=H (C.B. 1074) | a b s o l u t e l y | t o x i c | | | low |
| | c) dose: $10^{-2}$ M; feeding period: 1 day | | | | |
| R=$NH_2$ (C.B. 1128) | 12/361=3.3% | 1/306=0.33% | 5/269=1.9% | 18/ 936=1.9% | very high |
| R=H (C.B. 1074) | 8/724=1.1% | 6/727=0.83% | 5/441=1.1% | 19/1892=1.0% | low |
| R=$OCH_3$ (C.B. 1045) | 3/350=0.86% | -/369=0.00% | -/333=0.00% | 3/1052=0.29% | high |
| R=CHO (C.B. 1077) | 4/756=0.53% | 1/651=0.15% | 1/575=0.35% | 7/1982=0.36% | very low |
| R=Cl (C.B. 1053) | 1/690=0.14% | -/481=0.00% | -/343=0.00% | 1/1511=0.07% | very low |
| | Control 0.10 ± 0.03% | | | | |

DeMarinis, F. Cleveland State University.

A comparative effect of the nucleotide bases on the development of the Bar eye ♂♂.

Amides tend to increase the number of facets in the Bar eye Drosophila. Kaji and his co-workers (Annot. Zool., Japan 29:23, 1956) concluded that it was the amide group $CONH_2$, that was responsible for the increase in facets. Abd-El-Wahab (Jour. Gen. 56:288, 1959) showed that other eye size mutants are also affected by amides, and concluded that amides are not strictly Bar modifiers but enter into the general process of facet formation. DeMarinis and Sheibley (Abs. Genetics 45:66, 1960) found that the amide link, -CONH-, when incorporated in cyclic structures, is less effective that it was in open chain compounds; and then (Abs. Proc. XVI Inter. Cong. Zool. 2:203, 1963) showed that double amides of the type $NH_2CO(CH_2)_nCONH_2$ are more effective in increasing the number of facets in Bar.

In this report a further study has been made using nucleotide bases, mainly uracil, cytosine, thymine guanine, and adenine.

In brief, each compound was added to Pearl's standard formula in proportionate amounts and Bar stock eggs were deposited and permitted to complete development on it. The effect on eye size was determined by counting the number of facets in the eye of the males. All tests were carried out at 25°C. The results obtained are listed in the tables and graph below.

Uracil and cytosine showed the greatest effect on the eye. Over 90% of the flies, completing their development on a mixture of 2% uracil, emerged as phenotypically wild-type eye (over 600 facets). However, at this concentration of uracil very few flies emerged. On the other hand, cytosine showed much less toxicity, and at 2% concentration approximately 90% emerged as wild-type eye.

Adenine and thymine have a relatively less effect in increasing the number of facets in the Bar ♂♂, adenine reaching a peak at 0.5% concentration with an average number of approximately 280 facets (control, Bar ♂♂ average 88 facets) and thymine at 0.25% with an approximate average of 104 facets. These compounds seem to interfere with many other basic physiological processes of development besides the modifying action on the development of the Bar eye.

1% Guanine shows no particular effect on the eye-size, nor on the number of flies that emerge. At this concentration it appeared quite inert and therefore no further tests were made with it.

Tables 1, 2, 3, 4: Relationship Between Concentration of Nucleotide Base and Eye Size in Bar ♂♂

Table 1

| % Cytosine | Bar ♂♂ size of eye, number of facets |
|---|---|
| 4.50 | 565 ± 12.1 |
| 3.00 | 500 ± ? |
| 2.00 | 484 ± 12.2 |
| 1.75 | 442 ± 18.1 |
| 1.50 | 301 ± 17.5 |
| 1.25 | 219 ± 13.1 |
| 1.00 | 286 ± 13.3 |
| Control | 88 ± 3.9 |

Table 2

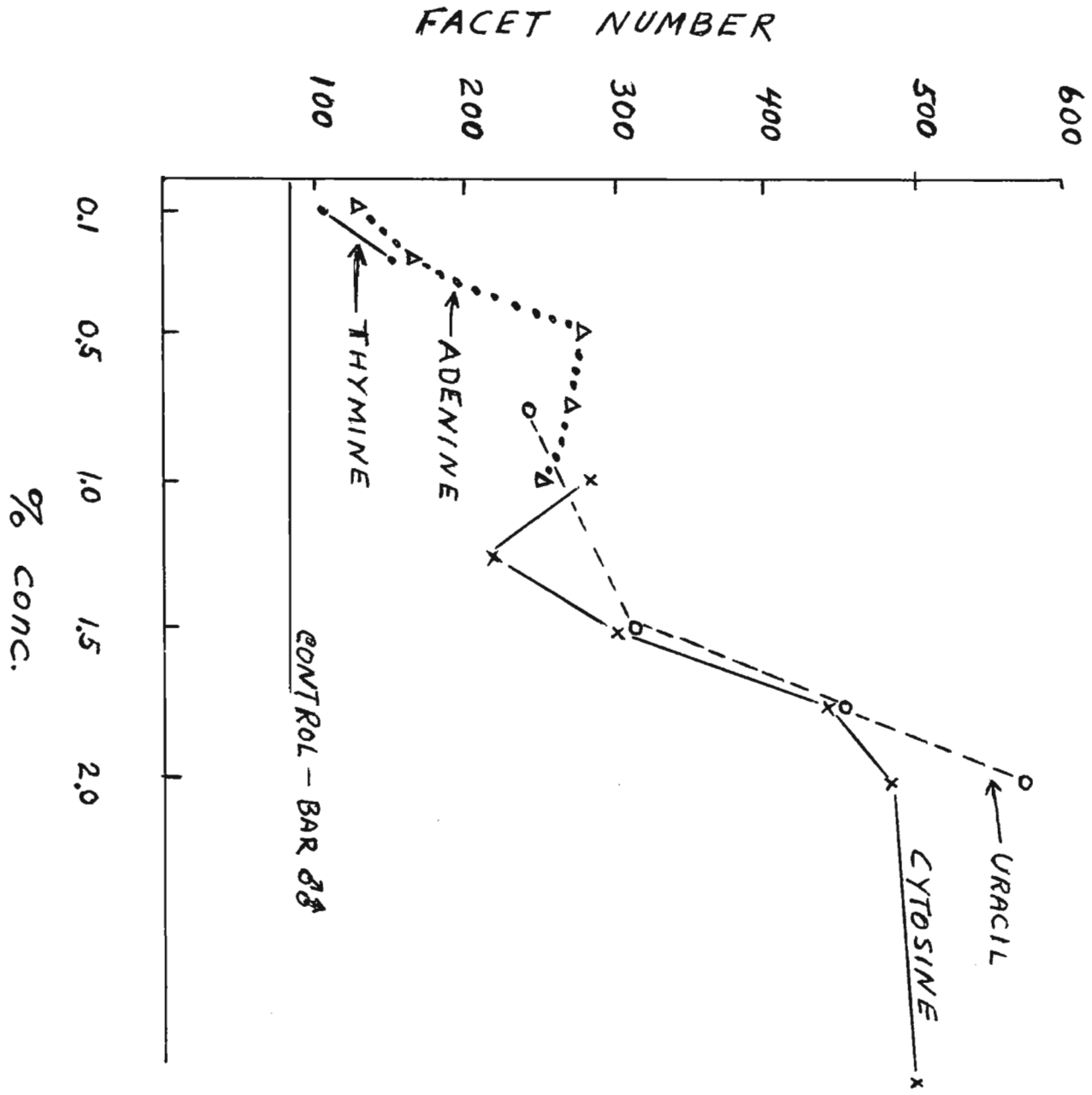
| % Thymine | Bar ♂♂ size of eye, number of facets |
|---|---|
| 0.25 | 155 ± 10.1 |
| 0.10 | 104 ± 3.7 |
| Control | 88 ± 3.9 |

Table 3

| % Adenine | Bar ♂♂ size of eye, number of facets |
|---|---|
| 1.00 | 254 ± ? |
| 0.75 | 273 ± 19.9 |
| 0.50 | 281 ± 14.8 |
| 0.25 | 164 ± 10.6 |
| 0.10 | 130 ± 5.2 |
| Control | 88 ± 3.9 |

Table 4

| % Uracil | Bar ♂♂ size of eye, number of facets |
|---|---|
| 2.0 | 575 ± 17.9 |
| 1.75 | 450 ± 13.8 |
| 1.50 | 310 ± 15.0 |
| 0.75 | 244 ± 18.1 |
| Control | 88 ± 3.9 |



DeMarinis, F. Cleveland State University.
A further survey of amides and their effect on the development of Bar eye.

It has been shown from the work of Kaji, DeMarinis, Luce, and Abd-El-Wahab that amides in general, when mixed with standard Pearl's formula and fed to Bar larvae of Drosophila increase the number of facets in the eye. Some of these amides have a slight but significant effect, others act more markedly, changing a genetically Bar eye (88 ± 3.9 facets) to a phenotypic wild-type eye (over 600 facets).

In attempting to find the key compound or compounds which has the maximum effect with the least toxic reaction, a number of pure compounds were investigated. Each compound was mixed in proportionate amounts in Pearl's standard formula. Approximately 400 eggs of the Bar stock were used in testing each mixture. Each batch of eggs was permitted to complete development on each experimental mixture. The ratio between the number of adults that emerged and the number of eggs started served as an index in estimating the toxic and the optimum concentration for each compound. The toxic level was arbitrarily taken as when only a few or no adults emerged; the optimum concentration was arbitrarily set when approximately 50% of the eggs emerged as adults. In the experience of the author this level of recovery has been found most productive in carrying out more detailed experiments with some of these compounds.

As a result of these preliminary tests a table of toxic concentration and optimum concentrations for each compound tested has been established. In it are also indications whether they have an effect of increasing the number of facets in the eye or not. These are shown by plus or minus signs in the third column.

Toxic Concentration Table

| Compound | Toxic Conc. (%) | Optimum Conc. (%) | Facet Increase in Bar ♂♂ |
|---|---|---|---|
| Acetamide | 1.25 | 0.75 | + |
| Iodoacetamide | 0.50 | highly toxic | ? |
| n-Propionamide | 1.00 | 0.75 | + |
| n-Butyramide | 1.00 | 0.75 | + |
| n-Valeramide | ? | 2.00 | - |
| n-Hexanamide | ? | 2.00 | - |
| Oxamide | ? | 2.00 | - |
| Malonamide | 4.00 | 2.00 | + |
| Succinamide | ? | 2.00 | + |
| Glutaramide | 4.50 | 2.50 | + |
| Adipamide | non-toxic | 2.00 | + |
| Uracil | 1.00 | 0.75 | + |
| 5-methyluracil (thymine) | 0.40 | 0.20 | + |
| 6-methyluracil | 1.00 | 0.50 | - |
| 5-bromouracil | 1.00 | 0.25 | - |
| 5-nitrouracil | ? | 2.25 | - |
| 5-Aminouracil | 0.10 | highly toxic | ? |
| 6-Aminouracil | ---- | 1.00 | - |
| Adenine | 1.00 | 0.50 | + |
| Guanine | ---- | 1.00 | - |
| Cytosine | 2.50 | 1.75 | + |
| 5-methyl cytosine (HCL) | 1.25 | 0.75 | - |
| Urea | 2.75 | 2.00 | + |
| Methylurea | 0.60 | 0.35 | + |
| Ethylurea | 0.50 | 0.25 | + |
| Biurea | ? | 2.00 | - |
| 1,3-dimethylurea | 0.10 | highly toxic | ? |
| N,N'acetylmethylurea | 0.25 | highly toxic | ? |
| Biuret | 0.35 | 0.15 | - |
| Acetylurea | 0.75 | 0.35 | + |
| Allylurea | 0.50 | 0.25 | - |

| | | | |
|---|---|---|---|
| Hydantoin | 1.25 | 0.75 | + |
| 1-Methylhydantoin | 2.00 | 1.00 | + |
| Allantoin | ? | 2.00 | - |
| Uric Acid | ? | 2.00 | - |
| N-methylformamide | 0.50 | 0.35 | + |
| N,N'dimethylacetamide | 0.25 | highly toxic | ? |

Sobels, F. H. State University, Leiden, Netherlands. Oxygen dependent differences in radiosensitivity between fully mature and almost mature spermatozoa.

Experiments by Lefevre and Jonsson (1964, Mut. Res. 1:231-246) showed that after X-irradiation of 3-day-old Drosophila males the mutation frequency decreases from the first to the third mating. Similar differences in radiosensitivity, though slightly less pronounced, were observed between sperm obtained from the first mating of 7-day-old males and that from 1.5-hour-old males. A number of experiments with X-irradiation in $O_2$, air or $N_2$ were carried out to investigate whether these differences in radiosensitivity between fully mature, motile spermatozoa and the immotile, late spermatids (in Lefevre's terminology) are associated with differences in oxygenation. The most radiosensitive kind of sperm was sampled by using the first ejaculate from 7-day-old males. Sperm with lowest sensitivity was obtained from the first ejaculate of 1-hour-old males. After radiation exposures in $O_2$ and $N_2$, post-treatments with $N_2$ or $O_2$ were given, after irradiation in air with $N_2$ or air.

The pooled results from a number of replica experiments (see table) show that only after irradiation in air considerably higher mutation frequencies were obtained for sperm from 7-day-old males than for that from 1-hour-old males; $X^2$ of the difference is 9.41, with $P < 0.003$. After radiation in $O_2$, the radiosensitivity in sperm of 7-day-old males was not significantly higher than in that from 1-hour-old males, and a similar result was obtained after irradiation in $N_2$.

The frequencies of recessive lethals (in the $X^{C2}y$ B chromosome), obtained from the first ejaculates of 1-hour and 7-day-old males which had been exposed to X-irradiation in $O_2$, air or $N_2$.

| | | 1-hour-old ♂♂ | | 7-day-old ♂♂ | |
|---|---|---|---|---|---|
| Radiation Exposure | Post Treatment | No. chromosomes tested | % lethals | No. chromosomes tested | % lethals |
| 2000 R in $O_2$ | $N_2$ | 1675 | 8.6 | 1162 | 9.0 |
| | $O_2$ | 1024 | 8.4 | 1555 | 9.5 |
| 3000 R in Air | $N_2$ | 695 | 9.4 | 587 | 12.9 |
| | Air | 626 | 8.5 | 430 | 12.8 |
| 4000 R in $N_2$ | $N_2$ | 1277 | 7.8 | 1639 | 7.4 |
| | $O_2$ | 790 | 9.0 | 1828 | 8.4 |

The oxygen enhancement ratio under comparable conditions of post-treatment, and this radiosensitivity in the presence of oxygen, is only slightly higher for sperm in 7-day-old males than for that in 1-hour-old males. The pronounced differences in sensitivity after radiation in air therefore clearly originate from a greater availability of oxygen for sperm in the old than for that in the young males, and a priori it is not unlikely that similar causes underly the differences in sensitivity of successive ejaculates derived from 3-day-old males.

The present results confirm an earlier conclusion by Oster (1961, J. Cell. Comp. Physiol. 58, suppl. 1:203-207), based on observations for first and second day sperm.

(Research carried out partially within the frame of the Association between Euratom and the University of Leiden, Contract Nr. 052-64-1 BIAN, and supported by the Institute for Radiopathology and Radiation Protection, and the Health Research Organization T.N.O.)

McCrady, W. B. and R. L. Sulerud, Arlington State College, Texas and Augsburg College. Additional factors which affect delayed-recovery from $CO_2$ in D. melanogaster.

In continuing studies of the phenomenon of delayed-recovery from $CO_2$ exposure in D. melanogaster, certain factors in addition to those reported earlier (Gen. 50:509-526) have been found to influence the recovery activity of TDR (Texas Delayed Recovery) flies following $CO_2$ exposure. Two of these factors are discussed below.

1. Genetic background: Differences in recovery activity have been observed in three TDR strains. Since controlling the age and the condition of the flies does not diminish the distinctness, the differences have been tentatively interpreted as being primarily due to the existence of different genetic modifiers of delayed-recovery in the strains indicated. The strains studied are all homozygous for Dly, the semidominant gene primarily responsible for delayed-recovery, but consistent variation between them has been found in recovery time and survival percentage following a standard $CO_2$ test (15 minute exposure to pure $CO_2$ at 14°C). An example of the variation found is given in Table 1. The strain designated TDR-$BC_3$ is least affected by $CO_2$ exposure. (This strain was developed by crossing heterozygous delayed-recovery progeny of the third backcross generation of crosses involving the mating of $CO_2$-resistant Oregon-R females and TDR males.) In the test shown, most (95%) TDR-$BC_3$ flies recovered, with all recovery taking place within an hour after testing. TDR-orange, an orange-eyed TDR mutant, is the "strongest" of the three strains. In this case recovery of survivors is seen to have required more than an hour, and only 61% survival occurred. (In other studies survival was often less than 50% for this strain.) Strain TDR-1a (derived from a female isolated from one of the original substrains established) is clearly intermediate between the other two as to recovery time and percentage of survivors. Other $CO_2$ tests of these strains have revealed similar differences between them, notwithstanding the fact that variation within the strains also exists. Studies have been initiated to determine the effects of selection on the recovery ability of these strains.

Table 1. Recovery activity of three TDR strains following $CO_2$ exposure for 15 minutes at 14°C.

| | | Percent apparently recovered after: | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Strain | n | 15 min. | 30 min. | 1 hr. | 2 hr. | 4 hr. | 8 hr. | 16 hr. |
| TDR-orange | 140 | 0 | 0 | 0 | 31.4 | 65.0 | 61.4 | 61.4 |
| TDR-1a | 185 | 0 | 3.2 | 15.7 | 49.7 | 70.3 | 86.5 | 87.6 |
| TDR-$BC_3$ | 62 | 3.2 | 37.1 | 100 | 100 | 100 | 95.2 | 95.2 |

2. Fly age: Members of the three strains indicated above were aged for varying periods and then exposed to $CO_2$ for 10 minutes at 10°C. The results are given as follows:

Table 2. Recovery activity of TDR flies of different ages following $CO_2$ exposure for 10 minutes at 10°C

| Strain | | Percent apparently recovered after: | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| and Age | n | 15 min. | 30 min. | 45 min. | 1 hr. | 1 1/2 hr. | 2 hr. | 2 1/2 hr. | 3 hr. |
| TDR-orange | | | | | | | | | |
| 1/2-1 1/2 days | 24 | 0 | 0 | 0 | 8.3 | 62.5 | 91.6 | 91.6 | 91.6 |
| 1 1/2-2 1/2 days | 20 | 0 | 0 | 0 | 0 | 40.0 | 90.0 | 95.0 | 85.0 |
| 2 1/2-3 1/2 days | 28 | 0 | 0 | 0 | 0 | 21.4 | 60.7 | 78.5 | 75.0 |
| 3 1/2-4 1/2 days | 24 | 0 | 0 | 0 | 0 | 4.1 | 25.0 | 45.8 | 58.3 |
| 4 1/2-5 1/2 days | 24 | 0 | 0 | 0 | 0 | 4.1 | 25.0 | 54.1 | 62.5 |

| | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| TDR-1a | | | | | | | | | |
| 1/2-1 1/2 days | 27 | 3.7 | 29.6 | 33.3 | 59.2 | 96.2 | 96.2 | 96.2 | 96.2 |
| 1 1/2-2 1/2 days | 36 | 0 | 0 | 5.5 | 33.3 | 83.3 | 88.8 | 100 | 100 |
| 2 1/2-3 1/2 days | 43 | 0 | 0 | 6.9 | 27.9 | 69.7 | 97.6 | 100 | 100 |
| 3 1/2-4 1/2 days | 28 | 0 | 0 | 7.1 | 28.5 | 57.1 | 96.4 | 96.4 | 96.4 |
| 4 1/2-5 1/2 days | 28 | 0 | 7.1 | 17.8 | 28.5 | 64.2 | 60.7 | 71.4 | 75.0 |
| TDR-$BC_3$ | | | | | | | | | |
| 1/2-1 1/2 days | 5 | 0 | 40.0 | 100 | 100 | 100 | 100 | 100 | 100 |
| 1 1/2-2 1/2 days | 6 | 0 | 33.3 | 100 | 100 | 100 | 100 | 100 | 100 |
| 2 1/2-3 1/2 days | 15 | 0 | 13.4 | 80 | 100 | 100 | 100 | 100 | 100 |
| 3 1/2-4 1/2 days | 21 | 0 | 4.7 | 66.6 | 85.7 | 95.2 | 100 | 100 | 100 |
| 4 1/2-5 1/2 days | 11 | 0 | 18.1 | 100 | 100 | 100 | 100 | 100 | 100 |

In keeping with what has been reported earlier, the recovery times are shorter and the recovery percentages are higher than would have been the case had testing been conducted under standard conditions (14°C for 15 minutes). It is also seen that TDR-orange was influenced the most by the test and TDR-$BC_3$ the least, as was expected. But in addition, the older flies of each strain were, in general, more susceptible to $CO_2$ treatment than younger flies. This is reflected in TDR-$BC_3$ by the somewhat longer recovery time for the older flies (the 4 1/2-5 1/2 day old flies are an exception to this rule). For the other two strains, not only was recovery accomplished more slowly by older flies but survival decreased appreciably with age. After an age of four or five days, the recovery time and recovery percentage stabilizes, so that flies older than this recover in a similar manner until the onset of senility.

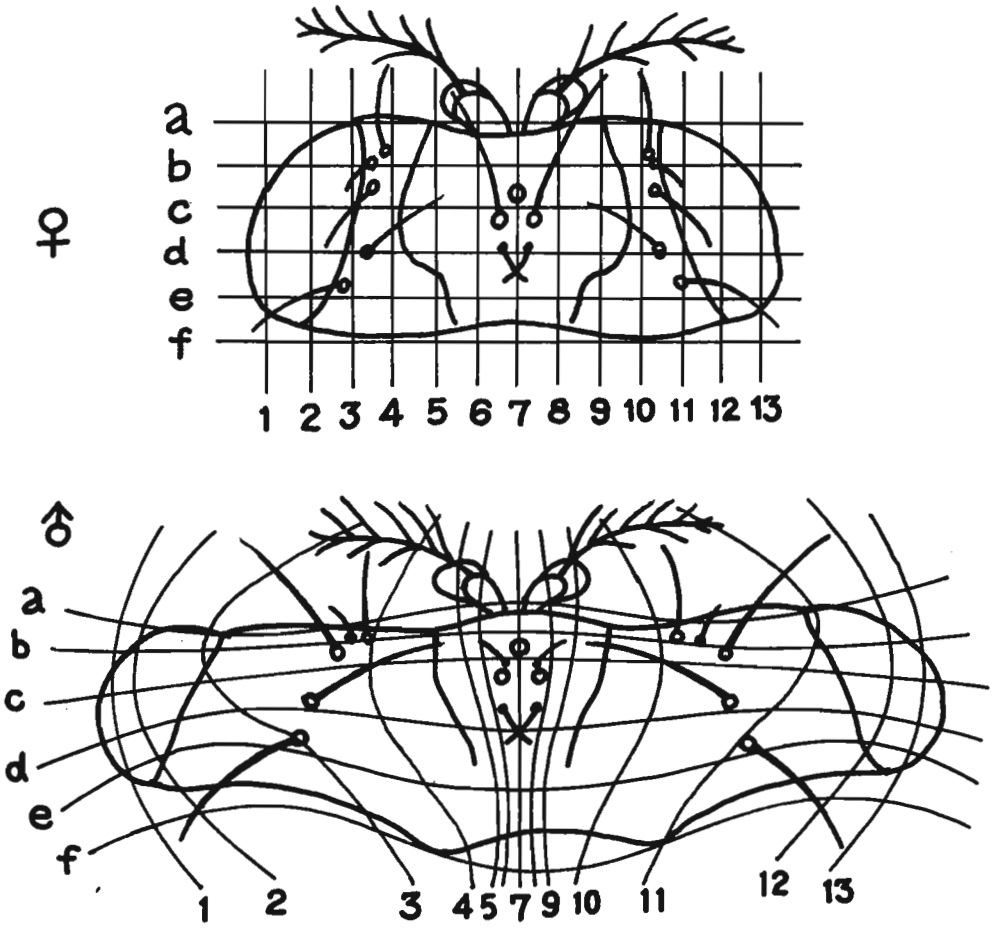
Bairati, A. Jr. and B. Baccetti. University of Milan and Entomologia Agraria Firenze, Italy. Observations on the ultrastructure of male germinal cells in the $X^{Lc}Y^{S}$ mutant of Drosophila melanogaster Meig.

We have subjected the testes of the $X^{Lc}Y^{S}$ mutant of Drosophila melanogaster to an electron microscope investigation with the techniques we use for studying normal spermiogenesis in the same species (Baccetti and Bairati, 1964 Redia 49: 1-29) with the object of obtaining comparative data on the ultrastructure of the flagellum in sperm that other workers consider to be immobile. The first conclusion we reached is that the majority of the sperm do not attain maturity, although the spermatid may undergo considerable lengthening and almost complete maturation, evinced by the development of the nucleus, of the mitochondrial derivatives and of the axial filament complex of the flagellum. Indeed all the cysts we examined presented degenerative features and no isolated mature sperm were observed. In the spermatids the mitochondrial derivatives presented several types of deviation from the norm and only rarely did they develop as in normal males. The alterations we observed were as follows: the presence of an electron opaque fiber in both of the mitochondrial derivatives that lie alongside the flagellum instead of in one of them only; complete absence of mitochondrial derivatives; separation of one or both derivatives from the axial filament; complete absence of the osmiophilic fiber inside both mitochondrial derivatives; vesicular swelling of one or both mitochondrial derivatives; the presence of a third element of the mitochondrial derivatives. These observations partially confirm the results of Shen (Zeit.f.Zellforsch. 1932, 15:547-580) and of Brosseau (Genetics 1960, 45: 257-274) regarding the sterility of males of this mutant, due to the absence of mature sperm through degeneration of the sperm and not because the mature sperm are immobile, but, owing to the greater resolving power of the electron microscope, one can detect abnormal spermiogenesis; since the alterations visible in the spermatids affect all the cells of the cyst, they probably begin at the premeiotic stage. The absence of a segment of the Y chromosome affects spermiogenesis by influencing the development of the mitochondrial derivatives.

Okada, Toyohi. Tokyo Metropolitan University. Material compensation revealed by the Cartesian Coordinates.

The genus Zygothrica is one of a few genera of Drosophilidae which involve species sexually dimorphic in the shape of heads. A species of this genus from Okinawa and Amami Islands, Z. asiatica (Okada), is an example, female head being normal and male head extensively prolonged laterally. An analysis was made of this prolongation of male head taking female head as standard by the method of Cartesian Coordinate of d'Arcy Thompson (fig.). It was found that the transformation can be analyzed into four processes. i. Transverse extension of the periorbits especially at anterior part (ordinates 2-5). ii. Transverse suppression at middle of front especially at posterior part (ordinates 5-9). iii. Vertical extension of the vertex (abscissae d-f). iv. Vertical suppression of the anterior regions of front and periorbits (abscissae a-c).

The combination of these processes reveals the occurrence of compensatory transformations along abscissae and ordinates in such manners as A) extension along abscissa (i) and suppression along ordinate (iv) at the anterior region of periorbits, and B) extension along ordinate (iii) and suppression along abscissa (ii) at the central region of vertex. This is an instance of the phenomenon of "compensation of the body material" (Rensch, 1954), one of the chief factors of transspecific evolution, having been revealed by the method of Cartesian Coordinates.



Robertson, A. and J. H. Louw. Institute of Animal Genetics, Edinburgh, Scotland.

Polymorphism of genes affecting amount and distribution of black pigment in the abdominal cuticle of D. melanogaster.

In the course of an investigation of lines selected for sternopleural bristles, it became clear that one line was a multiple recessive stock at several loci controlling the amount of pigment in the abdominal cuticle. Using this stock, two such loci have been clearly identified and located and the existence of others on the third chromosome is indicated. The effects of the genes are completely limited to females. Because of the existence of several loci with the same effect, the descriptions must be limited to the effects of substitutions in the multiple recessive stock. This has a large almost square black spot at the sides of the dorsal part of the 6th segment (Fig. I). A gene on the 4th chromosome, dominant at 25°, produces a black band, slightly narrower than the spot itself, round the dorsal side of the segment (Fig. II). Another gene, located at 3:-1, entirely removes the spot from the multiple recessive stock at 25°, although the heterozygote is intermediate at 18° (Fig. III). This dominant effect appears to be epistatic over other loci producing black pigment on the segment such as that located on the 4th chromosome. We have evidence of probably two other loci on the 3rd chromosome producing black pigment, one of which may be the e locus.

In our standard outbred population, the dominant gene at 3:-1 has a frequency of about 0.40 and the segregation appears to have little effect on fitness. The recessive on the 4th chromosome, on the other hand, much reduces male mating ability and female fertility and is at a very low frequency in the outbreeding population. It also reduces the number of sternopleural, abdominal and ocellar bristles. Temperature modifies both the effects of the segregations (in general lower temperatures making the flies darker) and their dominance relationships. The critical period for temperature changes appears to be shortly before emergence from the pupa. (All stocks reared at 25°.)

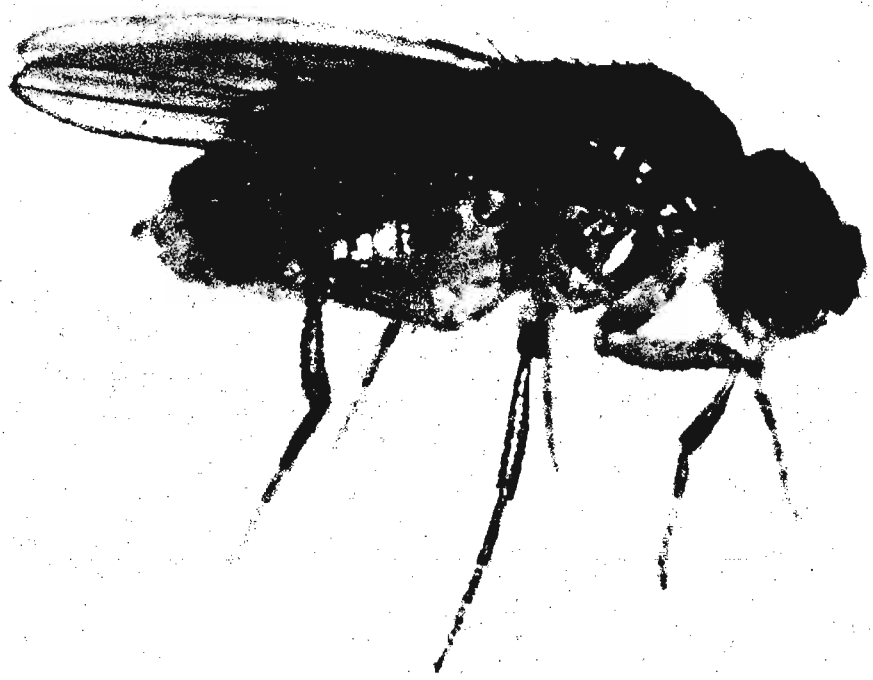


Fig. I

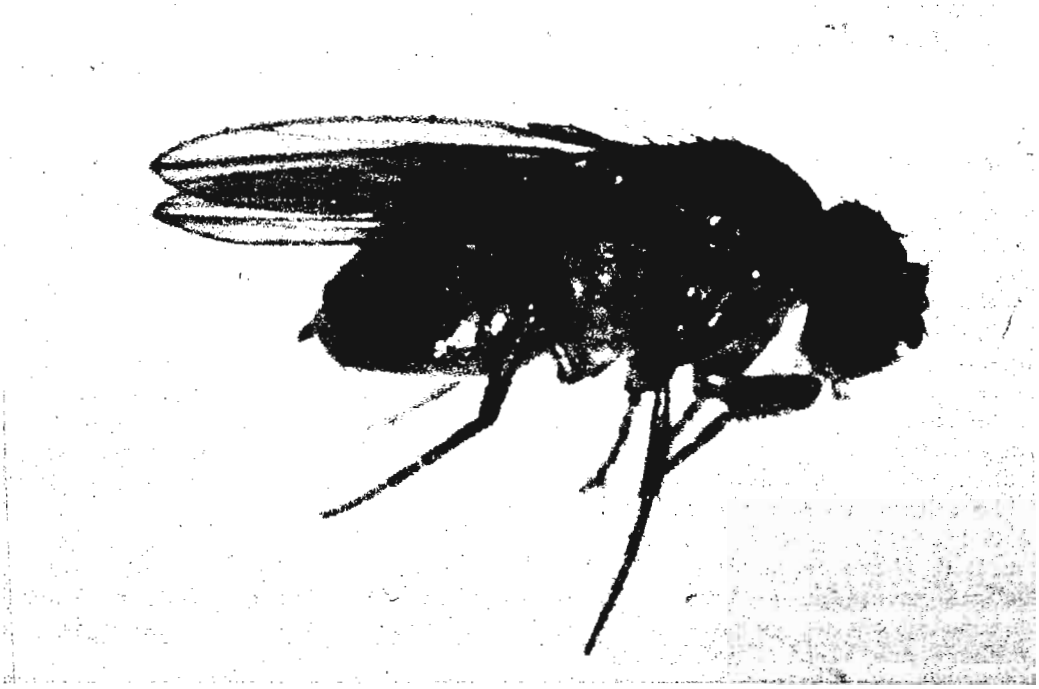


Fig. II

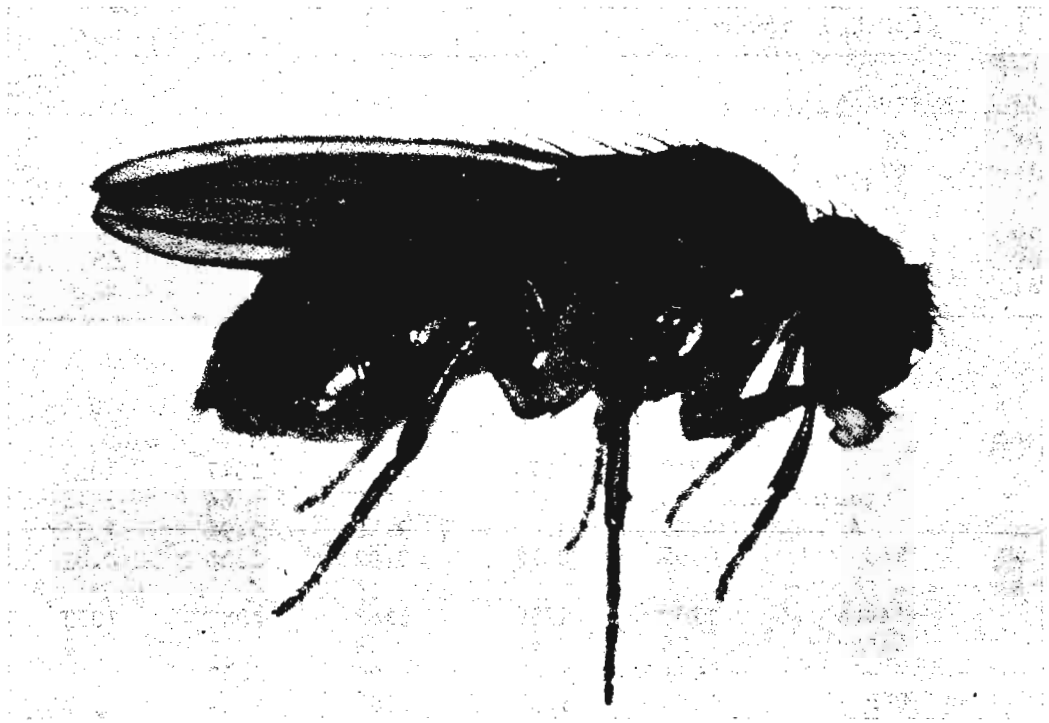


Fig. III

Lamb, M. J., T. W. McSheehy and C. E. Purdom. M.R.C. Radiobiological Research Unit, Harwell, England. Mutagenic effect of 600 MeV proton irradiation.

Differences in the mutagenic efficiency of radiations of different types and energies have been reported by several authors. These differences have been attributed to differences in the Linear Energy Transfer of the radiations. The present experiments, comparing proton irradiations with X-irradiation (250kVp, 15mA, HVL 1.2mmCu.) are part of a programme to investigate the relative effectiveness of different parts of the proton beam which have different LET values.

Oregon-K male flies 32 ± 3 hours old were exposed to 600 rads of protons or X-rays at dose rates of 150 rad/min and 540 rad/min respectively. Males were mated individually to 2 Cy/BIL females every three days, and the frequency of IInd chromosome recessive lethal mutations was scored among the offspring from 6 successive broods. The results are shown in Table 1. A very pronounced difference between the percentage of mutations induced by protons and X-rays was found in brood IV ($\chi^2 = 9.28$ $p = 0.0022$) but otherwise no significant differences were observed. This may be explained either on the assumption that the irradiated germ cell stages contributing to sperm used in brood IV were much more sensitive to proton than to X-irradiation, or that the mutagenic efficiency of protons was slightly greater than that of X-rays and a slight shift, or sampling error, during the brood sequence concentrated the effect into a single brood.

The second explanation seemed to be more plausible but the results of later experiments suggest that there is no difference between these radiations. In these further experiments 250kVp X-rays (800 rad) were compared with 600 MeV protons (800 rad) and with attenuated protons in the Bragg peak (400 rad). The results are shown in Table 2. In only one brood was there any significant deviation from an RBE value of 1 relative to X-rays. This was brood II of the 600 MeV proton series which was significantly low at the 5% level. Overall analysis, however, showed no deviation from one and there was no indication, therefore, that the increase in frequency of nuclear interaction in the Bragg peak region was accompanied by any increase in genetic effectiveness. (Acknowledgement: We are very grateful to Dr. J. Baarli of CERN for arranging the proton irradiation and advising on the problems of proton dosimetry.)

Table 1

| Brood | 600 MeV Protons | | | 250 kVp X-rays | | | $\chi^2$ | P |
|---|---|---|---|---|---|---|---|---|
| | No. of tests | No. of lethals | % | No. of tests | No. of lethals | % | | |
| I | 1288 | 28 | 2.17 ± 0.41 | 800 | 24 | 3.00 ± 0.60 | 1.07 | 0.30 |
| II | 1411 | 57 | 4.04 ± 0.52 | 776 | 26 | 3.35 ± 0.65 | 0.48 | 0.50 |
| III | 1325 | 82 | 6.19 ± 0.66 | 795 | 51 | 6.42 ± 0.87 | 0.013 | 0.91 |
| IV | 1160 | 97 | 8.36 ± 0.81 | 737 | 34 | 4.61 ± 0.77 | 9.28 | 0.0022 |
| V | 1369 | 41 | 2.99 ± 0.46 | 764 | 28 | 3.66 ± 0.68 | 0.51 | 0.48 |
| VI | 1381 | 27 | 1.96 ± 0.37 | 792 | 11 | 1.39 ± 0.42 | 0.64 | 0.42 |

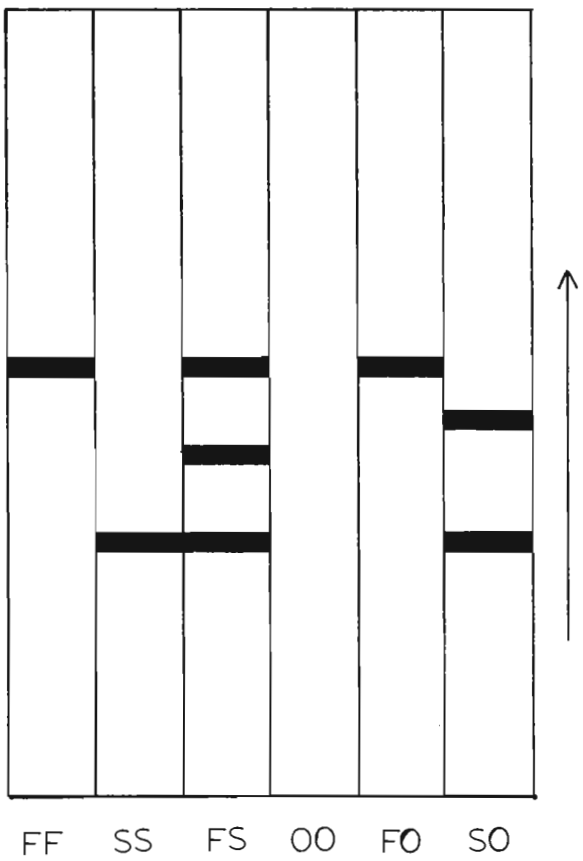
Table 2

| | Brood | I | II | III | IV | V | VI |
|---|---|---|---|---|---|---|---|
| Proton Beam | | | | | | | |
| 800 rad | Tests | 1246 | 1176 | 1119 | 1077 | 1050 | 1292 |
| | Lethals | 56 | 47 | 107 | 64 | 14 | 15 |
| | % | 4.49 | 4.00 | 9.58 | 5.94 | 1.33 | 1.16 |
| | RBE | 0.95 | 0.70 | 0.92 | 1.28 | 0.82 | 1.31 |
| Bragg Peak | | | | | | | |
| 400 rad | Tests | 1269 | 1226 | 1183 | 1080 | 1053 | 1288 |
| | Lethals | 28 | 42 | 58 | 31 | 8 | 13 |
| | % | 2.21 | 3.43 | 4.90 | 2.87 | 0.76 | 1.01 |
| | RBE | 0.94 | 1.23 | 0.85 | 1.26 | 0.87 | 2.30 |
| X-rays | | | | | | | |
| 800 rad | Tests | 1287 | 1248 | 1072 | 1009 | 1102 | 1348 |
| | Lethals | 61 | 71 | 112 | 46 | 18 | 12 |
| | % | 4.74 | 5.69 | 10.45 | 4.56 | 1.63 | 0.89 |

Johnson, F. M. University of Wisconsin.
The inheritance of a deficiency of larval alkaline phosphatase in D. melanogaster.

During the latter hours of larval development a densely staining alkaline phosphatase (APH) zone is detectable when single individuals are electrophoresed in starch gels. Previous reports (Nature 20: 321, 1964; Genetics 49: 829, 1964) have described the genetic control of an electrophoretic variation in that zone and noted the appearance of a hybrid enzyme in heterozygotes. The observations are consistent with the hypothesis that the acitve enzyme is a dimer consisting of two identical subunits in homozygotes and that combination of unlike subunits in heterozygotes produces the enzyme of intermediate mobility.

An investigation of the various isozyme patterns of an inbred car strain of Drosophila revealed a complete lack of demonstrable APH activity in the area of the normally dense APH component. After confirming the deficiency in several consecutive generations, adult flies fron the stock were mated with Fast ($Aph^F/Aph^F$) and Slow ($Aph^S/Aph^S$) types and the $F_1$ larval offspring examined. As shown in the accompanying diagram the deficient x Fast hybrid produces only a Fast band, the intensity of which is only slightly if any less dense than that of $Aph^F/Aph^F$ type. Alternately, when deficient x Slow hybrid larvae are examined, in addition to the expected Slow band a band in a position slightly above that of the hybrid zone of $Aph^F/Aph^S$ heterozygotes is also observed.




Schematic comparison of larval APH phenotypes in phosphate buffer, 0.01 M, pH 6.5. Genotypes are indicated below their respective patterns.

Except for the "extra" band in the deficient x Slow heterozygotes the phenotypes are compatible with deficient control by a "silent" $Aph^O$ allele. This is supported by the segregation ratios resulting from backcrosses, outcrosses and $F_1$ x $F_1$ mating of deficiency heterozygotes. A summary of the mating experiments is shown in the Table. Preliminary

Summary of crosses demonstrating segregation of $Aph^O$

| Parental Aph Combinations | Offspring FF and/or FO | OO | SO | FS | SS | Total |
|---|---|---|---|---|---|---|
| FF x OO | 31 | | | | | 31 |
| SS x OO | | | 29 | | | 29 |
| FO x OO | 74 | 52 | | | | 126 |
| FO x FF | 84 | | | | | 84 |
| SO x OO | | 97 | 69 | | | 166 |
| SO x SS | | | 83 | | 87 | 170 |
| SO x FF | 24 | | | 21 | | 45 |
| FO x SS | | | 70 | 67 | | 137 |
| FO x FO | 84 | 29 | | | | 113 |
| SO x SO | | 27 | 60 | | 20 | 107 |
| | | | | | | 1008 |

support for allelism has been obtained by comparing map distances between Aph and a nearby locus using the deficiency as well as the electrophoretic variation.

The presence of the "extra" band in $Aph^S/Aph^O$ heterozygotes is possibly the result of combination of an S subunit and the product of the "silent" allele, in which case $Aph^O$ is producing protein which cannot dimerize or does dimerize but is inactive for some other reason (under the test conditions employed). No indication of a double band has yet been found in $Aph^F/Aph^O$ type larvae. All attempts in starch with continuous and discontinuous buffer systems at pH 9.5, 8.5, 7.5, 7.0, 6.5, 6.0 and 3.0, polyacrylamide slabs at pH 8.5 and 7.0, and disc electrophoresis (Ornstein and Davis, 1961) running at pH 9.5 and 6.6 (cf. brochure, Canal Industrial Corporation) show only the single band. This might suggest a structural difference between Fast and Slow APH which prevents the Fast and "silent" protein subunits from combining into active enzyme.

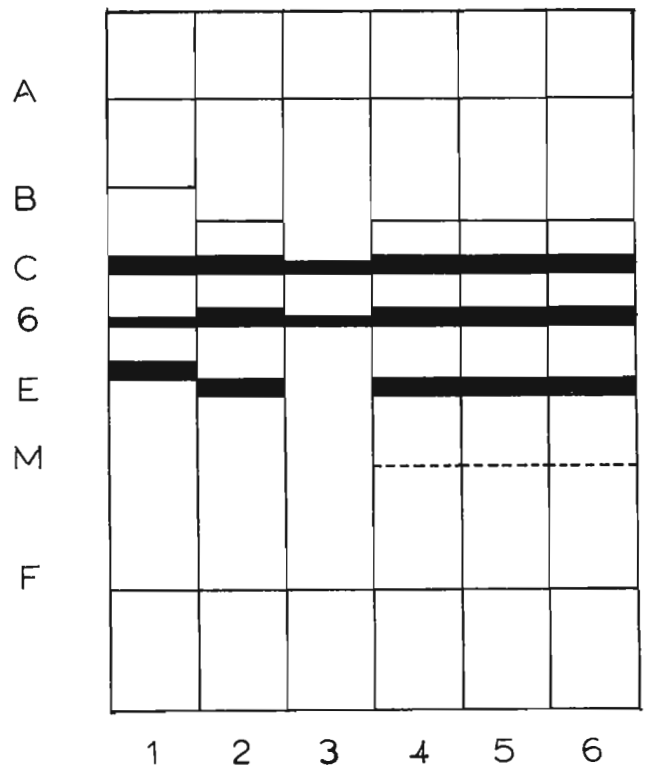
Johnson, F. M. University of Wisconsin.
Carmen G. Kanapi University of Hawaii.

Esterase differences between male and female D. melanogaster.

Esterase zymograms of single adult Drosophila show a quantitative difference in Esterase 6 between males and females - the males having a greater amount of the enzyme or a more active form. A minor esterase component. Esterase M, sometimes not observable, when present is of greater intensity in males than in females or entirely absent in females, depending apparently on the strain under examination.

Multiple homogenates, adjusted to equivalent protein concentrations, show in addition to the quantitative variations, a slightly greater mobiltiy of Esterase B and Esterase E in females. Repeated freezing and thawing aids in making the mobility difference apparent.

Suspecting either a male esterase enhancing substance or a female esterase suppressing substance and assuming a non-random distribution of such a substance within the body, individuals of both sexes were severed in the mesothorax with a lancet and homogenates of the anterior and posterior parts prepared. On examination of the homogenates in starch gels, very little difference could be observed among female anterior, male whole, male anterior and male posterior homogenates. Only female whole homogenates show the increased mobility of Esterase bands B and E while female posterior homogenates have a decreased intensity in nearly all bands. Hence, the complete set of esterase differences can be explained on the basis of a female suppressing substance located in the posterior portion of the fly. The pertinent esterase patterns are diagrammed in the figure.




Females of the constitution XXY show a typical female esterase pattern and XO males show the pattern of normal males; XXY tra/tra females with a phenotypic resemblance to males show a male esterase pattern. The inheritance, therefore, appears to be sex-limited.

A tendency for males to exhibit a female-like esterase pattern when raised on media containing progesterone or stilbestrol has been indicated from preliminary experiments. Unfortunately the purity of the hormones was questionable in these first experiments.

Legend for figure:

1) Female multiple whole fly homogenate
2) Female multiple anterior homogenate
3) Female multiple posterior homogenate
4) Male multiple whole fly homogenate
5) Male multiple anterior homogenate
6) Male multiple posterior homogenate

Johnson, F. M., B. B. Wallis and C. Denniston. University of Wisconsin.

## Recessive esterase deficiencies controlled by alleles of Est C and Est 6 in D. melanogaster.

Electrophoretic variation of two esterase zones, Esterase C and Esterase 6, in starch gels is controlled by two independent loci on Chromosome III. A further search for additional inherited esterase pattern differences has revealed one Drosophila strain, sc ec ev $et^6$ v $g^2$ f/FM3 $y^{31d}$ $sc^8$ dm B 1, which exhibits no detectable Esterase C and another, car, which shows no Esterase 6 activity.

Offspring resulting from crosses of individuals lacking either enzyme band to corresponding fast and slow types resemble, with little or no apparent difference in activity, the fast and slow types to which they were mated. Backcross and $F_2$ progenies include enzyme deficient types in ratios generally not contradictory to single recessive factor inheritance. Control by a recessive suppressor does not seem likely since a heterozygous (FS) pattern did not result from any mating, though, logically a suppressor cannot be completely ruled out. The Aph to Est C and Aph to Est 6 linkage map distances have been reditermined, using the deficiencies rather that electrophoretic variation, and values similar to previous results were obtained.

The segregation pattern, granting allelism and provisionally designating the "silent" allele by an O, in which the deficient types were mated to flies of the most common esterase genotype (Est $C^F$/Est $C^F$, Est $6^S$/Est $6^S$) and then backcrossed and inbred, is summarized in the following table.

| Mating | | Offspring | | Total | $\chi^2_1$ |
|---|---|---|---|---|---|
| Est C | F/O x O/O | 81 F/O | 81 O/O | 162 | 0.0 |
| Est 6 | S/O x O/O | 108 S/O | 54 O/O | 162 | 18.0** |
| Est C | F/O x F/O | 64 Fast (F/F & F/O) | 30 O/O | 94 | 2.5 |
| Est 6 | S/O x S/O | 79 Slow (S/S & S/O) | 15 O/O | 94 | 4.1* |

The tendency for the Esterase C deficient types to be produced in expected ratios and Esterase 6 deficient flies to occur less often than expected has been observed consistently in a number of small scale experiments (designed for other purposes) also. Since there exists a striking quantitative variation between males and females in regard to Esterase 6, and because of the abberant ratios, an important biological function for the enzyme is intuitively suspected, though still not known.

No association between esterase electrophoretic mobility of deficiency and external morphology has yet been detected. It has not been determined whether or not the deficiencies reflect a lack of protein, inactive enzyme, or as suggested by T. R. F. Wright, labile enzyme unable to tolerate the conditions of electrophoresis.

Glassman, E. University of North Carolina Medical School, Chapel Hill.

## Chemical selector agents for xanthine dehydrogenase (XDH) mutants of Drosophila melanogaster.

For many years it has been apparent that a chemical selector system for backmutations and wild-type recombinants at the ma-1 and the ry mutants would be very useful. Recently we have discovered that if 0.01 to 0.02% purine is added to our Drosophila media (which is a modification of the media devised by Dr. E. B. Lewis) ma-1 and ry are killed during development, while most lxd and wild-type survive. Presumably this very toxic compound is converted to hypoxanthine by XDH, and only flies having this enzyme survive. Thus, extensive studies on backmutation and genetic fine structure at the ma-1 and ry loci are now possible. When the level of purine is raised to 0.06% even wild-type flies do not survive. After treatment with mutagens it should be possible to produce and select for purine-resistant stocks. Hopefully the mechanism of the resistance will be the presence of high amounts of XDH in these flies. This method supplants the use of 4-hydroxypyrazolo(3,4-d) pyrimidine which inhibits XDH in vivo and which converts wild-type into phenocopies of ma-1 and ry flies as reported by Keller and Glassman (Nature, in press).

A. S. Mukherjee and Ashish Dutta Gupta.
University of Calcutta, India. The role of heterochromatin in the control of gene activity.

In a recent report from Dr. J. Schultz's laboratory, it has been shown that a block of heterochromatin when brought close to certain euchromatin bands inhibits puffing of those bands in the salivary gland chromosomes of Drosophila melanogaster (Rudkin 1964). It appears that heterochromatin may have a general inhibitory role on the genetic activity.

While studying the puffing pattern in Drosophila ananassae (a local population), in our laboratory, we have found certain features which do not permit us to generalize the inhibitory role of heterochromatin. Salivary gland chromosomes of D. ananassae contain a large number of chromosomal rearrangements (Jha, 1964). In addition, chromosome aberrations involving only one nucleus or two nuclei are found in high frequency in the $a^{6}+$ (Calcutta) stock currently under study. On the basis of observations so far made it appears that the reasons for such frequent "aberration mosaicism" may be due to the presence of heterochromatin of varying size over the length of the chromosomes. The details of the distribution of heterochromatin, degree of heterochromatinization of different bands and their relation to chromosomal aberrations will be published elsewhere. It is intended to present here a few cases of puffing which appear to be differentially modulated by the heterochromatin adjacent to them.

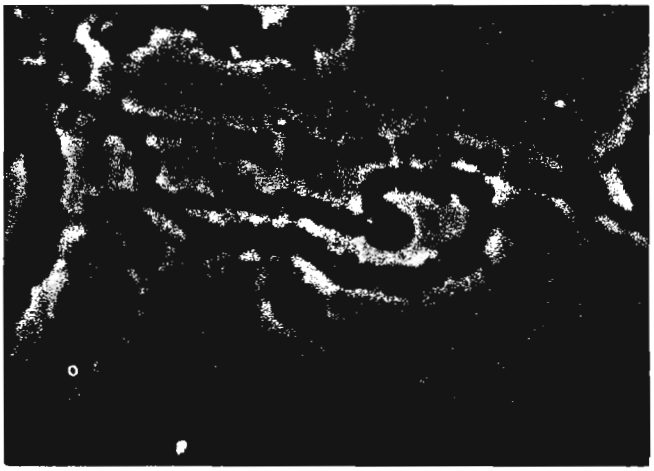


Fig. 1. Photograph of the subterminal inversion in the 3L. Arrow indicates the region of puff.

Figure 1 presents an inversion loop (the delta inversion of Jha, 1964) in which the two breaks are in 8A4 and 1A, respectively. The distal break is between the last dark band and the tip puff. Normally the tip remains puffed throughout the third instar. In this case, however, the tip of one and the same member of the two homologous chromosomes always shows puffing, while that of the other almost always ends in one or more dotted bands. A thin delicate thread-like connection is frequently observed under phase contrast, in the latter homolog, between the region preceding the dotted bands and any other non-homologous region. Such ectopic pairing indicates the presence of heterochromatin (intercalary?) in that region. This means that due to the inversion a piece of heterochromatin has been transposed into the region preceding the tip. It is clear, therefore, that this small piece, perhaps comparable to one single band, of heterochromatin is capable of inhibiting the tip puff. It is interesting to note that in cases, where the chromosomes concerned are completely unpaired, one homolog ends in a puff, the other shows two dark bands followed by one faintly stained dotted band.

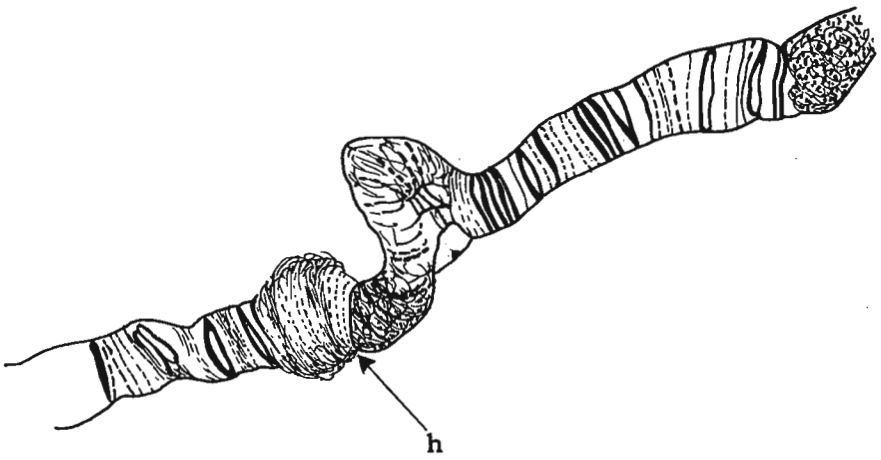



Fig. 2. Camera Lucida drawing of the puff and the adjacent heterochromatin in the XR. h=heterochromatic region.

The second set of cases on the other hand has larger segments of heterochromatin adjacent to puffs. One such case is presented in Figure 2. This represents the presence of a big puff close to a rather big block of heterochromatin. Clearly, the puff remains

unsuppressed even in presence of the heterochromatin closeby.

These preliminary findings show that there may be at least two functionally different kinds of heterochromatin. In normal gene sequence the bigger blocks of heterochromatin may have no or little effect on the control of genetic activity, while smaller intercalary heterochromatin bands may inhibit gene expression. Salivary gland chromosomes of D. ananassae proves to be a unique material for the study of the function of the heterochromatin in a normal genic complement. It remains for future investigation to examine the puffing in chromosomes homozygous for the inversion and also in homozygous normal chromosomes.

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Burnet, B. University of Sheffield, England. Allelism of tumour genes.

Hartung (1950, J. Hered.,41: 269) reports the location of a melanotic tumour gene at 2-83.9 in the $mt^A$ strain of D. melanogaster. This tu allele was used by Kanehisa (1956, Jap. J. Genet.,31: 144) for the synthesis of a number of tumour strains incorporating other mutants affecting eye pigmentation. Glass (1954, DIS 28: 74) reports that the tumour gene on the second chromosome of the su-er tu bw; st er su-tu strain is also an allele at 2-83.9. A detailed study of gene environment interactions involving this locus is given by Burnet and Sang (1964, Genetics 49: 223-235 and 599-610). The tumour gene on the second chromosome of the tu-B3 strain described by Barigozzi and De Pasquale (1956, Rend. Ist. Lomb. Sci. Lett., 90: 484) appears to be an allele at the same locus. The tumour penetrance (percentage of tumorous individuals) in crosses in all combinations between tu-B3, su-er tu bw; $+^{su-tu}$ and the $tu^{48a}$ vg bw strain described by Gélélovitch (1958, Biol. Méd., 47: 711) is shown in Table 1. The tumour gene $tu^{48a}$ is located at 2-29.5.

Table 1

| ♀♀ \ ♂♂ | tu bw; $+^{su-tu}$ | tu-B3 | $tu^{48a}$ vg bw |
|---|---|---|---|
| tu bw; $+^{su-tu}$ | 97.50 | 95.00 | 1.85 |
| tu-B3 | 85.20 | 90.40 | 2.14 |
| $tu^{48a}$ vg bw | 1.21 | 1.38 | 96.60 |

The allelism of the tumour genes in the su-er tu bw and tu-B3 second chromosomes is further supported by their interaction with the suppressor locus on the third chromosome st er su-tu. On a standardized first chromosome background both alleles are suppressed by su-tu, whereas $tu^{48a}$ does not appear to interact with the suppressor, as shown in Table 2.

Table 2

| | tu bw | tu-B3 | $tu^{48a}$ vg bw |
|---|---|---|---|
| $\frac{st\ su-tu}{+\quad +}$ | 93.60 | 93.40 | 43.1 |
| $\frac{st\ su-tu}{st\ su-tu}$ | 7.14 | 8.69 | 45.0 |

The reduction in penetrance observed in both these $tu^{48a}$ combinations is due to dispersion of the modifier background particularly in the first chromosome of the original strain. Further observations are necessary to decide whether the effects of the suppressor are restraictd to tu alleles at locus 2-83.9.

Mac Intyre, R. J. Cornell University. Ithaca, N.Y. Acid phosphatase variations in D. melanogaster and D. simulans.

Single larva, pupae or adults from both species were electrophoresed in starch gels prepared in a tris-hydrochloric acid buffer, pH 8.6, 0.05M (see Wright, 1963, Genetics 48:787). The gels were then incubated in acetate buffer, pH 5.0, containing alpha napthyl phosphate (100 mg/100 ml), $Mg^{+2}$ and $Mn^{+2}$ (10 drops of 10% solution/100 ml), polyvinylpyrillodone (0.5 g/100 ml), NaCl (2g/100 ml) and Fast Blue BB (200 mg/100 ml). Three phenotypes were found in each species in a survey of stocks at our disposal. They are diagrammed in Figure 1 as solid lines. Stocks were established which were monomorphic for the "fast" and "slow" bands, and it was found that the three band pattern was always obtained in $F_1$ progeny of a cross between "fast" and "slow" strains. Backcross and $F_2$ progeny in both species fell into two and three phenotypic classes in numbers statistically consistent with a monogenic mode of inheritance. The genes have been called Acph-1 for the first acid phosphatase gene-enzyme system to be described in Drosophila. The codominant alleles, which presumably interact to form a hybrid enzyme (along with both parental forms) in the heterozygote have been designated as $Acph\text{-}1^A$ (melanogaster or simulans) and $Acph\text{-}1^B$ (melanogaster or simulans) in the order of their discovery. Fortuitously, the $Acph\text{-}1^A$ alleles in both species specify the enzymes with the slowest rates of migration described to date. The interaction of allele products indicates that the enzyme exists in molecular form at least as a dimer. Both genes were mapped on element E, just to the right of the homologous claret genes in the two species. The locus in D. melanogaster is 101.4 ± 0.1: in D. simulans, 133.7 ± 2.7.

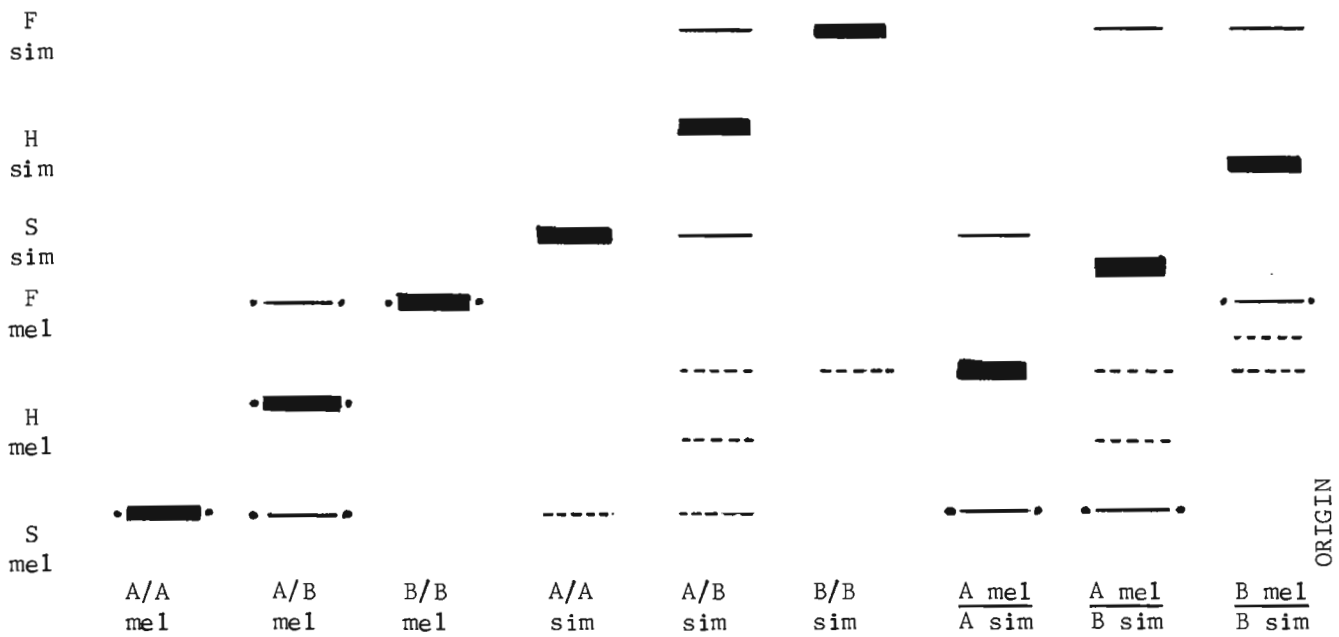


Figure 1. Diagrammatic representation of composite zymograms of acid phosphatases in D. melanogaster and D. simulans and three interspecific hybrids. "F", "H" and "S" refer to "fast", "hybrid" and "slow" enzymes respectively. A and B are designations for the codominant alleles, $Acph\text{-}1^A$ and $Acph\text{-}1^B$, of each species. The genotypes producing each pattern, including those of the interspecific hybrids, are indicated directly below the line of origin. Solid lines refer to the position of the bands of each genotype in starch prepared in tris-hydrochloric acid buffer. Dotted lines indicate the positions of the same enzymes in starch gelled in a tris-EDTA-boric acid buffer. The pattern indicated by •———• denotes that one or more enzymes had the same rate of migration in both buffer systems.

This close correspondence in the positioning of the two genes within homologous elements, plus the fact that interspecific hybrids show patterns typical of heterozygotes (see Figure 1) strongly suggests that the acid phosphatases of the sibling species are controlled by homologous structural genes. However, as yet no allele has been found that is shared by both the species. An additional difference between enzymes of D. melanogaster and D. simulans may be indicated by the differential effect of EDTA (and/or boric acid) on their rates of migration in starch prepared in a tris-EDTA-boric acid buffer. As shown in Figure 1, the enzymes specified by alleles of D. simulans (including those "heterozygote" enzymes of interspecific hybrids) show reduced rates of migration. This is not true of the acid phosphatases characteristic of D. melanogaster. The migration rate of the Acph-$1^A$ "homozygote" enzyme in tris-EDTA-boric acid starch appears to be the same as that of the Acph-$1^A$ enzyme of D. melanogaster in starch of both buffer systems. However, the enzyme specified by Acph-$1^B$ homozygotes of D. simulans migrates in tris-EDTA-boric acid starch to a position between the two "homozygote" bands of D. melanogaster. The results summarized in this communication will be reported in detail elsewhere (MacIntyre, R. J. The genetics of an acid phosphatase in D. melanogaster and D. simulans. Manuscript in preparation.)

Gfeller, Sister M. David. University of Oregon, Eugene. A quantitative comparison of the fluorescing eye pteridines in male and female D. melanogaster.

Five day old + Ore-R males and females have similar quantities of all the fluorescing eye pteridines (described by Hadorn and Ziegler, 1958, Z. Vererb.-Lehre, 89: 221-234) unless a weight factor to correct for the smaller head size of males as compared to females is introduced. After correction for weight, males have a significantly (0.01 level) greater quantity of all the pteridines.

On the other hand, if isoxanthopterin and xanthopterin (usually measured as one fluorescing spot) are measured separately, + Ore-R females show significantly more isoxanthopterin than males, whether a weight factor is introduced or not. The fluorometric means, corrected for weight, are as follows:

| | Females | Males |
|---|---|---|
| Drosopterins | 40.1 + 2.77 | 51.3 + 2.96 |
| Isoxanthopterin | 56.3 + 4.16 | 50.7 + 4.87 |
| Xanthopterin | 34.2 + 1.82 | 43.6 + 4.71 |
| HB* + sepiapterin | 21.8 + 1.76 | 27.3 + 2.11 |

*HB consists of 2-amino-4-hydroxypteridine and biopterin.

The isoxanthopterin results are in accord with the report of Munz (1962, DIS 36:96) that Ore-R females have greater xanthine dehydrogenase activity than males. However, the above results are contrary to Hadorn and Ziegler's (1958) report that + Sevelen males have twice as much isoxanthopterin as females and less of all the other pteridines. The introduction of a weight correction for Ore-R pteridine values may account for some of the discrepancies between males and females of this and + Sevelen wild type but hardly for the great differences in isoxanthopterin content.

Even greater biochemical sex differences are found in the eye pteridines of the mutant white-blood ($w^{bl}$) and compounds of $w^{bl}$ than in Ore-R. But the differences are not in the same direction in $w^{bl}$ and $w^{bl}$ compounds. In the mutant $w^{bl}$, as in Ore-R, males have a significantly smaller amount of isoxanthopterin and greater amounts of all the other pteridines than females. However, in $w^{bl}$,v, $w^{bl}$;cn and $w^{bl}$;st compounds with genetic blocks to ommochrome formation, males have a significantly greater amount of isoxanthopterin as well as the other pteridines.

The quantity of isoxanthopterin appears to be modified not only by sex but also by genes associated with both the formation of the pteridines and the ommochromes and thus may be a necessary component for the production of both pigments.

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Ayala, Francisco J. The Rockefeller Institute. New York. Competition experiments between Drosophila species.

Six populations, each with two competing species, were started at two temperatures. One of the species was always D. dominicana the other species were D. pseudoobscura, D. nebulosa and D. melanogaster. (In the case of D. melanogaster the mutant vg was used at 19° C and a strain carying several sex linked mutants at 25° C). Each population was started with 200 flies of each species in a 1/2 pint milk bottle with Spassky's medium. Once a week, the surviving flies were scored and then transferred to a fresh bottle. On the same day, the flies hatching in the previous bottles of each series were scored and then added to the bottle with the old flies. At 25° C the bottles were discarded at the end of the 4th week, and at 19° C at the end of the 6th week. The frequencies of D. dominicana in each population are presented in Fig. 1.

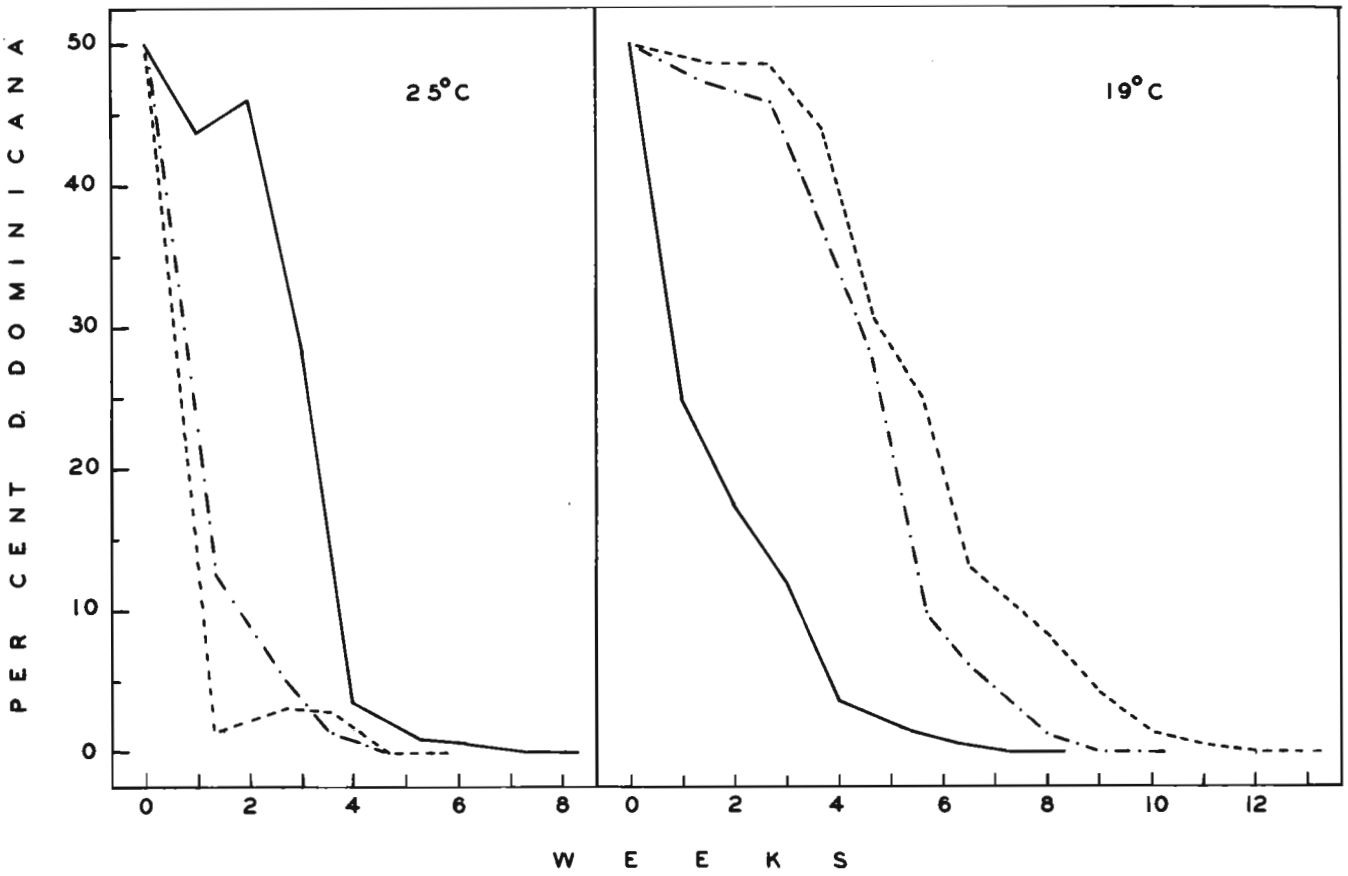


Fig. 1. Frequency in per cent of D. dominicana: ——— in competition with D. pseudoobscura; ------ in competition with D. nebulosa; — . — . in competition with D. melanogaster.

At 25° C it had been eliminated by D. pseudoobscura by the 8th week, and by the other two species by the 5th week. At 19° C D. dominicana was eliminated by the 8th week by D. pseudoobscura, by the 9th by D. melanogaster, and by the 12th by D. nebulosa. D. pseudoobscura has the highest competitive ability at 19° C and the lowest at 25° C; the reverse is true of D. nebulosa.

Three populations were started at 25° C with D. serrata and D. melanogaster (wild type), one with 200 individuals, the second with 800, and the third one with 1600 individuals, of each species. The technique was the same as before. It was thought that at higher densities D. serrata might be at an advantage, but it was eliminated in all cases, and faster when the density was higher (the 5th week) than when it was lowest (the 8th week).

In a population started at 25° C with 200 individuals of D. dominicana and 200 of D. serrata, the former was eliminated by the 8th week.

Sena, Elissa and R. J. MacIntyre. Cornell University. Adenosine triphosphatase activity of 3rd larval instar alkaline phosphatase (Aph).

Several non-specific enzymes have been described in Drosophila melanogaster. Among these is an alkaline phosphatase reported by Beckman and Johnson (1964). It has been demonstrated that larval alkaline phosphatase bands separated by starch gel electrophoresis and stained by the technique of Beckman and Johnson (1964) all show adenosine triphosphatase activity (ATP-ase activity). The latter activity is observed using a modified lead conversion method (Allen, 1963). The identity between alkaline phoshpatase and ATP-ase banding in larva can also be shown with the alizarin red S method (see Sandler and Bourne, 1961).

Attempts at identification of the non-specific alkaline phosphatase as a true ATP-ase have made use of the known specific inhibitors of ATP-ase activity, N-Ethyl maleimide (NEM) and p-chloromercuribenzoic acid (PCMB) reported by Padykula and Herman (1955 a and b). These inhibitors were tested in the following manner: After electrophoresis of the larval enzymes, the starch gels were sliced into thicknesses of 2 mm. One strip was placed into barbital buffer at pH 9.0 containing the inhibitor in question. Concentrations of NEM tested were: $5 \times 10^{-4}$ M, $5 \times 10^{-3}$ M, and $1 \times 10^{-3}$ M. The PCMB concentrations used were $5 \times 10^{-3}$ M, $2.5 \times 10^{-3}$ M, $2 \times 10^{-2}$ M and $1 \times 10^{-2}$ M. These gels were incubated at room temperature for 1/2 to 1 hour, then rinsed in two changes of 2% $CaCl_2$ for ten minutes and in distilled water. Finally, the gels were stained for ATP-ase activity by the lead conversion method (Allen, 1963). The intensity and patterns of banding were compared with controls, i.e. 2 mm strips from the same initial gel, which had been subjected to the same treatments except that the inhibitor was absent from the barbital buffer. In this way qualitative effects of the inhibitors could be detected, but substantial inhibition was not observed with either inhibitor at any concentration. In order to re-name this non-specific alkaline phosphatase as an ATP-ase, specific and reversible inhibition of this enzyme should first be demonstrated. At this point, the adenosine triphosphatase activity of the 3rd larval instar alkaline phosphatase must be regarded as spurious.

References

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Falke, Ernest V. and R. J. MacIntyre. Cornell University. The genetic localization of a non-specific leucine aminopeptidase in Drosophila melanogaster.

Leucine aminopeptidase (Lap) activity has been demonstrated in starch gel zymograms of Drosophila melanogaster pupae. One of the zones of activity, the D zone, or Lap D, was found to exist as two forms which differed in electrophoretic mobility. Genetic tests revealed that the two forms of the enzyme are controlled by codominant alleles on Chromosome III (Beckman and Johnson, 1964).

Stocks homozygous for the alleles controlling either the "fast" enzyme or the "slow" enzyme were obtained from Dr. T. R. F. Wright and used to more precisely map the locus of the gene. The third chromosome marker stocks used were ru h th st cu sr $e^s$ ca (rucuca) and st sbd $e^s$ ro ca (steroca). Both stocks were found to be monomorphic for the "fast" band and, thus, presumably homozygous for the Lap $D^F$ allele.

The approximate position of the gene was determined with the rucuca stock and a stock which was homozygous for the allele controlling the "slow" band (Lap $D^S$). The final analysis was done with the steroca stock.

Since enxyme activity is strong in single pupae but not in single adults, the testcross adult progeny could be classified by morphological phenotype only. It was therefore necessary to cross single males carrying recombinant chromosomes, whose morphological phenotype was known, to homozygous Lap $D^F$/Lap $D^F$ virgin females, and analyze single pupal squashes from these crosses. This procedure then completed the phenotypic analysis of the testcross progeny.

The analysis of the pupae was carried out using the methods outlined by Wright (1963) for starch gel electrophoresis. The trays were run for four hours at 5-8 v/cm and stained using L-leucyl-beta-naphthylamide as the substrate and Black K salt as the dye (Beckman and Johnson, 1964). Crosses with the rucuca stock indicated that the Lap D gene is located between ro and ca. The relative frequency with which the Lap D alleles assorted with the outside markers ro and ca of the steroca chromosome, in crossovers in the ro-ca region, was used to determine the exact position of the Lap D gene. Twelve pupae were tested individually from the mating of each testcross male carrying a recombinant chromosome. If the male's genotype was Lap $D^F$/Lap $D^S$, two bands were observed in the D zone of approximately half the pupae so analysed. If the male's genotype was Lap $D^F$/Lap $D^F$, only one band was observed in the D zone of all the pupae. In this way, sixty-five recombinant chromosomes were tested. From the data obtained, the locus of the Lap D gene was found to be $98.3 \pm 0.5$ on Chromosome III.

DL-alanyl-beta-naphthylamide-HCl (AAP) was substituted for the Lap substrate (L-leucyl-beta-naphthylamide-HCl, or LAP). When the top half of a gel was stained with the AAP substrate and the bottom half with the LAP substrate, the banding apttern was the same for both. If the gels were placed one on top of the other, the bands were superimposable. Although the enzymes produced better bands with the LAP substrate, they also showed an observable amount of activity with the AAP substrate.

Although squashes of single adults gave little, if any, activity in starch gel, 10 lambda of a concentrated homogenate of a large number of adult flies when inserted into starch gel gave a heavy banding pattern of aminopeptidases, some of which are apparently different from those observed in the pupae.

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Beckman, L. and F. M. Johnson, 1964, Hereditas 51: 221.; Wright, T. R. F., 1963, Genetics 48: 787.

Rowan, Sister M. Joan. University of Rochester. Is $Plum^K$ homologous to the mutants of the brown locus?

A mutant, $Pm^K$, which phenotypically resembles previously known Plum mutants, has been reported by Krivshenko (DIS 38:75) This mutant is associated with an inversion in 2R (proximal break at the beginning of 41 and distal break in 57 E-F). Slatis (Genetics 40:5, 1955) has described about 30 variegated position effects of the brown locus; all are due to chromosomal rearrangements involving 59D to 59F and the chromocentral heterochromatin of Y, 2, 3, or 4. However, the breakage point in the case of $Pm^K$ is definitely in the region 57C - 57F. It was thus necessary to determine whether $Pm^K$ is indeed homologous to the mutants of the brown locus or whether it is a separate mutant associated with the 57 region itself.

Initially it was found that the lethals associated with $Pm^K$ and Pm (laboratory stock) are not homologous since $Pm^K$ and Pm were viable in the homozygous condition as if they were associated with separate loci and were both present in the heterozygous condition.

If $Pm^K$ is homologous to the bw locus, it should be possible to separate it from its associated inversion. (However, the fact that the presence of the inversion may or may not be necessary for the expressivity of the Plum character must also be considered). Slatis was not able to carry out such analyses because of the close proximity of the breakage points of his variegated bw mutants to the bw locus itself.

For genetical analysis, the Pasadena stock: px slt sp (#278, DIS, 1964) was used. The location of these markers on 2R is as follows; the bw locus is also indicated as well as the distal end of the inversion associated with $Pm^K$.

| distal break | px | bw | slt | sp |
|---|---|---|---|---|
| 99-100 | 100.5 | 104.5 | 106.3 | 107.0 |

Females $Pm^K$/px slt sp were test-crossed to px slt sp males, and among 3,548 progeny were found two plexus flies and one $Pm^K$ slt sp fly (this latter female was crossed to

Canton stock males and a cytological analysis of the salivary chromosomes of her progeny revealed the presence of the $Pm^K$ inversion). The px flies were readily accounted for by assuming that $Pm^K$ is indeed associated with the distal end of the inversion (57E-F) and that a crossover occurred in the region between px and slt. It is still possible to speculate that $Pm^K$ is allelic to bw but depends upon the inversion for expressivity. In this case the crossover just cited could occur, but the px flies could in actuality be px $Pm^K$ with the $Pm^K$ not being expressed in the absence of the inversion. However, this is highly improbable in light of the fact that considerable cytological analyses of larvae have never revealed the inversion's presence without also observing $Pm^K$ flies in the progeny. Also, the $Pm^K$ slt sp fly could have been the result of a crossover in the region to the left of the bw locus, in which case $Pm^K$ could still be allelic to bw. However, further genetic and cytological analysis of crossover classes using a px bw sp stock from Le Mars, Iowa (#d15, DIS, 1964) has adequately justified the conclusion that $Pm^K$ is not homologous to the mutants at the bw locus but it represents an analogous mutant, phenotypically similar to Pm mutants and located somewhere in section 57 of 2R.

Subsequent genetic analysis involving $Pm^K$ and a Punch mutant, $Pu^2$ (Mutants of Drosophila Melanogaster; Bridges & Brehme; p 152) kindly supplied to us by Dr. D. Lindsley, has shown that $Pm^K$ and $Pu^2$ are either allelic or else they have a lethal factor in common.

Meyer, Helen U. University of Wisconsin, Madison. Use of a dominant male sterile factor in second chromosome mutation studies.

In another attempt to avoid the need for virgin females in the $F_2$ generation of second chromosome breeding procedures, a dominant male sterilizing factor in chromosome 2 is now utilized. This is a modification of a scheme previously described by Abrahamson and Meyer (DIS 40:95, 1965) in which a Minute was used to delay the eclosion of the non-wanted class of $F_2$. Now, males of this class are made sterile by being heterozygous for a dominant male sterile factor. Both schemes are simple to use, and neither requires special marker or sterilizing genes in the chromosomes to be tested for mutations.

The search for such a dominant male sterile factor in chromosome 2 was initiated by a suggestion of Dr. H. J. Muller, who also advised that such factor could then be kept in stock by balancing it with a dominant female sterile factor. Two mutations of this kind were obtained from X-rayed stage 7 oocytes having wild type (Canton-S) second chromosomes. The male sterile (Ms) is located close to, and just right of, the cn locus. The location of the female sterile (Fs) is not known. Neither has any effect on the external phenotype of the flies carrying it, except that the males heterozygous for Fs are small, much reduced in number and late hatching.

The stock ("Ms") used in this scheme has the composition S Sp Ms $bw^D/dp^{txI}$ Cy,Ins0 pr $cn^2$ sp and $Fs/dp^{txI}$ Cy,Ins0 pr $cn^2$ sp. Only the brown-eyed, Ms/Cy females and the non-brown, Fs/Cy males are fertile. Since Fs males hatch late, one should transfer this stock not too early to fresh culture bottles to keep it going. On the other hand, this shortcoming has the advantage that often only virgin females are present during the first few days of hatching.

In its simplest form, the breeding procedure is as follows: Individual $P_1$ males are crossed to brown, curly virgin females from the "Ms" stock; $P_1$ parents should be removed. In $F_1$ one crosses the non-brown, curly males (a desired number from each $P_1$ culture) individually back to brown, curly females from the "Ms" stock; removal of parents is unnecessary. In $F_2$ one selects the non-brown, curly flies for brother-sister matings; parents may again be left in. The $F_3$ is scored in the usual manner for presence or absence of non-curly homozygotes.

Should it be necessary to treat $P_1$ females and not males, and in those cases where it is important to utilize all, not only half, of the $F_1$ males produced by a treated $P_1$, one can combine this stock with Muller's method of "criss-crossed lethals" (Muller, H. J. 1953, DIS 27:104-105). For this purpose the factors S, Sp, $dp^{tx}$ and Cy are present. The procedure is the same as outlined in the above-mentioned note in DIS 40, with the only difference that the "Ms" stock is used instead of the "M" stock described there.

Berendes, H. D. Genetisch Laboratorium der Rijksuniversiteit, Leiden, Netherlands. The effect of temperature shocks in some related species of the genus Drosophila.

Temperature shocks are known to induce specific changes in the chromosomal puffing pattern of Drosophila hydei (Berendes et. al., 1965). These treatments were considered to produce a temporary shortage in oxygen in the extracellular and/or intracellular milieu. Arguments in favor of this interpretation were obtained by treatments which affect the oxygen uptake by the larvae (v. Breugel, 1965). It was assumed that the temporary shortage in oxygen acts in some way upon the activity of certain specific genes which may restore the normal metabolism which was disturbed by the treatment. The large number of related species of the repleta group, which differ in the banding pattern of their salivary gland chromosomes mainly by large paracentric inversions (Wasserman, 1962), offer favorable material to test the specificity of the genes involved in the reaction to temperature shocks.

Ten different species of the group were treated by transferring the larvae at a stage just before puparium formation from 25° to 35° C. The species used belonged to different subgroups. Five species, D. hydei, D. eohydei, D. neohydei, D. nigrohydei and D. bifurca belong to the hydei subgroup. This group was considered to have the most primitive banding sequence in the salivary gland chromosomes. Three species, D. mulleri, D. buzzatii and D. hamatofila belong to the mulleri subgroup. One species, D. mercatorum belongs to the mercatorum subgroup, and D. repleta belongs to the melanopalpa subgroup. The chromosomal rearrangements which have occurred during evolution of the repleta group were described in detail by Wasserman (1962) and they are all based on the banding sequence of D. repleta. Some of the rearrangements in a number of the species listed above were also described on the basis of the banding pattern of D. hydei (Berendes, 1965).

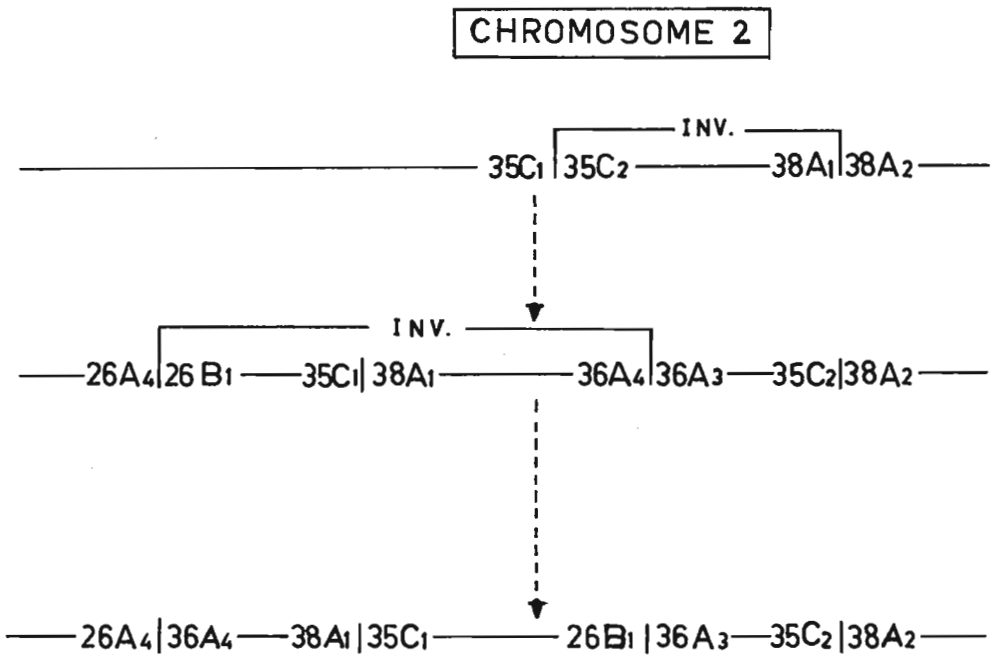
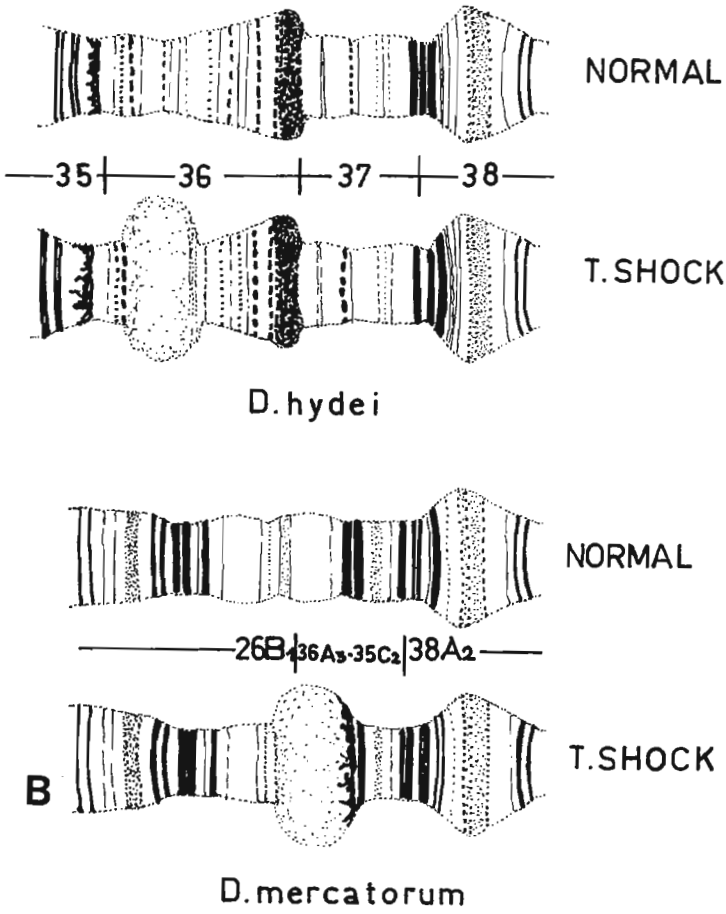



fig. 1
A

All species as far as investigated showed identical specific reactions in their puffing pattern after temperature shocks applied to larvae as well as given to salivary glands in vitro. The regions which are affected in their activity are: I 4CD, II 31C, II 32A, II 36A, II 48B, IV 81B and IV 85B.

Especially in the banding pattern of the second chromosome numerous rearrangements have occurred during evolution of the group. This has led to quite different locations of regions II 32A and II 36A in the different species as compared with D. hydei. However, the change in position of these loci, which involves the presence of different groups of genes in their neighborhood, did never change their reaction to temperature shocks. The specific reaction of the loci can even be observed when only a very small region containing such a locus is transferred to another location on the chromosome during evolution. This situation was met for region II 36A in D. mercatorum. In Fig. 1A the possible sequence




in the occurrence of two paracentric inversions in the second chromosome is shown. These inversions give rise to the banding sequence as observed in D. mercatorum when we assume that the banding sequence of D. hydei is the most primitive sequence. In addition to these two inversions there have occurred a large number of other rearrangements in this chromosome during evolution to the mercatorum sequence (Berendes, 1965). The reaction of region II 36A after a temperature shock is shown in Fig. 1B. The puffing pattern of the treated larvae of the two species can be compared with the normal state of activity in this region of the chromosome.

From the results it may be evident that the regions affected by temperature shocks are highly specific, which indicates that this treatment influences in some way a definite metabolic pathway. Moreover, the observed specificity might allow in favorable cases to conclude the homology of genes and their location in related species.

References: Berendes, H. D., Genen en Phaenen 10, 32 (1965). Berendes, H. D., F. M. A. van Breugel and Th. K. H. Holt, Chromosoma 16, 35 (1965). van Breugel, F. M. A., DIS 40, 62 (1965). Wasserman, M., Univ. Texas Publ. 6205, 63 (1962).

Duyvestyn, C. G. University of Melbourne, Australia. Wing mutant in D. robusta.

Several D. robusta lines were set up in June 1963 from pair matings taken from a single stock which had its origins in a female collected by Dr. M. Levitan at Blacksberg, Virginia in August 1962. A wing mutant was first noted in one line in October 1964 and in three other lines soon afterwards. Seven other lines as well as the parent stock have not developed the mutant.

In external phenotype, the mutant appears to be similar to the "dumpy-like" mutant reported by Levitan (DIS 26). He found the mutant character to be determined by a recessive gene and that both sexes were sterile.

The same locus was found to be involved in the mutants produced by three of the lines. The mutant from the fourth line was lost before adequate tests could be performed. The gene responsible for the abnormal wing was also found to be recessive. Mutant individuals survive for a limited time after emergence from the pupal case. Most die within a week at 25°C before reproductive activity is possible. There does not appear to be any difference in survival time at 20°C or 25°C.

Mutant females have degenerate ovaries but mutant males possess normal testes and are able to produce motile sperm if they reach sexual maturity. It is not clear whether or not they are fully fertile as copulation between mutant males and normal females was not observed nor were progeny obtained from such crosses.

Hunter, A. S. and A. Navarro. University of the Andes, Bogotá, Colombia. Ecological studies of Drosophila of Columbia.

Ecological studies of the Drosophila of Bogotá and surroundings have been carried out for several years and the results are now in press. During the last year and a half collections of the natural population of Drosophila in a limited region of Fusagasugá have been made. This is at an altitude of 1,746 meters and has an average temperature of 21.5°C which shows little or no seasonal fluctuation throughout the year. In monthly plots of the number of adults collected of each of the dominant species there are three major peaks which are similar for all the species. These three peaks in number of adult specimens collected fall 2 - 3 months after three similar peaks in the recorded rainfall of that area. In addition the weights of the collected flies have also been recorded and in some cases it is significantly higher for the flies collected during the "rainy" months than for those collected during the "dry" months.

The presence of cyclical fluctuations in the number of Drosophila collected is in agreement with the data of the Bogotá region. It is concluded that the amount of rainfall affects the Drosophila population in number and size of adults.

Shima, T. Hokkaido University, Japan. Notes on the copulation, insemination reaction and sperm storage of D. nigromaculata in homogamic matings.

The first mating of the virgin female and male of this species was observed during the period from the 5th day to the 10th day after the emergence at 20°C, and most of the females were mated on the 6th day. Copulation times were recorded for 100 pairs of flies, and the average time was 6 minutes and 11 seconds (ranged 1 min. 10 sec. - 8 min. 24 sec.). The reproductive organ was dissected out under a binocular microscope in a saline solution. A total of 962 females was dissected at various times, starting immediately after copulation and extending through about 40 days. At the 2-minute dissections the uterus was enlarged and contained many sperm in the small granular, opaque reaction mass. Some sperm had already entered the ventral receptacle. At the 15-minute period there was a small amount of reaction mass in the uterus, and a few motile sperm in the ventral receptacle and spermathecae. At the one-hour period, the reaction mass grew up into maximum size. Then the reaction mass disappeared and the uterus had returned to its normal condition by 48 hours. Motile sperm occurred in the ventral receptacle from the beginning of the dissection to 360 hours. The spermathecae had motile sperm from the 5-minute dissection to 450 hours. After the first mating a female was able to produce fertile eggs for about 20 days.

Strangio, V. A. University of Melbourne, Australia. Germ cell sensitivity in the irradiated adult and pre-adult testes.

D. melanogaster males were irradiated with 800r X-rays as: larvae aged 30, 50, 65, 80, 92 hours after eclosion; pupae 0, 6, 24, 48, 72 hours after puparium formation or adult males 4 hours old. The comparative testis histology of these forms was also determined. After the males had matured sexually, the usual experimental and breeding procedures were carried out. Successive brood frequencies were obtained for certain exceptional forms as listed in a previous publication (1961). First-brood frequencies only for sex chromosome loss, Y marker deletion, induced X-Y exchange and non-disjunction as well as for egg non-hatchability are shown in the accompanying table. Note that the frequencies in the first two rows have been corrected for contributions from induced X-Y non-disjunction or exchange. Hypersensitivity to loss aberrations is apparently associated with germ cell types present in the posterior region of the testes about or following puparium formation, i.e. cells in late prophase or metaphase of meiosis I. There is some evidence to suggest that the peak and exceptionally variable egg non-hatchability in the late larval forms may be partly due to inadequate egg fertilization resulting from sperm shortage superimposed on the usual dominant lethality component, i.e. young primary spermatocytes are extremely susceptible to radio-destruction in situ (see also Riemann 1964).

Table 1: First brood frequencies after irradiation of adult (A), pupal (P) and larval (L) males aged as indicated.

| Male Form | Control | A4 | P72 | P48 | P24 | P6 | PO | L92 | L80 | L65 | L50 | L30 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Sex chromosome loss | 0.0461 | 0.1002 | 0.2201 | 0.4359 | 0.7938 | 0.8900 | 1.3152 | 0.8116 | 0.2240 | 0.0658 | 0.0000 | 0.0000 |
| $B^S$ Y-marker deletion | 0.0077 | 0.0351 | 0.0629 | 0.4722 | 0.5531 | 0.7734 | 1.3746 | 0.9434 | 0.7268 | 0.2521 | 0.0447 | 0.1211 |
| X-Y exchange | 0.0077 | 0.0091 | 0.0000 | 0.0436 | 0.0000 | 0.3179 | 0.3436 | 0.7383 | 0.6886 | 0.0658 | 0.3167 | 0.0427 |
| X-Y non-disjunction | 0.0231 | 0.0091 | 0.0000 | 0.0000 | 0.0346 | 0.0000 | 0.0573 | 0.2461 | 0.1530 | 0.0000 | 0.0975 | 0.0285 |
| Egg non-hatchability | 5.29 ± 0.62 | -- | 19.27 ± 1.33 | 45.50 ± 1.40 | 47.08 ± 1.28 | 52.24 ± 2.02 | 56.81 ± 1.83 | 51.66 ± 4.20 | 54.65 ± 5.70 | 48.82 ± 13.18 | 12.07 ± 1.50 | 8.24 ± 1.25 |

Giesel, Betty Jean. University of Oregon, Eugene. Structure of $In(1)X^{c2}$, $w^{vc}$.

Original observations of the mitotically unstable ring chromosome $In(1)X^{c2}$, $w^{vc}$ showed that variegation occurred at the white locus and that there was a spreading effect to the $rst^+$, $spl^+$, and $_+N^+$ loci. Under these conditions, an inversion of the $X^{c2}$ chromosome due to breaks between $y^+$ and $w^+$, and in the heterochromatin between $car^+$ and the centromere would explain the observed variegation (Catcheside and Lea, 1945, Hinton, 1955).

When $In(1)X^{c2}$, $w^{vc}$ was irradiated in the course of another experiment, several products were obtained which suggest that the hypothesized structure may be incorrect.

The recovery of two chromosomes, both showing deletions of both $w^+$ and $car^+$, and of a fragment containing $y^+$ and $w^{vc}$ suggests that the inversion may actually involve breaks between $w^+$ and $rst^+$ and in the heterochromatin adjacent to $car^+$.

| | Deleted Loci | Loci Shown to be Present |
|---|---|---|
| deleted rings | $w^{vc}_+$, $car^+$ | $y^+_+$, $m^+_{vc}$, $sn^+$, $ac^+$, $sc^+$, $pn^+$ |
| fragment | $m^+$, $car^+$ | $y^+$, $w^{vc}$ |

Hendrickson, R. J. University of California, Los Angeles. Cytogenetic evidence bearing on non-polarization of the dumpy nest of pseudoalleles.

Salivary gland chromosomes have been studied in the "dumpy-warped" ($dp^w$) mutants (Carlson DIS 32; Carlson and Schalet DIS 30), all of which manifest a variegated position effect phenotype. In addition to four stocks remaining from earlier work with X-rays ($dp^{w1}$, $dp^{w2}$, $dp^{w3}$, $dp^{w7}$), three additional mutations ($dp^{w8}$, $dp^{w10}$, $dp^{w11}$) have been obtained from cobalt irradiation ($Co^{60}$, 4,500r). Each of them is the result of a major chromosomal rearrangement, these being identified in the table at the end of this note. In all cases one break is in 2L and is located either between bands 24F8 and 25A1 or to the right of 25A1 within the "shoebuckle set of four bands", 25A1-4 (cf., Bridges' maps, 1935 and 1943). When in compound with $dp^{ov1}$, these mutations fall into two phenotypic classes which can be designated "moderate" and "strong". The former yields a certain proportion of flies having a wild phenotype, or nearly so, with most of the remainder being markedly asymmetric for mild-to-moderate oblique and vortex effects. There is a dissociation of these two effects with respect to their degree of expression and the side on which they are most strongly manifested in individual flies. "Strong" mutant stocks on the other hand produce no flies having a wild phenotype, and the oblique and vortex effects, while still being asymmetrical, are much more extreme in their manifestation, tending toward an extreme $dp^{ov}$ as a limit. In some cases, for example, wings are seen having the "charred" appearance of "dumpy-truncate" ($dp^{olv}$) mutants. In no case is leg morphology affected.

The differentiation of classes was first noted as the result of a selection against apparent modification of the mutant phenotype due to continued inbreeding in three of the mutant stocks ($dp^{w1}$, $dp^{w3}$ and $dp^{w8}$). In each case, males showing the strongest and most asymmetrical phenotypes were selected and mated to homozygous ed $dp^{ov1}$ cl virgin females from our standard tester stock. $dp^{w1}$ and $dp^{w3}$ responded positively to this selection, showing, after several generations, a marked increase in degree of mutant expression, while $dp^{w8}$ showed no such response. Recently, another moderate mutant, $dp^{w11}$, has been obtained.

The fact that in the mutants $dp^{w3}$ and $dp^{w11}$ cytologically identical break points have yielded mutants having quantitatively different degrees of expression, while in $dp^{w8}$ and $dp^{w11}$ different break points yield quantitatively similar degrees of expression, seems to indicate an absence of polarity for the dumpy nest of pseudoalleles. While the possibility exists that the 3R heterochromatin is differentiated with respect to variegation-induction as is known to be the case for the heterochromatic portion of X, this would not seem to lessen the argument materially.

Pertinent information regarding these mutant stocks is summarized below. Cytology in all cases has been on heterozygotes of the genotype $dp^w$/ed $dp^{ov}$ cl. Homozygous stocks are being synthesized and will be examined when obtained.

| Stock | Alternate Designation | Degree of Expression | Rearrangement | Break Pt(s) |
|---|---|---|---|---|
| $dp^{w1}$ | T(2:3) $dp^{w1}$ | Strong | 2:3R (entire) Reciprocal translocation | to right of 25A1; 3R heterochromatin |
| $dp^{w2}$ | T(Y:2) $dp^{w2}$ | (Stock lost - being replaced) | | |
| $dp^{w3}$ | T(2:3) $dp^{w3}$ | Strong | $2^{L}$:3R (entire) Reciprocal translocation | Between 24F8 and 25A1; 3R heterochromatin |
| $dp^{w4}$ to $dp^{w6}$ | lost | | | |
| $dp^{w7}$ | T(Y:2) $dp^{w7}$ | Strong | $2^{L}$:Y (arm not known) | Between 24F8 and 25A1 |
| $dp^{w8}$ | T(2:3) $dp^{w8}$ | Moderate | 2:3R (entire) Reciprocal translocation | To right of 25A1 3R heterochromatin |
| $dp^{w9}$ | Lost | | | |
| $ep^{w10}$ | In(2L(R?)) $dp^{w10}$ | Strong | Inversion of 2L (including centromere?) | Left: to right of 25A1; Right: either 2L or 2R heterochromatin |
| $dp^{w11}$ | T(2:3) $dp^{w11}$ | Moderate | 2L:3R (entire) Reciprocal translocation | Between 24F8 and 25A1; 3R heterochromatin |

Jungen, H. University of Zürich, Switzerland. Chromosomal polymorphism in a natural population of D. subobscura from Tunis.

In the spring of 1965 a sample of D. subobscura was caught near Tunis. The following structural types were observed (number of chromosomes in parenthesis): chromosome A, St(6), 2(10), 1+2(22), 1+2+3 (18); chromosome I, St(2), 1(98); chromosome E, St(4), 1+2(71), 1+2+9(4), 1+2+9+3(1), 1+2+9+4 (16), 1+2+9+12(4); chromosome U, St(1), 1+2(20), 1+2+3(1), 1+2+8(78); chromosome O, St(1), 3+4 (5), 3+4+6(2), 3+4+7(2), 3+4+8(80). The data refer to 56 A-chromosomes and 100 of each autosome. In the O-chromosome, two unknown structural types were present (10). The structural types A1+2, A1+2+3, and E1+2+9+4 were recently reported by W. Götz in Z. Vererbungsl. 96: 285-296 (1965), from a Marocan population.

Popper, Joan. University of Oregon, Eugene. X-autosome translocations in a sex-ratio strain of D. pseudoobscura.

It has been reported by Novitski and Ehrlich (Drosophila Research Conference, Seattle: see this volume) that chromosomal rearrangements in cells carrying homologs showing meiotic drive, can appreciably alter and even reverse the drive. In this connection, it should be noted that an array of translocations involving the sex ratio X-chromosome and the third chromosome of D. pseudoobscura may alter the amount of drive from 99% recovery of the X at one extreme, to 60% at the other. The relationship between the breakpoints of the translocations and the degree of modification of the drive is under investigation.

Makino, S., E. Momma and A. Kaneko.
Hokkaido University, Sapporo, Japan.

Collection records of drosophilid flies from the three islands of Northern Hokkaido.

Collections were made in August, 1965, in the three small islands located at Japan Sea apart from Northern Hokkaido: Yagishiri Is., Rishiri Is. and Rebun Is. Most of the flies were collected by the use of fermented banana, at one hour intervals, from sunrise to sunset for three successive days. A total of 2440 flies were represented by 5 genera, 24 species, as given in the accompanying table. Amiota dispina was noted as a new member of the Drosophila fauna in Hokkaido. Abundant species were D. lacertosa in Yagishiri Is., D. testacea and D. bifasciata in Rishiri Is. and D. nigromaculata and D. lacertosa in Rebun Is., respectively. Remarkable was the fact that five species of robusta group (6 species known in Japan) were collected in Rebun Is., most northerly of the three islands.

Table 1. Numerical data of drosophilid flies collected in the three islands of Northern Hokkaido, August, 1965.

| Species | Yagishiri Is. | Rishiri Is. | Rebun Is.* | Total |
|---|---|---|---|---|
| Amiota alboguttata f. furcata | - | 1 | - | 1 |
| Amiota dispina | - | 1 | - | 1 |
| Microdrosophila cristata | - | - | 3 ( 3) | 3 |
| Leucophenga quinquemaculipennis | - | 4 | - | 4 |
| Scaptomyza pallida | - | 2 | 75 (69) | 77 |
| Scaptomyza apicalis | - | - | 4 ( 4) | 4 |
| Scaptomyza okadai** | - | - | 15 (12) | 15 |
| Drosophila histrioides | 5 | 8 | - | 13 |
| Drosophila coracina | 2 | 49 | - | 51 |
| Drosophila bifasciata | 16 | 376 | 40 | 432 |
| Drosophila suzukii | - | 4 | - | 4 |
| Drosophila lutea | - | 6 | - | 6 |
| Drosophila auraria race A | 16 | 7 | 97 | 120 |
| Drosophila brachynephros | 2 | - | - | 2 |
| Drosophila unispina | 4 | - | 12 | 16 |
| Drosophila nigromaculata | 40 | 71 | 224 (18) | 335 |
| Drosophila testacea | 93 | 535 | 108 | 736 |
| Drosophila histrio | - | 3 | - | 3 |
| Drosophila tenuicauda | - | - | 17 (17) | 17 |
| Drosophila lacertosa | 188 | 142 | 149 | 479 |
| Drosophila moriwakii | - | 35 | 23 | 58 |
| Drosophila okadai | 2 | - | 33 | 35 |
| Drosophila sp. like okadai*** | 4 | - | 16 | 20 |
| Drosophila pseudosordidula | - | - | 8 | 8 |
| | 372 | 1244 | 824 (123) | 2440 |

* The numericals in the parentheses denote the number of specimens collected with the use of net sweeping.

** Scaptomyza okadai has been reported in Japan as S. unipunctum before.

*** Drosophila sp like okadai was first found at Toya, May, 1964, as a new species of the sixth member of robusta group in Japan, and will have been reported as a new name, Drosophila neokadai, on the Annotationes Zoologicae Japonenses, Vol. 39, No. 1 (in press).

Frye, Sara H. P.O. Box 267, Irvine, Kentucky. Open letter to Drosophila geneticists pertaining to the simultaneous occurrence of a double marker mutant in Drosophila melanogaster.

On April 24, 1965, a single exceptional male, Y/y ac In49 $B^{M1}$*, was recovered among progeny whose origin were parents consisting of relatively young males of genotype, Y/y $ac^+$ In49 $B^{M1}$, which had been exposed to 2 kr (i.e., 2000 r delivered at 400 r/minute, 210 kvp., 15 ma., 1mm Al + 1/2 mm Cu filter) and mated to a series of virgin females of different genotypes, one of which was Y/y $f^+$ virgin females (i.e., phenotypically yellow wings, yellow body color, yellow-two (?) bristles, and forked bristles). Simultaneous and identical controls did not yield any exceptional $F_1$ fertile yellow and/or achaete males even though the same precautions were used to avoid environmental selection against any exceptional phenotype of spontaneous origin occurring among the expected phenotypes.

In49 $B^{M1}$ chromosomes are representative of chromosomes of normal structure for the yellow region, i.e., there is no chromocentral heterochromatin adjacent to the yellow region (as in scute-8 chromosomes) and $B^{M1}$ chromosomes were used by Belgovsky for this reason. In my opinion, yellow mutants induced in $B^{M1}$ chromosomes are a better test in regards to the problem as to whether two or more closely linked markers are ever involved in mutational events induced by X-irradiation than yellow mutants induced in scute-19i chromosomes. Scute-19i chromosomes contain an insertion from the distal tip of the X-chromosome bearing the normal alleles of the mutant markers yellow, achaete, scute, and scute-lethal into the second chromosome between the normal alleles of the mutant markers, dumpy (2, 11.0) and clot (2, 16.5). Consequently, the yellow region is now free of chromocentral heterochromatin. As to the presence of intercalary heterochromatin in the neighborhood of the yellow region in scute-19i chromosomes or In49 $B^{M1}$ Chromosomes I am not qualified to give an opinion (see Prokofyeva-Belgovskaya papers between 1930-1939). I sincerely hope that several (not just one) investigators will examine this stock cytologically (see Frye, materials available, this issue, DIS 41) and report their findings to the other Drosophila workers at the 8th annual Drosophila Conference which meets in Chicago, May 27-29, 1966.

* y ac In49 $B^{M1}$ chromosome is hereafter designated as y ac In49 $B^{M1}$ to indicate the simultaneous occurrence of the double marker mutant phenotype (see Frye, new mutants, this issue DIS).

I proceeded with a reverse mutant phenotype experiment by irradiating young males of genotype, $sc^8$ Y/y ac In49 $B^{M1}$ at 2 kr or 4 kr and mating them to Y/y $f^+$ virgin females and scoring for non-yellow (symbolized $y^+$) and/or non-achaete (symbolized $ac^+$) male-viable phenotypes. Between August 31, 1965 and September 8, 1965 I recovered some non-achaete ($ac^+$) phenotypes, but I did not recover any non-yellow ($y^+$) phenotypes or any non-yellow, non-achaete ($y^+$ $ac^+$) phenotypes. Later observations made in the author's office in Irvine, Kentucky showed that some of the non-achaete phenotypes ($ac^+$) were fertile on progeny-testing and that several forward mutant phenotypes* had been superimposed in the irradiated y ac In49 $B^{M1}$ chromosomes.

The pattern of investigation (multiple genetic perspectives) employed in approaching the problem of structure and function of the yellow-achaete region within the framework of a replicating, functioning chromosome is considered to be of utmost experimental and interpretative value.

I acknowledge with great pleasure the assistance in much of the routine work by my son, Mark Evan Frye. I want to thank Dr. Burke Judd for space in his laboratory in the Genetics Foundation at the University of Texas, Austin, Texas. I would like to thank Drs. Faberge (1962) and Schultz (1963) for emphasizing in written communication to the author the importance of the genetic scheme and the structure of the chromosome used in the recovery of yellow marker mutants.

* These mutants will be printed in the next issue of DIS.

Strangio, V. A. University of Melbourne, Australia. Brood sensitivity patterns after the irradiation of males bearing a rod, ring or inverted-X and a doubly marked Y-chromosome.

Males bearing either a rod (R), ring ($X^{C2}$) or inverted (M5) X-chromosome and a doubly marked Y-chromosome ($B^{s}$ Y $y^{+}$) were irradiated with 800 r X-rays. Experimental and breeding protocols were as published previously (1961, 1962). Partial or complete sex chromosome loss, induced X-Y exchange and/or non-disjunction were recorded. The accompanying table shows the daily brood sensitivity patterns for some of these aberrations. In general, the sex chromosome loss patterns are comparable except for the markedly increased amplitude found in the ring-X series as expected and as previously noted by Sobels (1963). An unexpectedly high recovery of yellow-Bar females, i.e. recombinant X's from induced exchange between the inverted-X (a Barless Muller-5 chromosome) and $B^{s}$ Y $y^{+}$ during a study of spermatogenic sensitivity to the induction of subterminal deletions (Lüning 1954) led to a re-appraisal of supposed non-disjunctional exceptionals from rod-X experiments (Strangio 1961), confirming studies independently undertaken by Zimmering and Wu (1964). Induced X-Y exchange is predominant over non-disjunction in both rod and inverted X experiments. This is not immediately apparent in the results given for the inverted-X which have not been adjusted here for a relatively inflated spontaneous rate of primary non-disjunction. However, the position is definitely reversed for the ring X. The dicentric configuration produced after exchange between the ring X and Y probably accounts for this situation. However, a rare rupture of this dicentric in a heterochromatic region may sometimes be followed by healing and the recovery of a monocentric recombinant X.

Table 1: Brood frequencies after the irradiation of rod (R), ring ($X^{C2}$) and inverted (M5) X chromosomes.

| BROOD | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 |
|---|---|---|---|---|---|---|---|---|---|---|
| Sex chromosome loss | R | 0.1187 | 0.0634 | 0.1441 | 0.3297 | 0.5780 | 1.0419 | 1.6765 | 1.4657 | 0.2577 |
| | $X^{C2}$ | | | 1.3390 | 1.6320 | 1.7707 | 4.1941 | 6.3018 | 3.4010 | 0.9949 |
| | M5 | | | | 0.7558 | 0.9196 | 1.2935 | 1.4597 | 1.3229 | 0.6394 |
| X-Y exchange | R | 0.0091 | 0.0000 | 0.0000 | 0.0094 | 0.0000 | 0.2368 | 0.6343 | 0.4728 | 0.0537 |
| | $X^{C2}$ | | | 0.0000 | 0.0000 | 0.0000 | 0.0200 | 0.0237 | 0.0597 | 0.0362 |
| | M5 | | | | 0.0000 | 0.0000 | 0.1866 | 0.2567 | 0.2970 | 0.0246 |
| X-Y non-disjunction | R | 0.0091 | 0.0181 | 0.0303 | 0.0188 | 0.0246 | 0.0710 | 0.1812 | 0.0946 | 0.0215 |
| | $X^{C2}$ | | | 0.1708 | 0.1533 | 0.1005 | 0.3395 | 0.4264 | 0.2983 | 0.1447 |
| | M5 | | | | 0.1425 | 0.2362 | 0.2574 | 0.3770 | 0.3510 | 0.3050 |

Hosgood, Sally W. M. and P. A. Parsons. University of Melbourne. Differences between D. simulans and D. melanogaster in tolerances to laboratory temperatures.

Four strains of D. melanogaster and three of D. simulans were collected in Victoria, Australia, and set up at 29.5°, 27.5°, 25°, 20° and 15°C. It was found that after 5 generations, all strains of D. melanogaster were living at all temperatures. However, at this stage the three strains of D. simulans were living at 20° and one at 25°. At 29.5° and 15° all the D. simulans strains had died out by the second generation, and at 27.5° by the third generation.

Thus D. simulans is much more restricted in its tolerance to diverse temperatures than D. melanogaster. This distinction may help to explain distribution differences in the two species. At first sight, therefore, D. melanogaster is much more versatile ecologically than D. simulans in Victoria.

Aubele, Audrey M. and H. L. Plaine. Ohio State University, Columbus. Occurrence of the erupt effect in the al b c $sp^2$ strain of D. melanogaster.

Previous experiments (Plaine, H. L. DIS 40: 56) with eggs and larvae of the Su-er tu bw;$er^+$ Su-$tu^+$ strain gave the phenotype characteristic of extreme erupt after X-irradiation in air. Since, theoretically, this strain possesses the wild type allele of erupt ($er^+$), further studies as to the nature of this response have been conducted.

This laboratory strain (Su-er tu bw;$er^+$ Su-$tu^+$) had been derived originally by substitution of the second chromosome of the Suppressor-erupt or bw st strain which had the highest frequency of the erupt phenotype after X-irradiation, and the third chromosome of the al b c $sp^2$ strain which did not express the erupt phenotype after X-irradiation in air. Series of eggs and larvae from both of these parent strains were exposed to X-irradiation according to the methods described in the previous report. Since an atmosphere of $O_2$ had been shown to increase the frequency of the erupt phenotype when used in conjunction with X-irradiation, the Su-er;$er^+$ and the al b c $sp^2$ strains were also exposed to X-irradiation in an atmosphere of 100% $O_2$.

The phenotype characteristic of extreme erupt was found in all irradiated series (Table 1). All series subjected to X-irradiation differed significantly from the non-irradiated controls of the same strain. The frequencies of extreme and total erupt after X-irradiation also differed significantly among the strains tested.

These results indicate that the suppressor-erupt system is present in the al b c $sp^2$ strain. Failure to detect the suppressor-erupt system in this strain is highly probable if a small number of flies is examined or if special techniques for enhancement of the erupt response, such as an atmosphere of $O_2$, are not used. Studies of the relationship of the suppressor of erupt and erupt alleles present in the al b c $sp^2$ with respect to those present in the Suppressor-erupt or bw st strain are now in progress.

While the results obtained in this present study confirm the presence of the suppressor-erupt system in the al b c $sp^2$ strain, there is no indication that this expression is due to the direct enhancement of the mutant gene rather than to inhibition of the suppressor. If this were the case, we would expect the response in both the Su-er; $er^+$ and the al b c $sp^2$ strains to be equivalent, since both have the same erupt allele. It seems more probable that both loci are involved to some extent in the response. Further studies to clarify the relationship of the erupt locus and its specific suppressor locus in the response of the suppressor-erupt system to X-irradiation are now in progress.

Table 1: Frequency of erupt eye in irradiated and non-irradiated series of the Su-er;$er^+$, al b c $sp^2$, and Suppressor-erupt or bw st strains.

| Treatment | Total counted | | | Phenotype of eyes (%) Normal | | | Extreme erupt | | | Total erupt | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Treatment | (1) | (2) | (3) | (1) | (2) | (3) | (1) | (2) | (3) | (1) | (2) | (3) |
| X-ray; Air | 1268 | 1027<br>594<br>1087 | 729<br>325 | 81.2 | 74.6<br>74.1<br>75.8 | 7.7<br>26.2 | 1.8 | 5.4<br>5.2<br>6.2 | 65.0 | 18.8 | 25.4<br>25.9<br>24.2 | 92.3 |
| X-ray; 100% $O_2$ | 1509 | 1129 | --- | 67.8 | 42.0 | ---- | 5.3 | 28.7 | ---- | 32.2 | 58.0 | ---- |
| Non-irradiated Controls | 1250 | 1511 | 1010 | 99.4 | 99.7 | 93.9 | 0.0 | 0.0 | 0.09 | 0.6 | 0.3 | 6.13 |

(1) al b c $sp^2$ (2) Su-er tu bw;$er^+$ Su-$tu^+$ (3) Suppressor-erupt or bw st

Himoe, E. and L. Lowenstein. University of Wisconsin, Madison. Spontaneous sex-linked recessive lethals in D. melanogaster.

A series of spontaneous sex-linked recessive lethals have been recovered from a suspected "mutater" system, which unfortunately could not be maintained. These lethals have been approximately localized, using an X-chromosome containing the markers y, cv, m, f, and car. No cytological analyses have been performed yet, but genetic analysis shows no obvious aberrations associated with any of them. The following is a list of these lethals by region, and their approximate positions within the regions, based on total counts of roughly 200 to 300 male progeny for each. Stocks of these lethals may be obtained from the Department of Zoology, University of Wisconsin, if anyone wishes them for further study.

| Region | Lethal no. | Approximate location |
|---|---|---|
| 0 (no recombinants recovered between y and lethal) | 1L1 | covered by $sc^8$•Y |
| | 14L1 | not covered by $sc^8$•Y |
| | 27L1 | not covered by $sc^8$Y |
| | N31 | not tested with $sc^8$•Y |
| 1 (y - cv) | 23L2 | 1.4 |
| | 30L1 | 1.6 |
| | 32L1 | 2.3 |
| | 33L3 | 2.6 |
| | 29L2 | 4.0 |
| | 31L2 | 13.5 |
| 2 (cv - m) | 5L1 | 16.3 |
| | 14L2 | 19.7 |
| | 31L1 | 20.6 |
| | 15L1 | 21.0 |
| | 4L1 | 21.5 |
| | 31L3 | 22.8 |
| | 3L3 | 23.2 |
| | 28L2 | 26.1 |
| | 33L2 | 28.0 |
| | 25L1 | 32.6 |
| | 23L3 | 32.7 |
| | N28 | 34.2 |
| 3 (m - f) | 13L1 | 37.4 |
| | 28L1 | 44.8 |
| | 9L1 | 47.3 |
| | 12L1 | 48.5 |
| | 27L2 | 48.7 |
| | 11L1 | 49.2 |
| | 23L1 | 51.7 |
| | N15 | 52.4 |
| | 33L1 | 52.8 |
| 4 (f - car) | 28L3 | 62.1 |
| no recombinants recovered between car and lethal | 10L1 | |
| | N30 | |
| | N5 | |

Seeley, A. A., J. B. Peterson and M. H. Smoler. University of Wisconsin, Madison. Relative biological effectiveness of X-ray and gamma radiation.

The purpose of the experiment was to compare the relative biological effectiveness of 140 kvp X-rays to gamma rays delivered from a Cesium 137 source. The frequency of induced sex linked recessive lethals in Drosophila was observed using the standard methods. Dose rate measurements were made using a high intensity Victoreen ionization chamber for Cesium and a regular ionization chamber for X-ray. Four series of tests were made using P1 Canton S males; in series one, the males were given 3200 r X-rays at a dose rate of 234 r/min; series two males were given an intense dose of 3200 r gamma at a dose of 466 r/min; series three males were given a dilute dose of 3200 r gamma at a dose rate of 6.9 r/min; series four was a control. Results showed a very significant difference in mutation induction between X-rays and both series of gamma, but no significant difference between the two gamma treated series. These results confirm an earlier study by Edington (1956, Genetics 41:814-821) on cobalt and X-rays, which showed a RBE of approximately 1.6.

| Series | No. lethals/No. tests | % lethals ± % S.E. | Significance Test |
|---|---|---|---|
| 1) X-ray | 85/822 | 10.34% ± 1.065 | Series 1 vs. 2 & 3 p<.001 |
| 2) Gamma dilute | 55/974 | 5.65% ± o.74 | Series 2 vs. 3 p=0.17 |
| 3) Gamma intense | 42/969 | 4.33% ± 0.655 | |
| 4) Control | 0/139 | | |

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

Twenty second chromosomes with at least one recessive lethal were extracted from the natural population of D. melanogaster. Double, triple and quadruple lethals were accumulated in cis-phase on second chromosomes through recombination of females with two or more lethals in trans-phase. Preadult viability of wild phenotype flies were determined by the Cy-Pm technique. Following results are obtained:

| No. of lethals per zygote | No. of crosses | Preadult viability (Cy/Pm = 1.0000) |
|---|---|---|
| 0 | 55 | 1.0281 ± .0116 |
| 1 | 33 | 1.0147 ± .0239 |
| 2 | 65 | .9782 ± .0185 |
| 3 | 57 | .9524 ± .0269 |
| 4 | 45 | .9170 ± .0223 |
| 5 | 37 | .8391 ± .0365 |
| 6 | 12 | .8092 ± .0664 |
| 7 | 7 | .7367 ± .1078 |

This synergistic interaction between lethals compared to induced ones is very relevent to the problem of the maintainance of genetic load in natural populations.

Burckhardt, B. and E. Hadorn. Zoologisches Institut der Universität, Zürich, Switzerland. The ductus ejaculatorius of Drosophila melanogaster as a test object for physiological media.

Genital apparatuses of young adult males have been prepared free and immersed in different salt solutions. Quality and persistence of movement of the ductus ejaculatorius are used as the criteria of the quality of media.

Our table shows how long "normal" and declining movements are maintained in vitro in a few of the tested solutions (room temperature). Solution component quantities are given for 1000 cc aqua bidest.

| Medium | | Movement "normal" | Movement declining |
|---|---|---|---|
| 1. Holtfreter: | NaCl 3,5 g<br>$CaCl_2$ ($2 H_2O$) 0.066 g<br>KCl 0.05 g<br>$NaHCo_3$ 0.2 g | 0 - 15 Min. | until 10 h |
| 2. Insect Ringer: | NaCl 7.5 g<br>KCl 0.287 g<br>$CaCl_2$ ($2H_2O$) 0.287 g | 0 - 60 Min. | until 29 h |
| 3. NaCl: | 1 - 3 g | | - - - |
| | 4 - 8 g | | 10 - 14 Min. |
| | 9 - 10 g | | 7 - 9 Min. |
| | 11 - 15 g | | 1 - 3 Min. |
| | 16 - 20 g | | - - - |

Among many others of the tested combinations, "Insect Ringer" proved to be the best medium.

Brändle, E. and E. Hadorn. Zoologisches Institut der Universität, Zürich, Switzerland. Cell numbers in adult corpora allata of Drosophila melanogaster.

Corpora allata were fixed in alcohol-acetic acid and stained in toto with gentian-violet. Numbers of cells were determined for glands of inseminated females, virgin females and males at 1, 2, 4 and 8 days after hatching of the adults. In each of the 12 sex-age groups, 4-12 counts were made. No significant differences between sexes or age-groups were found. The numbers varied between 12 and 22. Mean: $15.31 \pm 0.189$.

Ditman, W. F. Purdue University. Observation of D. melanogaster behavior.

The following observations were made while developing an apparatus and an experimental procedure for testing depth perception preference in D. melanogaster. General testing procedure: Flies were released into the apparatus, 50 at a time from a clean, half-pint culture bottle. They were given 30 seconds to respond to the visual stimuli. Then the trap doors of the collecting chambers were closed and the flies were removed with an aspirator and counted. For details of the apparatus and procedure see: An Improved Method for Determining Visual Depth Preference in Large Numbers of D. melanogaster, this newsletter.

Experiment One: Effects of Ether on Flying Behavior

Flies used were wild stock (CS-200), kindly supplied by Dr. A. B. Burdick. The apparatus was specifically designed to admit light only through the ceiling. Yet, despite their positive phototropism and negative geotropism, less than 50 percent of the flies could be induced to ascend the inside (27") of the box to the ceiling. During preliminary work on depth preferences, the flies were etherized for counting out lots of 50.

A review of the preliminary data collected suggested that etherizing affected the flying behavior of D. melanogaster. In a test of this hypothesis, 400 flies were divided into groups of 50. Counting for this and all subsequent experiments was done by letting flies crawl singly through a narrow tube into half-pint bottles. The eight bottles were then arranged into an ABBA ABBA order in which all "B"s were etherized very lightly. The flies were tested in the same counterbalanced order six hours later.

Table 1

| Group | No. Released | No. Flew | % Flew | $X^2$ Obs. |
|---|---|---|---|---|
| Etherized | 197+ | 86 | 45.6 | 10.62** |
| Un-Etherized | 206+ | 158 | 76.7 | |

+No. deviates from 200 because of miscount in dividing flies into groups of 50 each.
**significant at the .01 level of probability.

The Chi Square value of 10.62 is significant at the .01 level of probability, indicating a difference in the behavior of etherized and un-etherized flies.

Conclusion: Flies that have been etherized even lightly and then given adequate time to recover show a significantly lower propensity for flying when given the opportunity than un-etherized flies. This phenomenon may be readily observed by placing an un-etherized group of flies into a clean dry culture bottle and an etherized group into a second bottle. After the flies have had time to recover the un-etherized flies will be observed crawling about their bottle with greater vigor than those in the other group. If the lids are removed more of the un-etherized flies will escape.

Curiously, it was noted that at certain times the drawing of the hand down along the side of the culture bottle stimulated the flies to rush up the inside of their bottle. When the hand was drawn up the sides of the bottle, the flies would circle the inside of their bottle. This phenomenon is most pronounced when flies are used which previously had been lightly etherized. Flies that had been heavily etherized were sluggish and did not respond. Un-etherized flies, on the other hand, were so responsive that almost any stimulus caused

them to rush up the side of their bottle. This may be a photokinetic effect but the circling behavior when the hand moves up seems unusual.

Experiment Two: Effects of Flight Distance on Flying Behavior and Depth Preference

Flies used in this experiment were wild D. melanogaster collected at a local fruit stand. Two main problems developed during the course of this experiment: (1) how to get more flies to fly and thus exhibit a preference, and (2) how to reduce the incidence of random choices and thereby achieve a better index of preference for one or the other of the two visual ceilings if a preference does exist. It is assumed that if there is no preference equal numbers will fly to the two stimulus ceilings. If the flies do have a preference the proportion of flies on the preferred slide should reflect the strength of that preference.

Since the distance of flight was hypothesized to be an important variable, a short flight distance (3"), and a long flight distance (49") were compared to the standard (27") by altering the height of the flight shaft accordingly. All other factors in this comparison were held constant.

Table 2

| Flight Distance | No. Released | No. Flew | % Flew | No. High | % High | $X^2$ Obs. (P = .5) |
|---|---|---|---|---|---|---|
| 3" | 500 | 418 | 80.4 | 206 | 49.5 | .09 |
| 27" | 339+ | 284 | 83.7 | 175 | 61.6 | 15.33** |
| 49" | 500 | 322 | 64.4 | 182 | 56.5 | 5.48* |

+should have been 350, difference due to miscount in dividing flies into groups of 50 each.
*significant at the .05 level of probability.
**significant at the .01 level of probability.

In Table 2 all observed response proportions were tested against the null hypothesis P = .5 to each of the two stimuli.

Under the 3" flight distance conditions the flies apparently distributed themselves in a random fashion. On the other hand, over a flight distance of 27" flies clearly appeared to prefer the high side. Less preference for the high side was observed when the flight distance was 49". The results for the short (3") flight distance seem relevant to the work of Lewontin (1959) who studied the negative phototropic response of D. pseudoobscura. He found that this tropism only occurred in his apparatus, a bell jar, when the flies walked rather than flew. When they flew their choices appeared random. Fifty percent went to the dark side in the two choice situation. He hypothesized that this difference in behavior was a function of activity level. Another hypothesis would be that the distance from the neck of the bell jar to the choice point (located in the plane formed by the joining of the sides of the bottle and its shoulders) was too short. The flying D. pseudoobscura did not have time to perceive and act upon the differential stimuli. The results reported above for depth perception in D. melanogaster when a similar apparatus was used makes the latter hypothesis attractive.

Experiment Three: Effects of Illumination and of Perceived Depth

Flies used in this experiment were wild D. melanogaster collected at a local fruit stand.

Data in Table 2 indicate that approximately 60% of the flies that had not been etherized prefer the high ceiling when given a 27" flight distance. Preliminary studies had established that difference of illumination between the two ceilings had no effect upon response proportions except when these differences were very extreme. To rule out the possibility that the flies were responding to stimuli other than depth, flies were tested under two conditions: (1) no light, and (2) no depth difference between ceilings.

In the first case lights in the apparatus were turned out and all testing was done at night in a dark room.

In the second case the high ceiling was brought to the same level as the low ceiling and illumination of the two ceilings was made equal. The results are presented in Table 3.

Table 3

| Condition | No. Released | No. Flew | % Flew | No. High | % High | $X^2$ obs. P = .5 | P = .6 |
|---|---|---|---|---|---|---|---|
| No light | 400 | 126 | 30.2 | 68 | 54.0 | .79 | 1.91 |
| No depth | 400 | 227 | 59.2 | 115 | 50.7 | .04 | 8.25** |

**significantly different from chance at the .01 level of probability

The results of testing without any light and thus no visual stimuli are equivocal because only 30.2% flew. Of the 126 that flew without phototropic stimulation 54% went to the high side. A Chi Square test shows that this is not significantly different from change (P = .5) nor significantly different from the expected response (P = .6).

The results of testing with no depth stimuli are obvious. Of the 227 that flew, 50.7% went to the former high ceiling. 50.7% is not significantly different from 50% but is significantly different from 60% at the .01 level using a Chi Square test. These results indicate that the depth related stimuli are the only ones operative in the previously observed 60% selection of the high ceiling.

Experiment Four: Strain Differences in the Depth Perception Preference of D. melanogaster

Behavior Genetics is a new field. As yet, no classical problem area of Psychology has been submitted to classical genetic investigation. To do so is the ultimate goal of this research. Having established the existence of depth perception preferences in Experiments Two and Three an attempt was made to discover strain differences. Data have already been obtained on wild fruit flies collected at a local fruit stand (see Table 2). Additional inbred strains: CS-200, negative geotropic and positive geotropic strains were tested. The latter two were developed by Erlenmeyer-Kimling, Hirsch and Weiss (1962). They were obtained for this study through the kindness of Dr. J. Hirsch. These flies had been selected for a specific geotropic response for 65 generations, after which selection was relaxed. They are relatively heterogeneous and were used here 149 and 150 generations from the beginning of selection. The comparison of the four strains is shown in Table 4. All of the flies were tested under the same conditions: the stimulus fields were of equal illumination; there was a 14" depth difference between High and Low ceiling; no ether was used; the flight distance was 27" and, as always, they were released in groups of 50.

Table 4

| GROUP | No. Released | No. Flew | % Flew | No. High | % High | $X^2$ obs. P = .5 | P = .6 |
|---|---|---|---|---|---|---|---|
| CS-200 | 500 | 307 | 61.4 | 183 | 59.6 | 11.34** | .02 |
| Neg. geotropic | 589+ | 435 | 73.9 | 239 | 54.9 | 4.25* | 4.64* |
| Pos. geotropic | 697+ | 540 | 77.4 | 330 | 61.1 | 26.67** | .27 |
| Wild | 339+ | 284 | 83.7 | 175 | 61.6 | 15.34** | .37 |

+No. deviates from multiple of 50 because of initial miscount
*significant at .05 level
**significant at .01 level

A number of interesting facts emerge from the comparison of the above data. The highly inbred CS-200 flies flew least frequently while the wild flies flew most frequently. The negative and positive geotropic strains were intermediate.

Other data not reported here have indicated that wild D. melanogaster would demonstrate a 60% preference for the high ceiling. Chance performance would dictate a 50% preference for the high ceiling. Therefore, Chi Square tests were performed to test two hypothesis: (1) that observed depth preference was random (P = .5), and (2) that the observed depth preference was not random but rather equal to a 60% preference for the high ceiling. As can be seen in Table 4, the percent of flies in each of the four groups that preferred the high side is significantly different from 50% at the .05 level. All except the negative geotropic strain were not significantly different from 60%. In fact, the 59.6%, 61.1% and 61.6%

observed are quite similar and extremely close to 60%. The 54.9% of the negative geotropic strain is unique. Apparently this group had a weaker preference for the high ceiling than did the other groups, yet this strain has been selectively bred for negative geotropism. Since they do not exhibit the strongest preference for the high ceiling, negative geotropism and depth perception preferences are probably not correlated.

Summary: An apparatus has been successfully tested which will allow D. melanogaster to exhibit a preference based on depth perception. The preference exhibited was for the higher of two visual ceilings in a box. Strain differences relative to the strength of the preference were observed. A strain of D. melanogaster selected for negative geotropism showed a weaker preference for the high ceiling than did any other strain tested. This latter observation suggests that depth perception preference for a high ceiling is independent of negative geotropism.

References: Erlenmeyer-Kimling, Loise, Hirsch, J. & Weiss, Jane M. Studies in behavior genetics: III. Selection and hybridization analyses of individual differences in sign of geotaxis. J. comp. physiol. Psychol., 1962, 55,722 - 731.

Lewontin, R. C. On the anomalous response of Drosophila pseudoobscura to light. Amer. Naturalist, 1959,93,321 - 328.

Jost, P. University of Oregon, Eugene.

Segregation in males with a normal or a $sc^4$-$sc^8$ X chromosome and an attached-4.

The segregation behavior of an X chromosome deficient for a considerable portion of the basal heterochromatin ($sc^4$-$sc^8$) shows a high frequency of primary non-disjunction (Gershenson, J. Genet. 28:297; Sandler and Braver, Genetics 51:573; Peacock, Genetics 51:573). Peacock confirmed cytologically that a high frequency of univalents occurr in meiosis. From Gershenson's conclusion that in $sc^4$-$sc^8$ YY males pairing was predominantly of the YY type, with the X unpaired, it can be reasoned that in a $sc^4$-$sc^8$ Y male the presence of a third unpaired chromosome that has exhibited a tendency to segregate non-randomly from the Y (in the female) might exhibit a non-random segregation. E. H. Grell (unpublished, cited by R. F. Grell, Genetics 50:151) and Jost (unpublished) have confirmed that an attached-4 chromosome does tend to show a non-random segregation in XX Y 4/4 females. The present experiment was designed to evaluate the behavior of an attached-4 (4/4) in $sc^4$-$sc^8$ Y 4/4 males.

Homozygous y w;AF, ci $ey^R$/gvl $sv^n$ (no free 4) females were crossed with two types of males, both carrying the $sc^8$·Y, which is marked with $y^+$, and an attached-4, AF, ci $ey^R$/ci $ey^R$ (no free 4). The X chromosomes used were a normal X, marked with $y\ w^a$, and $y\ sc^4\ f\ v\ cv\ sc^8$. All disjunctional and non-disjunctional classes of both parents were phenotypically distinguishable and diplo-4. An assumption of random segregation of the attached-4 with respect to the sex chromosomes was used to calculate the expected values shown below:

| | $y\ w^a/sc^8$·Y | | $sc^4$-$sc^8/sc^8$·Y | |
|---|---|---|---|---|
| | Observed | Expected | Observed | Expected |
| Y,4/4 non-disjunction | 417 | 424 | 701 | 741 |
| X,4/4 non-disjunction | 445 | 457 | 961 | 921 |

The frequency of recovered gametic types is consistent with those reported by other authors, with the exception that X and Y were recovered with approximately equal frequency. This is in contrast to the data of both Sandler and Braver and of Peacock, whose results showed that the X was recovered on the order of twice as frequently as the Y. The data, without considering the attached-4, are shown below:

| Paternal genotype | Sperm types | | | | Total $F_1$ | Proportion of X-bearing gametes | | |
|---|---|---|---|---|---|---|---|---|
| | X | Y | XY | O | | Of Total | In disjunctional classes | In nondisjunctional classes |
| $y\ w^a/sc^8$·Y | 2828 (0.554) | 2276 (0.446) | 1 | 3 | 5108 | 0.554 | 0.554 | --- |
| $y\ sc^4\ f\ y\ cv$ $sc^8/sc^8$·Y | 1360 (0.395) | 1258 (0.365) | 188 (0.055) | 637 (0.185) | 3442 | 0.450 | 0.520 | 0.228 |

If the Y and $\overline{44}$ tend to form a bivalent and the $sc^4$-$sc^8$ X remains unpaired (and if there is a tendency not to recover unpaired chromosomes), it might be expected that 1) the recovery of the X would be greatly reduced when compared to the recovery of the Y, and 2) the 44 would segregate non-randomly with respect to the sex chromosomes. Neither of these expectations is met. Instead the $\overline{44}$ segregates randomly and the X and Y are recovered with approximately equal frequencies. Estimation of nullo-X gametes cannot be made from these crosses, since their recovery is dependent on the frequency of $\overline{44}$ gametes from the test female. If the ratio of $\overline{44}$/nullo-$\overline{44}$ gametes is the same for both sexes (the data are not yet available to evaluate this), the recovery of $\overline{44}$ from the male is consistent with the reduction in viability associated with the $ci\ ey^R$ phenotype. No viability considerations are involved in evaluating other segregation classes since both 4th chromosome phenotypes occur in each class.

The possibility remains that the excess Y, expected from the formation of a Y, $\overline{44}$ bivalent and the unpaired $sc^4$-$sc^8$, is compensated for by a higher frequency of recovery of the shorter of the two chromosomes forming the bivalent. If this were the case, the proportion of nullo-XY class in the non-disjunctional gametes might be expected to show an increase. This proportion, however, is consistent with, but somewhat lower than, that found by earlier workers. Lastly, the presence of undetached YY males in the stock would, of course, tend to distort the results and this possibility cannot be excluded from the information available. These data must be considered as tentative and further tests will be made.

Novitski, E. and E. Ehrlich. University of Oregon, Eugene. Segregation in males carrying various chromosome rearrangements and the Segregation Distorter chromosome.

It has been shown by Peacock and Erickson (Genetics, 1965) that the action of SD is not through chromosome breakage, but that the virtual 100% recovery of the SD chromosome probably is caused by the preferential movement of the SD chromosome into the functional products of meiosis, much as proposed by Novitski and Sandler (PNAS, 1957). The results described below provide convincing evidence that a segregation phenomenon is in fact involved.

In the first place, when the non-SD chromosomes are involved in a Y translocation, the action of SD may be drastically modified, even to the extent of reversing the effect. With an ordinary T(Y-2) translocation, the percentages of recovery of SD are as follows: T(Y-2)A, 97%; T(Y-2)B, 34%; T(Y-2)C, 93%; Y(Y-2)E, 98%; T(Y-2)G, 78%; T(Y-2)J, 98% and T(Y-2)r1, 93%. All except the second show typical SD recovery rates. On the other hand, four Y-2 translocations involving the Y marked with $y^+$ gave rates of recovery 18%, 8%, 24% and 11%, respectively.

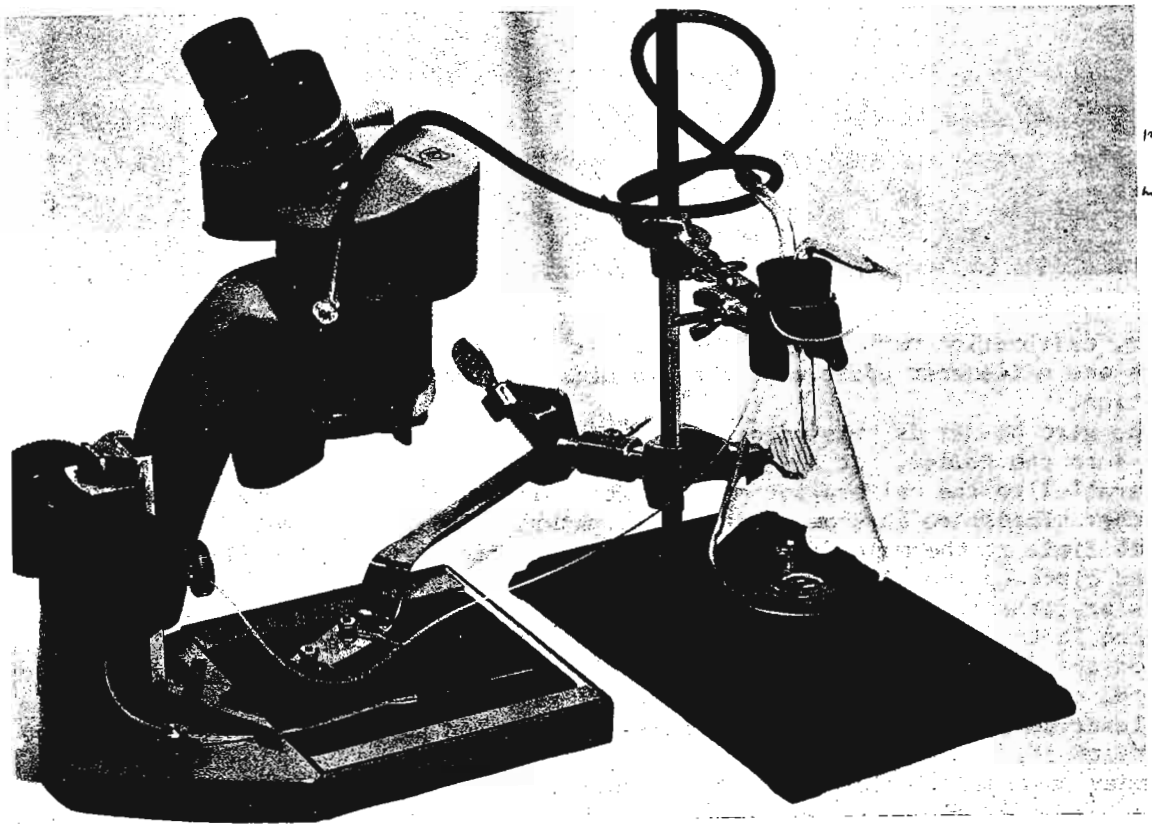
That this is not caused by the presence of the translocation involving the second chromosome per se is shown by the following series of results. Females carrying a normal and an XY X-chromosome and a normal and an SD second chromosome, produce $F_1$ males heterozygous for SD and carrying either a normal X or an XY X-chromosome. Those carrying the normal X gave the following rates of recovery of SD: 95%, 100%, 51%, 94%, 98%, 99%, 87%, 98%, a series not incompatible with the known action of SD. On the other hand, their brothers, who differed only by the possession of an XY instead of an X, gave the following: 58%, 67%, 55%, 60%, 66%, 56%, 64%, 50%, 86%, 70%, 63%, 57%, 50%, 76%, 61%, 63%, 52%, 65%. In no case was any result similar to the typical SD recovery.

From these observations, we conclude that the preferential recovery of SD is not only a segregation phenomenon dependent upon the physical make-up of the chromosomes, but can be reversed by the appropriate selection of homologs and may even be modified drastically by the judicious selection of structurally altered non-homologs! The basis for the latter effect may possibly be found in a non-homologous pre-segregation phenomenon such as that postulated by Novitski earlier (Genetics, 1964).

Seecof, R. L. City of Hope Medical Center.
An injection apparatus for Drosophila.

This injection apparatus is of very simple design, inexpensive and contains no wearing parts. The apparatus never requires adjustments and can therefore be operated by a technician. Many flies can be injected in a short time. Precision and accuracy are high because they are independent of micropipette size or the back pressure within the animal. The latter variables can cause inaccuracies when injections are made by methods that use a sudden blow to drive a pulse of inoculum.

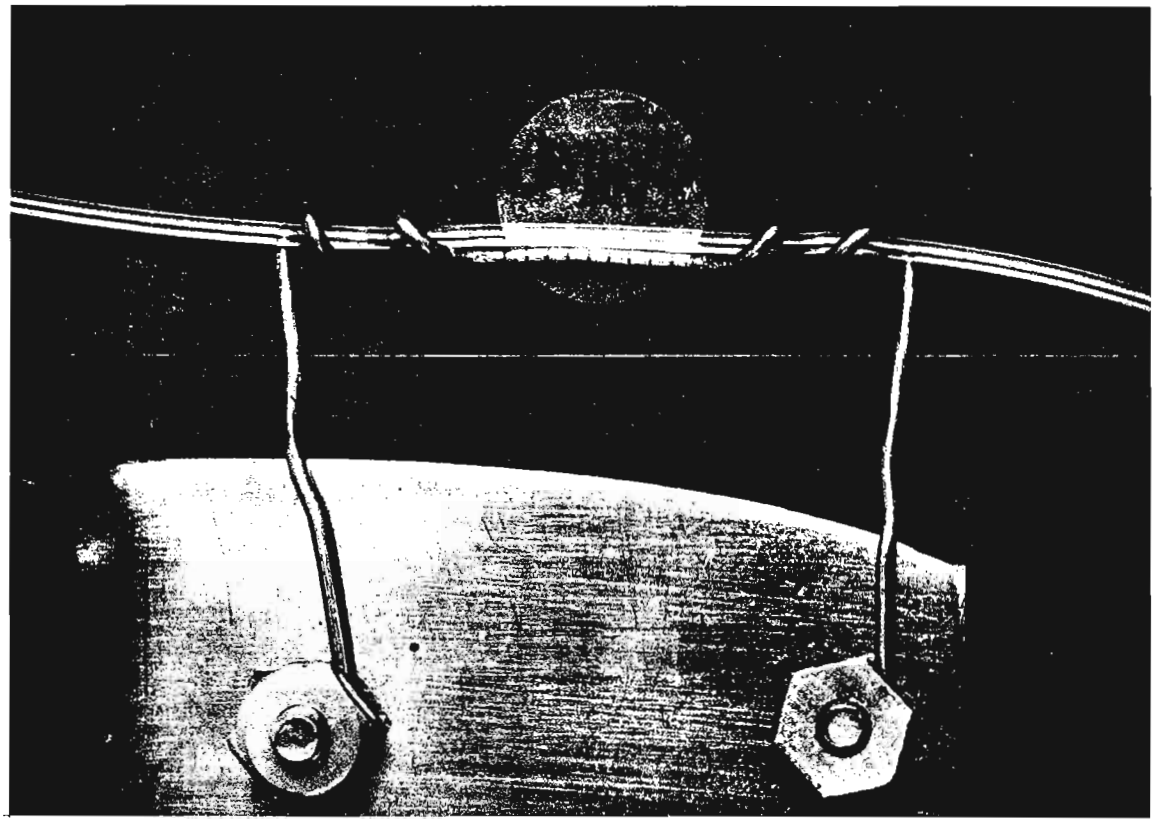
The injection apparatus is a mouth-pipetting arrangement (see illustration). The trap is a rubber-stoppered 125 ml Ehrlenmeyer flask connected to a glass mouthpiece by surgical tubing. The micropipette is attached to the trap by about three feet of polyethylene tubing (Clay Adams, PE 20, Aloe Catalogue)*. The polyethylene tubing is flared at one end to receive the tapered, fire-polished glass tubing from the trap and flared at the other end to receive the blunt end of the micropipette. Flaring is accomplished by holding the end of the tubing a few inches above a very small flame. Micropipettes, drawn from capillary tubing (OD 0.7-1.0mm), can be changed repeatedly without endangering the seal to the



* I.D. O.D.
mil .1015 .1043
mm .38 1.09

polyethylene tubing but their blunt ends must always be carefully fire-polished.

The polyethylene tubing is held in the microscope field by a wire attached to a supporting metal bar. The bar in the illustration is a bent table knife and the wire is a twisted common paper clip (Gem, no. 1, tinned steel wire 0.036 in diameter). The wire is shaped by twisting it around a piece of straight, rigid wire. After the wire is fastened



to the bar, calibration markings are cut into it with a razor blade. It is convenient to make marks one millimeter apart with the aid of a millimeter rule and twenty-power binocular magnification.

If the wire holder is twisted as shown, the polyethylene tubing need not be threaded end-first into the holder, but a loop of tubing can be folded into it. The tubing is held closely parallel to the calibration marks by the wire twists. When mounted in the field of an AO Spencer microscope as shown, seven millimeters of wire are readily visible at the side of the field at the magnification used for injection (20X). The light circle in the illustrated close-up shows the approximate size of the microscope field.

Flies are etherized and fastened in a row on a card by sticking their wings on to double-faced Scotch tape. The card can be pushed across the stage, beneath the wire holder, to position the flies in the microscope field for injection. The calibrated section of wire and the tubing against it are in nearly the same plane as the flies. No part of the wire is closer to the card than is the calibrated section so that the card can be moved freely beneath it.

In order to inject, water is first sucked about 30 cm into the tubing, then a micropipette is inserted and water blown down into the micropipette to fill it. A drop of mineral oil is then sucked up and, finally, the liquid to be injected is sucked into the micropipette. Air bubbles should be absent. The oil droplet prevents diffusion and is not sucked so far that it enters the plastic tubing. The plastic tubing is then drawn through the wire holder until the water meniscus within it is aligned with a calibration marking. The apparatus is arranged so that the flies, calibration marks and meniscus are visible in the microscope field (no meniscus is visible in the illustrations). The tip of the micropipette is then inserted into the ventral part of the flies' thorax, to either side of the midline, and liquid is introduced by mouth-pressure until the meniscus travels to the next calibration mark. The pipette tip need not be polished sharp; it will enter the imaginal thorax readily even though the tip is relatively blunt or poorly formed. Following the withdrawal of the pipette, bleeding will occur immediately (and the fly can be discarded) or not at all. Bleeding occurs in less than 10% of flies. The card can then be moved to align the

next fly and the injection repeated until the meniscus has reached the last visible calibration mark. Then the tubing can be drawn through the wire holder until the meniscus is repositioned at the first calibration mark and the next fly injected. Thirty flies can be fastened to the tape and injected in about 10 minutes. After injection the flies can be brushed from the tape with only minor damage to their wings. If the micropipette eventually becomes exhausted of liquid to be injected, more liquid can be sucked in without delay.

The volume of liquid delivered can be closely estimated by assuming the bore of the tubing to be uniform, sucking up a known volume of water, and measuring the length of tubing filled. An apparatus was checked by injecting dye into aliquots of buffer and found to deliver 0.164 microliters per millimeter injection (S.E. 0.014) over six millimeters. If millimeter injections were all made between the same two calibration marks the S.E. was lowered to about 0.01.

If quantitation is unimportant the apparatus can, of course, be used without the wire holder. Mortality is less than 5% if the flies are not overetherized. While the above description has been devoted to imago injection, the apparatus can be used to inject larvae or pupae or to deliver transplants if the micropipette is fashioned properly.

This investigation was supported by Public Health Service Grant AI-05038-03.

Marques, E. K.,[1] Marly Napp[1], Helga Winge[2] and A. R. Cordeiro[2]. Universidade do Rio Grande do Sul[1], Universidade de Brasília[2], Brazil. A corn meal, soybean flour, wheat germ medium for *Drosophila*.

Composition:

| | |
|---|---|
| Water | 14 liters |
| Wheat germ | 500 g |
| Wheat flour | 250 g |
| Corn meal | 1950 g |
| Soybean flour | 100 g |
| Sugar | 1550 g |
| Moldex (Nipagin or Tegosept M) | 45 g |
| Salt (NaCl) | 15 g |
| Hydrochloric acid 0,3 N. | 115 $cm^3$ |

This medium is inexpensive, its components are easily stocked and less variable than the one with bananas. The wheat germ and the small amount of soybean makes it very rich and productive. It can be autoclaved at higher temperatures than the banana agar food.

These proportions of wheat germ, wheat flour and soybean flour were adopted as a result of a factorial experiment using several species of *Drosophila*.

It is advisable to mix well the weighted dry flours with the moldex, packing them in the desired amounts. These packages can be sterilized to destroy any parasites (mites, fungi, etc.). This mixture is poured in tap water in which sugar and salt were added and they are boiled about ten minutes. The hydrochloric acid solution is then added. After about ten more minutes the mixture can be poured in the vials.

Mossige, Jeanne Coyne. Norsk Hydro's Institute for Cancer Research, Oslo, Norway. Fermented yeast for egg collection.

When large numbers of eggs are to be collected over a short period of time, the addition of acetic acid and alcohol to the yeast have been reported to stimulate oviposition. These procedures have improved egg laying, but none has been found to be consistently reliable, as is the following. Mix about 1/4 teaspoon of granulated sugar with 50 g of bakers yeast along with just enough water so that the mixture can be stirred with a spoon. This is covered and left in a thermostat at 25° for an hour or more, by which time it will be a foamy, spongy mass. When stirred with a spoon the $CO_2$ is released and the volume decreases. This yeast can then be spread or dropped on an appropriate surface for collecting eggs. It is readily manipulated as long as it is not too moist, and it consistently stimulates the females to lay large numbers of eggs.

Bircsak, Edmund and Dennis O'Brian.
Seton Hall University. A procedure for obtaining nanoliter samples of haemolymph from Drosophila melanogaster.

Utilizing a variation of the method developed by Felix and Salceda ("A technique for microinjection in Drosophila" DIS; 39:135; 1964), a technique has been developed in this laboratory whereby it is possible to measure accurately nano quantities of haemolymph extracted from individual Drosophila. This technique initially involves the calibration of the inner bore of a sacrificed thermometer by transferring into this bore, with the aid of a Clay Adams suction apparatus under 5x magnification, a two microliter volume of mercury from a volumized Pasteur disposable pipette. The tip of the pipette was previously reduced by means of a microflame so that it would concentrically fit within the thermometer cavity.

After a series of calibrations, it was determined that the volume between any two successive one-degree marks represented 30.3 nanoliters. The calibrated thermometer was then connected to a Neptune Dyna minivac type pump by means of a glass T-join and rubber tubing. Known volumes of mercury could then be taken up and dispensed by merely constricting the diameter of either the vacuum or pressure tubes respectively.

Haemolymph extraction was done under a 40x magnification by inserting a drawn out glass micropipette into the haemocoel of the larva. By capillarity, the haemolymph entered the micropipette and the level of rise was indicated by a dab of india ink. The pooled or individual samples of haemolymph were then stored for future analysis.

Once the haemolymph was collected, its volume could readily be determined by dispensing known volumes of mercury from the calibrated thermometer into the extraction micropipette. The results are summarized in Table 1.

This method is applicable to any insect form except that the adult stage requires a prepuncturing of the chitinous exoskeleton to facilitate insertion of the micropipette into the haemocoel.

Utilization of other calibration methods such as that described by Prager, Bowman and Vurek (Science; 147:606; 1965) require specialized apparatus which may not be readily available in the ordinary laboratory. Moreover, without a silicon-carbide cutter a serrated tip results when the micropipettes are broken into convenient sizes. However, this source of error is eliminated in the method described in this note in that the fluid nature of mercury accomodates any serrated portion of the micropipette tip and insures accurate calibration, while the serrated tip also penetrates more easily into the haemocoel of the insect. Finally, the ease of preparing the micropipettes and their calibration still favors the use of individually calibrated pipettes.

Table 1: Extremes of variation in collecting nanoliters of haemolymph from third instar larvae of Drosophila on two separate occasions. Mean values are given with the standard error.

| Number of trials | Number of units on thermometer | | Volume in nanoliters | |
|---|---|---|---|---|
| | A | B | A | B |
| 1 | 6 | 2 | 181.8 | 60.6 |
| 2 | 5 | 3 | 151.5 | 90.9 |
| 3 | 5 | 4 | 151.5 | 121.2 |
| 4 | 5 | 7 | 151.5 | 212.1 |
| 5 | 4 | 5 | 121.2 | 151.5 |
| 6 | 3 | 7 | 90.9 | 212.1 |
| 7 | 5 | 9 | 151.5 | 272.7 |
| 8 | 6 | 5 | 181.8 | 151.5 |
| 9 | 6 | 4 | 181.8 | 121.2 |
| 10 | 5 | 5 | 151.5 | 151.5 |
| 11 | | 5 | | 151.5 |
| Mean | | | 151.5±9.05 | 154.3±17.57 |

Komma, D. J. Columbia University, New York. Differential assay of G-6-PD produced by individual X-chromosomes in heterozygous D. m. females.

Glucose-6-phosphate dehydrogenase (G-6-PD) is produced by structural genes carried on the X chromosome of D. melanogaster (Young et al., 1964). Two alleles are known at the locus: ZwA (fast electrophoretic form) and ZwB (slow form)(Young, in press). A method has been developed here for measuring the ZwB form of the enzyme in the presence of ZwA. The key factor is the presence or absence of NADP (micotinamide-adenine dinucleotide phosphate) in the buffer in which the flies are homogenized. The standard buffer used here is a 0.05 M tris(hydroxymethyl)aminomethane-HCl buffer, pH 7.5, containing 0.01 M EDTA. The table shows the G-6-PD levels of homozygous ZwA, homozygous ZwB, and ZwA/ZwB females when 1) NADP (0.3 mg/ml) was present in the buffer at the time the flies were homogenized, and 2) when NADP was omitted from the buffer and not added until the assay was made. Activity is expressed as change in O.D. per minute per milligram of protein.

| | ZwA | ZwB | ZwA/AwB |
|---|---|---|---|
| 1) NADP present | .11 | .15 | .14 |
| 2) NADP absent | .00 | .12 | .02 |

The reason for the small amounts of ZwB in the heterozygotes is unknown. The effect seems to be real, since more ZwA than ZwB is also observed when heterozygotes are subjected to electrophoresis (cf. Young et al. 1964). In mixtures of the two homozygous types, the resultant activity when NADP is omitted is exactly the value expected if only the ZwB is being measured. (Research supported by NIH Grant #5-T1-GM-216-07.)

References: Young, W. J., J. E. Porter and B. Childs, Science 143:140, 1964
Young, W. J., J. Hered., in press

Chen, P. S. and Hanimann, F. Zoologisches Institut der Universität, Zurich, Switzerland. Qualitative and quantitative analysis of free ninhydrin-reacting components during the development of D. melanogaster by the automatic amino acid analyzer.

Using the automatic amino acid analyzer (Spackman, Stein and Moore, 1958) a detailed study of the changes in free amino acids, peptides and related compounds during the post-embryonic development of D. melanogaster has been carried out. The stages included larvae aged 1-4 days, pupae aged 1-3 days as well as one day old female and male adult flies. The samples used had usually a concentration of 0.3-0.5 g fresh weight per 2 ml methanol extract.

Qualitatively this technique is superior to paper partition chromatography. For example, leucine, isoleucine and phenylalanine can be separated very satisfactorily. The basic amino acids like lysine, histidine and arginine, which usually show low resolution and diffuse spots on paper chromatograms, also give rise to distinct peaks. Furthermore, we have found a considerable amount of ammonia which has thus far escaped our detection by paper-chromatographic analysis.

Of special interest is the appearance of more than ten acidic peptides and other related derivatives, all of which were eluted from the column before aspartic acid. Although no significant qualitative changes of these compounds have been revealed, the quantitative variations at various stages are however quite evident. During larval development their total content drops from about 9 to 6% of all free ninhydrin-positive compounds. At the time approaching puparium formation it increases rapidly to about 18%, and thereafter remains at a rather high level (12-18%) until the adult stage. Three components (fractions 3, 6 and 8) are especially concentrated and each of them has its own specific pattern. Such variations must be related to the metabolic processes that underlie the morphogenetic events. With help of the preparative column and the stream-divider attachment, large amounts of these peptides and derivatives have been collected for more detailed chemical analysis which is now in progress.

Brown, R. V., North Texas State University.
Use of Kelthane to control mites in Drosophila.

A recent study (Brown, R. V., 1965, J. of Econ. Entomol. 58:156-157) indicated the value of Kelthane (1, 1-bis (p-chlorophenyl) 2,2,2-tri-chloroethanol) for control of the genetic mite Histiostoma laboratorium (Hughes, R., 1950, J. Wash. Acad. Sci. 40:177-183) formerly called Histiostoma genetica (Stolpe, S. G., 1938, Anat. Rec. 72:133-134). Some additional experience in the use of Kelthane has been acquired in eliminating mites in Drosophila stocks of two other laboratories. The method used was to wash and autoclave bottles to be used for media. Bottle interiors were rinsed in a Kelthane suspension of 400 ppm and allowed to drain and dry (or nearly). A cornmeal-agar media was added. Flies were transferred to these bottles. As soon as the next generation began emerging, they were transferred to similarly treated bottles with fresh media. The third generation flies were examined and were found to be free of mites.

Another procedure was tried and found to be of value. Bottles heavily contaminated with mites were treated as follows: (1) All adult flies were removed and discarded; (2) a 75 ppm solution was poured into the bottles and quickly poured out; (3) bottles were stoppered. Less than one percent of the flies that emerged had mites one week later. This procedure requires quickness in rinsing, and cultures that are vigorous with large numbers of developing flies, as the rinsing removes many of the pupae.

Pre-treatment of bottles before addition of media allowed for drying of bottles. This was more satisfactory than when bottles with media in them were treated and stoppered without drying, as higher concentrations of Kelthane were not so toxic in dry bottles.

One incidental observation that may be of interest to some workers was noted. The toxic concentrations of Kelthane did not appear to be equally toxic for all stocks. Stocks that appeared most susceptible were ec cv v f, f, and v. This is simply an observation and has not been investigated.

Ditman, W. F. Purdue University.
An improved method for determining visual depth preferences in large numbers of D. melanogaster.

Efficient techniques for determining visual preferences of D. melanogaster are often desirable for behavioral studies. Usually flies are allowed to crawl singly through T- or Y-tubes, the arms of which differ on some visual dimension such as brightness or hue. This method is time consuming if large numbers of flies are to be tested. Also the small size of the tubes precludes testing preferences for multi-dimensional visual stimuli such as form or depth.

To overcome the restrictions of the T- or Y-tube a large shaft was used. Fifty flies were released at the bottom and removed at the top. Inside, the shaft was painted flat black and was approximately 14" L x 7" W x 27" H. The final (upper) 8" of the shaft was tapered to 12" L x 6" W. Atop the shaft was a collection box 12" L x 6" W x 3" H inside. This collecting box was divided into two 6" L x 6" W compartments by means of a clear plastic partition 3" high. The top of the collection box was a pane of glass mounted to slide aside for removal of the flies with an aspirator. The visual stimuli were placed above the glass. All light entering the shaft entered through the glass.

When different stimuli are placed over the two 6" x 6" compartments in this two-choice situation flies attracted to the first stimulus collect in one compartment, while flies reacting to the second stimulus gather in the other compartment. Flies are released from the culture bottle by the removal of a small trap door above the bottle. The negative geotropism and positive phototropism of D. melanogaster encourage the flies to fly toward the two visual stimuli. Thirty seconds after the flies are released, the sliding trap doors beneath the two 6" x 6" collecting chambers are closed, effectively isolating the two groups of flies which have chosen between the two different stimuli. (See Fig. 1).

To create a depth stimulus, a 4' x 4' x 2' high inside box was centered above the collecting chambers. A mirror, 4' x 2', was placed in the center of the box in a vertical plane directly above the plastic partition of the collecting box. On either side of the mirror sheets of translucent white plexiglass were suspended. The plexiglass had 3" square pieces of black construction paper glued to the underside in a checkerboard pattern.

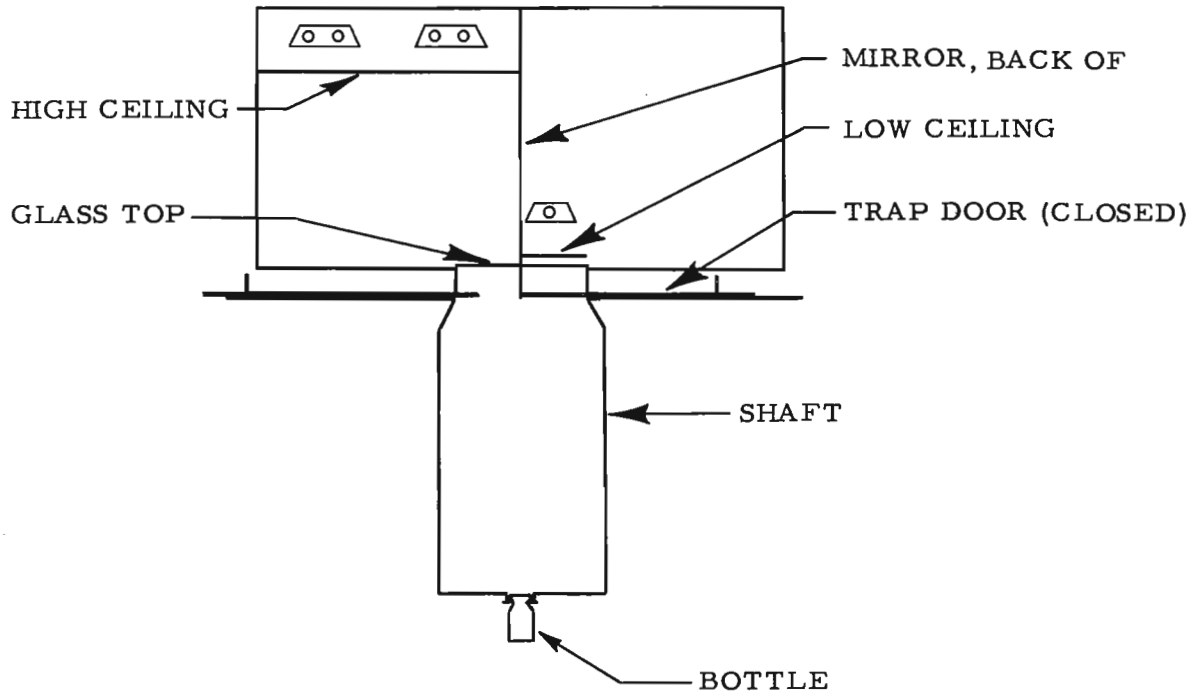



FIG. 1. BASIC VISUAL PREFERENCE APPARATUS WITH VISUAL DEPTH STIMULI

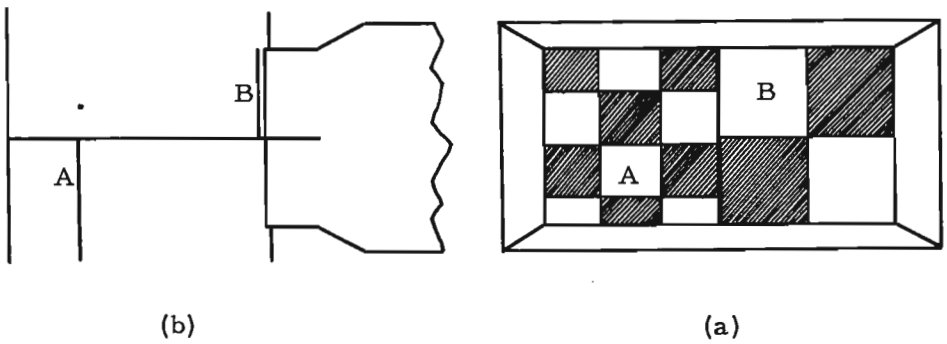



FIG. 2. a- VISUAL DEPTH STIMULI (A & B) AS SEEN FROM THE BOTTOM OF THE SHAFT. b- ACTUAL ARRANGEMENT OF A & B

Fluorescent lamps were mounted above the plexiglass. One plexiglass sheet was lowered until it rested on the glass pane of the collecting box, while the other piece of plexiglass was raised to a height of 14" above the glass pane. In this way an illusion of depth in the form of a high and low "ceiling" was created. Fig. 2 is an illustration of the appearance of the apparatus from inside looking up. The mirror is used to eliminate the cue of interposition. The edges of the plexiglass are not visible. With this arrangement only two depth-perception cues are available to the flies: motion parallax and textural differences of the stimulus' surfaces.

Doane, W. W. Yale University, New Haven, connecticut. Separation and quantitation of α-amylases with disc electrophoresis.

An improved method was developed to analyze α-amylases in Drosophila separated by acrylamide gel disc electrophoresis. The procedure provides both quantitative and qualitative estimates of enzyme activity from single flies or individual tissues. (A previous method, DIS 40:97, was abandoned.)

The technique of Ornstein & Davis (1962) is employed with minor modifications: 1) omit $K_4Fe(CN)6$ from small pore gel, 2) use 1/2 the amount of N,N,N',N'-tetramethylethylenediamine, 3) substitute 0.47 M tris-phosphate buffer, pH 6.9, for tris-HCl in large pore gels, and 4) add 1 µl. 10% 3-dimethylaminopropionitrile (DMAPN) to each sample gel to insure polymerization. Supernatant from a single fly, homogenized in 10 to 15 µl. distilled water and centrifuged in a capillary tube, is put directly on top of spacer gel, mixed with 0.1 ml. large pore gel and layered-over with another 0.1 ml. of this gel. Electrophoresis is done at 4°C. with a constant current of 4 ma per gel tube for 50-60 minutes.

After electrophoresis, gels are removed from their tubes and placed on a corrugated rack in a moist chamber. A glass plate (3 1/4" x 4"), coated with a starch-acrylamide film, is placed over the gels, weighted (125 gm.), and left to incubate at 25°C. for 30 minutes. Plates are made by mixing 2 ml. small pore gel as usual but altering the pH to 7.4 and including 1.5% Connaught hydrolyzed starch, previously boiled 5 min. in the water used in making "small-pore solution #2". Ten µl. of 10% DMAPN solution is added and the mixture spread between two glass plates, one of which is coated on its periphery with dried albumin. Cover-glass chips are set in the albumin to separate the plates so the film forms with uniform thickness. (Film thickness, sample size and incubation time must be suitably adjusted for densitometric analysis.) Films are polymerized over a fluorescent bulb 15-20 min. and immersed in tris-HCl buffer, pH7.4 (optimum for melanogaster, Doane, unpubl.), where they may be stored under refrigeration 1-2 weeks or used immediately. Prior to use, plates are rinsed with water, the outsides dried, and the two separated from one another, leaving the starch-acrylamide film on the side edged with albumin. The latter plate is placed directly on electrophoresed gels to incubate, care being taken to avoid any additional moisture.

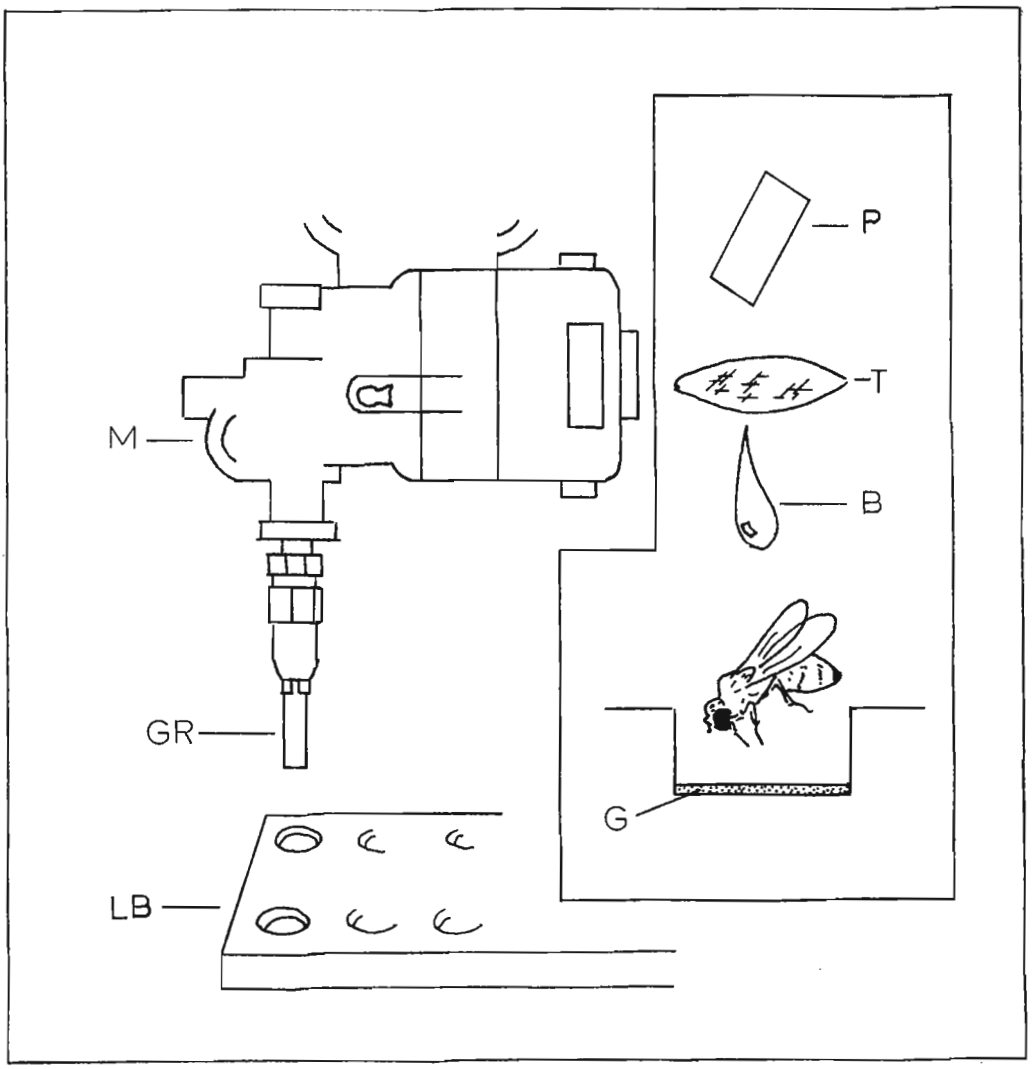
Following incubation, the starch-acrylamide plate is set in I-KI reagent 1-2 min. to stop the reaction and to render unhydrolyzed portions of the film blue. Not only do amylase bands stand out distinctly (ranging from lighter shades of blue to nearly colorless), but the imprint of the individual gels may be discerned along with their origins and leading edges (the latter caused by tracking dye). The stained plate is rinsed with water, then 7% acetic acid, and re-covered with the other glass plate removed prior to incubation.

The stained starch film, sandwiched between two plates, may be stored several months without deterioration by placing it, with a little 7% acetic acid, in a container in the dark and under refrigeration. It serves a double purpose: 1) it is used as a photographic negative for contact prints of amylase banding patterns, and 2) it is scanned densitometrically (Joyce-Loebl Microdensitometer) to determine relative activity in different bands (see research note, this issue). Amylases in the electrophoresed gels remain unharmed and may be cut out, eluted, and further analyzed.

Johnson, F. M. University of Wisconsin.

Rapid single fly homogenization for the investigation of Drosophila isozymes.

As a compromise between the extremely rapid but generally inefficient method of directly squashing flies on filter paper and the most thorough but time consuming and expensive procedure of using regular tissue homogenizers for extracting proteins, preparatory to electrophoretic analysis in starch gels, a simple device has been recently employed. First attempts to avoid the streaking of protein bands which often result when fragments of tissue are present in the sample necessitated mincing individuals with a lancet in a small volume of appropriate homogenizing fluid, covering the slurry with a piece of Kleenex (filter trap) and adsorbing the extract through the Kleenex into a piece of filter paper. Although fairly rapid and useful in examining electrophoretic variation in prominent proteins, the method left something to be desired in the resolution of minor components and sample uniformity.




The method currently used is similar to small scale homogenization in glass tubes but avoids the need for centrifugation and handling of separate tubes for each sample. A small Bodine motor, Model 102, Talboys Instrument Corp. (M) rotates a short length of 1/4 inch glass rod (GR) at 80% of maxium speed. A lucite block, 4 1/2" x 2" x 1/4", (LB) containing 12-1/8" deep depressions made with a 1/4" d. drill, broken off square at the bottom, contains the Drosophila; twelve individuals can be conveniently homogenized in one assembly line type process. A small amount of powdered glass (G) in the depression adds to the efficiency of homogenization. After the flies are placed in the depressions a measured amount of buffer (B) or distilled water is added to each Drosophila sample. With the lucite block in one hand and a Kleenex in the other, the flies are successively homogenized and the rotating rod wiped clean. About 20 seconds is usually sufficient for an adequate smashing job if some rotating and up and down movement is applied to the lucite block. When all 12 samples are homogenized, each is covered with a circular Kleenex filter trap (T), easily made with a 1/4" d. paper punch, and after all are covered a 5 mm. x 7 mm. piece of Whatman No. 1 filter paper (P) is placed at an angle into each depression. As the last filter paper is inserted, the first should be completely saturated and ready to be placed in the gel. Time elapsed in preparing 24 samples and placing them in the gel is about 16 minutes.

One gel, 7 mm. thick, is easily cut into three slices which means three isozyme systems can be examined from a single fly. Since evaporation is fairly slow from the depressions there is ample time for inserting three 2 mm. x 7 mm. filter papers rather than the single larger size paper, which triples the potential number of isozyme systems that can be examined from a single fly, with very little additional effort. A further increase can be realized by double staining.

Bennett, J. and S. Mittler. Northern Illinois University, DeKalb. Plastic Plugs for shell vials.

Polyurethane foam plugs have been used in shell vials here since the fall of 1962. Others (DIS 37) have reported using polyurethane plugs in bottles, however the commercially available sizes do not fit well in the 25 x 95 mm shell vials used here. Extensive correspondence with the supplier failed to elicit an appropriate size for vials. We have developed our own equipment for making the plugs and describe it here.

The basic tool is the male portion of a Greenlee, type 730, 1 1/16 inch (27 mm) diameter, #AV1763 radio chassis punch (such as Newark Electronics Corp., 223 W. Madison St., Chicago, Ill. 60606, Catalog #33F765 @ $2.70). The female portion is too large (designed for cutting metal) and must be discarded. A large arbor press (Greenerd Arbor Press, Nashua, New Hampshire, No. 3, S 31) was obtained from a federal surplus depot for schools for $35 (new value probably over $300). A local machine shop prepared an appropriate foot plate with a hardened, centerable insert (reworked from a Timken #15101 bearing race) machined to a finger tight fit with the male part of the punch. When installed in the press, Fig. 1., this provided a strong and reliable punching system for manufacturing plugs.

The plugs are cut from 1 1/2" x 18" (3.8 x 45.8 cm) cross section strips of foam, sold for upholstering at hardward stores, or from 2" (5.08 cm) thick strips from Montgomery Ward, Chicago, Illinois (Catalog #71B6070L, @ $0.66/linear foot (30.5 cm)). Care must be exercised in cutting to avoid overlapping a previous cut. This produces flat sides on the plugs and may result in escape of flies. The perforated strips of foam that are left over are excellent padding for shipping vials of flies, etc.

Use of the plastic plugs in vials: 1. Best fit is achieved if they are pushed all of the way in and then pulled back out about 1/3 of their length. 2. Watch for creases or folds that might provide escape or entry channels. This is especially applicable to new plugs. 3. Plugs may be autoclaved at 121°C (250°F) and handle more easily with repeated use. 4. Dirty plugs may be washed in a mesh bag (we use a household automatic washer), autoclaved, dried in a hot air oven (not over 100°C) and are better than new. Dry overheating will ruin the plugs.

The polyurethane foam plugs seem to have these advantages in our laboratories: economy of re-use makes them cheaper than cotton; elimination of the irritation of cotton fibers in room air, and consequent allergic responses of people and equipment; greater uniformity provides more reliability in general use.

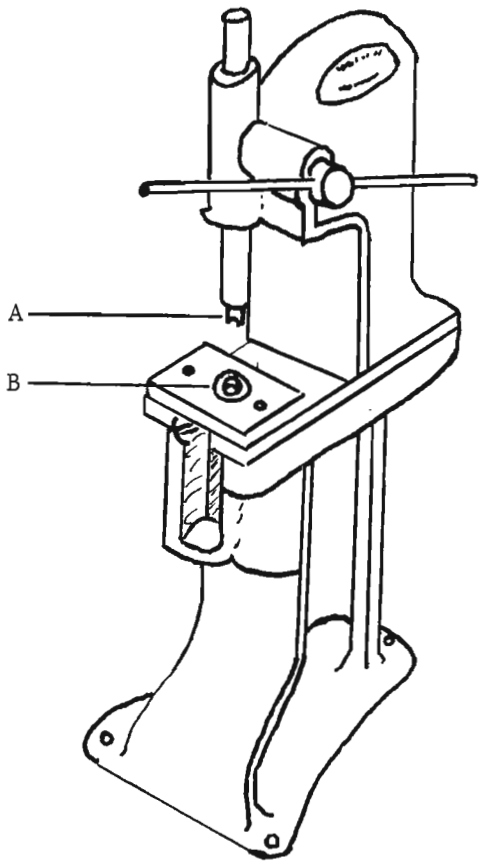



Figure 1. Sketch of press fitted with punch (A) and foot plate with hardened insert (B). Overall height of press frame approximately 145 cm (57").

Podger, R. N. and J. S. F. Barker. University of Sydney, Australia. Collection of large numbers of larvae of homogeneous age and development.

In recent experiments, where we have been studying the effect of variation in larval density on various fitness components, up to 30,000 newly hatched larvae were required on any one day. Larvae were to be used at up to 2 hours post-hatching, and it was important that they be as homogeneous as possible. However, previous experience had shown that crowding and disturbance of adults causes some females to delay laying of eggs subsequent to fertilization. Thus, the larvae hatching from even a short period egg collection will be at quite variable stages of development.

The technique described here allowed a steady supply of larvae throughout a day, and ensured that larvae of advanced development were excluded. Large numbers of 1 to 2 day old flies raised under uncrowded conditions on dead yeast fortified medium were collected and placed in 9x9x3 inch polythene population cages; 5000 to 6000 per cage. The food available to these adults comprised approximately equal amounts of a heavy live yeast suspension and ordinary cornmeal- treacle medium. Instead of the regular medium jars, bark corks (1 1/2 inch diameter) with a circular depression cut in the narrow end, were used as food receptacles. For egg collection, all 9 food containing corks were replaced by ones containing thin discs of 1.5% agar (1/16-1/8 inch thick). These discs remained in the cage for one hour, and when on any one day more than two egg samples were to be collected from a population, the discs were lightly smeared with a 5% dead yeast suspension before use. Up to 8 consecutive hourly egg samples were obtained each day. Provided food was available to the adults overnight, a population continued to produce large numbers of eggs for several days. On removal from a cage, egg collection discs were stored on 7x4 1/2 inch metal trays with a fibreglass mesh base. The trays were stacked in a plastic box and covered with calico to reduce drying out and to prevent contamination.

We had examined the hatching pattern of eggs of the strains being used and found the distribution to be markedly bimodal. For example, for D. melanogaster Oregon-R-C, eggs hatched from a few hours after laying, with a small peak at 19 hours. Hatching of the bulk of eggs commenced at about 20 hours, so that those hatching from about 19 to 20 hours comprise a mixture of "held" eggs and early-hatching normally laid eggs. The numbers hatching increased rapidly from 20 hours to a sharp mode at 21 hours, and hatching was essentially complete by 23 hours. The beginning of the peak hatching period was therefore known, and at this time after the mean of an egg sampling period, all hatched larvae were washed from the discs using a plastic squeeze bottle. Excess water was removed by gentle application of fine paper tissue. All larvae were readily removed in this way without disturbing unhatched eggs. All washing water and equipment used was held at 25° C before use, to allow equilibration to this standard environment.

Collection of larvae commenced one hour after washing, but of course, could follow sooner if younger larvae were required. Larvae were collected, using a dissection needle, for a one hour period fron each sample of discs, so that they were up to 2 hours old when used to initiate experimental cultures. The numbers of larvae available in this period from the 9 discs of a one hour cage sample varied from about 2000 to 4000. An experienced operator could collect between 1000 and 2000 larvae per hour, depending on the numbers of larvae and strain mixtures being placed in individual cultures.

Counce, S. J. Yale University, New Haven. Whole mounts of *Drosophila* embryos.

This technique for whole mounts of insect embryos is not original with me, but is easy, relatively quick, and gives good results with several species of *Drosophila*. Developmental details are so clear the technique could be used for screening for mutants affecting specific embryonic stages.

1. Dechorionate eggs in diluted Clorox and wash.
2. Fix in formol-alcohol-acetic acid, pricking the eggs with tungsten needles sharpened in melted sodium nitrite.
3. 30% alcohol, 15 minutes; 70% alcohol, 15 minutes.
4. Stain 24-48 hours in alcoholic borax carmine.* Best results were obtained with pre-World War I German stains kindly given me by Kenneth Cooper, but carmine obtained from G. T. Gurr gives good results. Superficial staining occurs within a few hours but nuclear details are better in embryos stained for longer periods.
5. Destain with acid alcohol (a few drops of concentrated HCl in 70% alcohol) until desired contrast is obtained, usually 24-48 hours. Embryos should be a pale cherry red and will deepen in color when placed in xylol.
6. Dehydrate through absolute alcohol, clear in xylol, and mount. To prevent fragmentation of embryos, the coverslip is supported on two sides by small pieces broken from no. 1 coverslips. Sealing with fingernail polish prevents shrinkage of the mounting medium away from the sides of the coverslip.

*Alcoholic borax carmine (after ROMEIS): Grind together finely 2-3 grams carmine with 4 grams borax. Dissolve by slowly heating in 100 ml distilled water. Cool. Add 100 ml 70% alcohol. Let stand for a week shaking frequently. Filter before use.

Spieth, Herman T.[1] University of California. A method for transporting adult Drosophila.[2]

In the course of recent investigations of the endemic Hawaiian drosophilids, some difficulties arose with the transportation via motor vehicles and/or inter-island airplanes of field captured adult Drosophila from the cool, wet rain forests to the much warmer coastal area of Honolulu. Not only are the insects sensitive to heat and desiccation, but also they are likely to become "stuck" in the food and to the walls of containers more often than is true for individuals of species from other parts of the world. The following method was devised and has proven eminently successful for the transportation of the flies not only from the field to the laboratory at the University of Hawaii in Honolulu, but also from Hawaii to the University of Texas via air freight.

Eight-dram vials are lined with water-dampened, 6.5 x 6.5 cm. pieces of chromatography paper that has approximately the same texture and thickness as that of a common desk blotter.

The following media is then prepared:

15 gm. Bactoagar
1000 ml. Water
50 ml. Karo syrup (dark)

The agar is added to the water and the mixture is heated sufficiently to dissolve the agar. The Karo syrup is then added and the resulting mixture is simmered for 2 to 3 minutes.

Into each paper-lined vial the hot mixture is poured to a depth of approximately 8 mm. (0.25 in.), the exact amount to be determined by the absorbency of the paper being used. The vials are tightly stoppered with non-absorbent cotton, and are then autoclaved for 15 to 20 lbs. pressure, after which the autoclave is slow-exhausted. When removed from the autoclave, all of the liquid medium should have been absorbed into the paper lining and only a very thin film should remain on the bottom of the vial.

The vials should be prepared 1 to 2 days before use but since they are sterile they do not need to be refrigerated. If they are to be kept a longer time before flies are introduced into them, they should be stored in a tight container to reduce evaporation via the cotton plug.

When the flies are introduced into the vials, they feed readily upon the surface of the media-impregnated paper and cling to the surface easily. Fecal materials are absorbed into the paper and condensation does not form upon the paper surface when the vials containing flies are placed in a cold insulated shipping container that is kept cooled by means of 2 or 3 frozen containers of "Scotch Ice," "Magic Cold," or other chilling agent. The individuals of all species tested to date have remained healthy for 4 to 7 days before it was necessary to change them to a fresh vial.

When preparing for shipment, the vials should preferably be laid on their sides in the shipping container and should be wrapped and padded with newspaper.

White blotting paper can be substituted for the chromatography paper but in our experience residual impurities in the paper, apparently localized to limited areas, cause a small percentage of the vials to become poisonous to the flies due to the release of the noxious materials as a result of the autoclaving.

[1]Visiting Colleague, University of Hawaii, July to December, 1964; Guest Investigator, University of Texas, December, 1964 to June, 1965.

[2]Supported in part by grants GB-711 (NSF) and GM 10640-03 (NIH).

Wrathall, C. Richard and E. W. Hanly. University of Utah. Another plug for culture vials.

We have found in this laboratory that large rayon balls, purchased from Kendall Co., Fiber Products Division, Walpole, Massachusetts, 02081 (No. 6898, size 580) make very successful plugs for the commercially available 8 dram shell vials. Their cost is low (5.00/2000), they fit perfectly into the vial (time saving) and retain their color and resiliency after many autoclavings.

Mellett, J. S. Iona College. Plastic beakers for culturing Drosophila.

Any workers (particularly those dealing with undergraduates in genetics laboratory courses) dissatisfied with the traditional glass bottle method of culturing Drosophila might be interested in the disposable "Multi-pour" beakers currently being marketed by Clay-Adams Inc., 141 E. 25th St., New York, N. Y., 10010. They are available in four sizes (50, 100, 250, and 400 ml), each with a tight fitting cardboard cap, on which a mass of information can be recorded. While probably all are suitable for Drosophila genetics work, I have found the 100 ml size the best for student experiments. (Fig. 1).

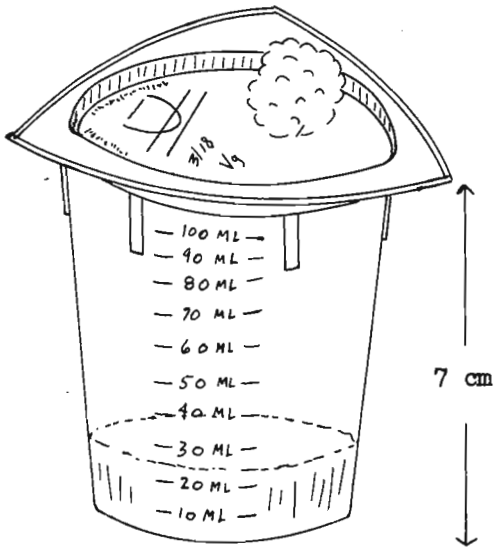



Fig. 1 Clay-Adams 100 ml "Multi-pour" beaker

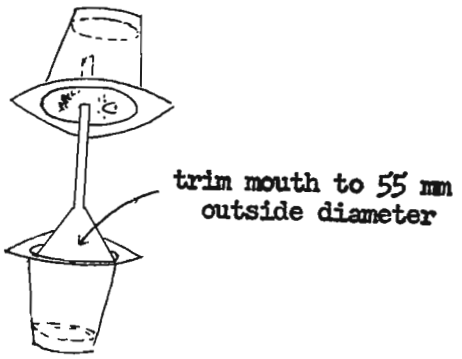



Fig. 2 Transfer method using polyethylene funnel

The beakers are composed of resistant, unbreakable polypropylene plastic, are graduated and can be subjected to repeated autoclaving without risk of melting. (Unless you actually prefer to discard them after one using).

While the narrow necked glass bottles are difficult to clean thoroughly even with a brush, the plastic beakers can be easily wiped out with a sponge. Because the beakers taper from top to bottom, when not in use they can be nested, one inside the other and stored in a very small area; six nested beakers take up slightly more volume than a single half-pint cream bottle.

Drosophila flies thrive on 20-25 ml of culture medium, which means that 2-3 plastic beakers can be filled with the same amount of food usually required for one glass bottle. Each beaker can be expected to produce anywhere from 200-400 flies, which is a respectable sample.

One note of caution must be made with regard to the cardboard caps--they are lined with a thin layer of plastic and cannot be used as is, since diffusion will not occur through them. The problem can be eliminated however by making a 1/4 inch hole in the cap (using an ordinary paper punch or pencil point) and stuffing it with a small tuft of cotton. Cultures of flies can be maintained in the beakers for well over a month if necessary once this modification is made. The hole in the cap also allows one to make transfers of flies from one beaker to another by using plastic funnels with mouths trimmed to fit the inside of the beakers. (Fig. 2).

I have found the standard glass bottles still useful in the laboratory for stock cultures of Drosophila strains; they are easily identified as such and are less likely to be misplaced or labeled incorrectly by students or personnel unfamiliar with laboratory procedures. By some coincidence, the mouth of the half-pint cream bottle fits perfectly just inside the opening of the 100 ml beaker, making reciprocal transfers of flies a trouble free procedure

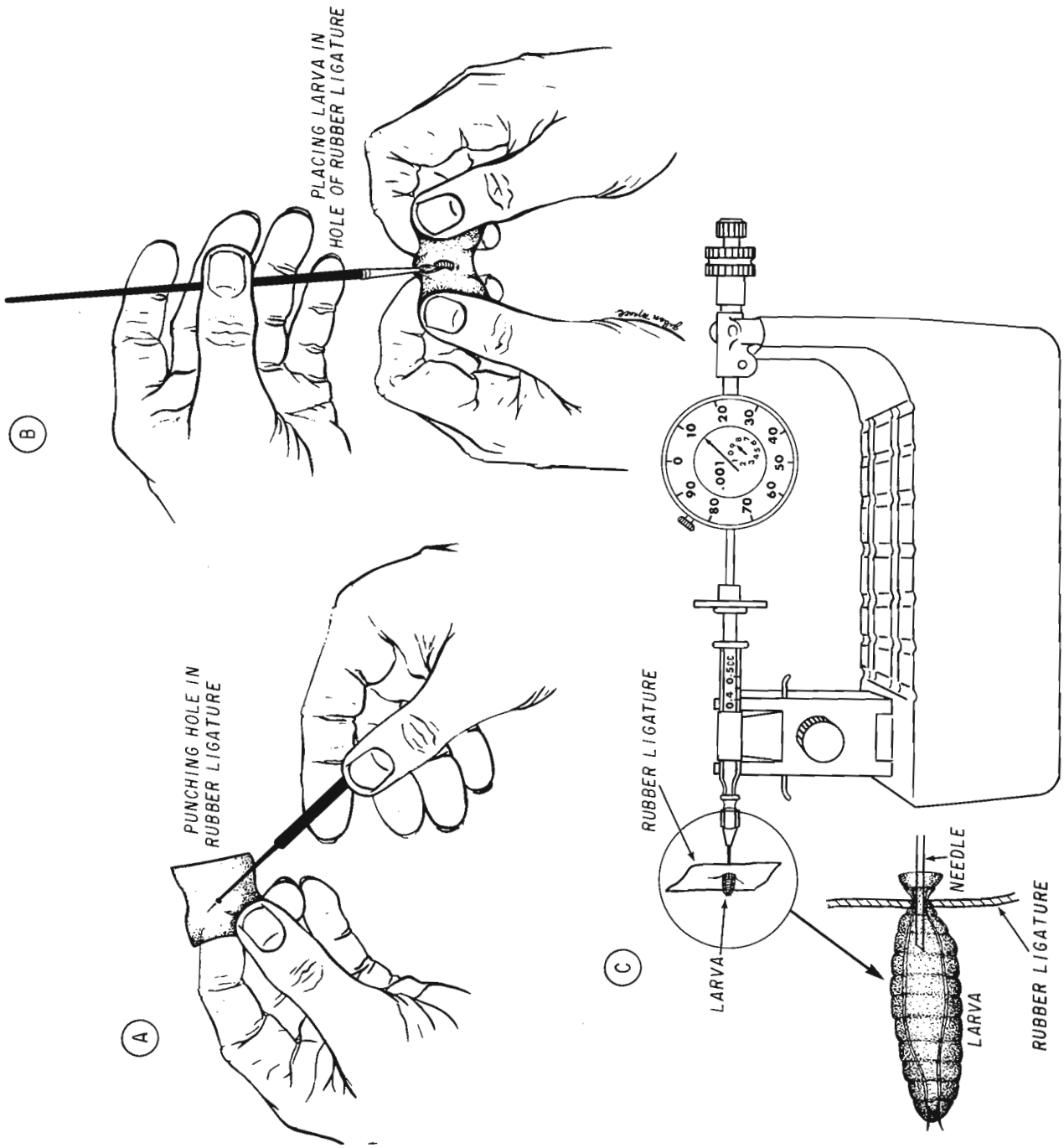
One minor disadvantage of the beakers is that they are not as clear as glass, although they are certainly transparent enough to enable one to sex flies and recognize the common mutants at a glance.

The beakers are sold in boxes of 100 (catalog no. A 3600) and cost about eight cents apiece. Caps are packaged 500 per box (catalog no. A 3608 B) and cost about a penny each.

Anderson, R., Maureen Hancock, and Walter J. Burdette. University of Utah College of Medicine. A simple method for ligating and injecting Drosophila larvae.

A hole is made with the tip of a dissecting needle in the center of a one inch square of rubber cut from (discarded) surgical gloves. The larva is inserted at the desired segment through this small hole. The rubber is stretched enough to make the hole large enough and the larva is inserted with the tip of a brush. The tension on the rubber is then released and the ligature is complete. This is easily done under a dissecting microscope.

For injections, a 30 guage needle with a standard hub on a 0.25 ml. disposable plastic syringe with polystyrene plunger is used (B-D Hypack, Discardit). This type of plunger is preferred because it eliminates backwash of materials. If available needles are too long, they may be cut so they are one quarter of an inch long and then resharpened. This assembly is attached to a microburet (model SB 2 Micrometric Instrument Co., Cleveland, Ohio). The caudal tip of the ligated abdomen may be left intact or snipped off with scissors, using the open end of the skin as a guide. The larva is then placed on the needle so that the needle passes through the ligature. A precalibrated dose is injected and the larvae is removed by sliding the rubber with the larva off the needle. The rubber hole will close as it is removed from the needle, preventing any backwash of material. The larva is then placed cutside down on a damp piece of filter paper in a large Petri dish. If the larva has been injured it will turn black at the point of injury. With experience, they are seldom injured and third-instar larvae will pupate.


PLACING LARVA IN HOLE OF RUBBER LIGATURE
B
PUNCHING HOLE IN RUBBER LIGATURE
A
RUBBER LIGATURE
LARVA
C
NEEDLE
LARVA
RUBBER LIGATURE

Kiriazis, W. C. and L. D. Friedman. Hiram College, Ohio. A modification in the technique for the paper chromatographic separation of pteridines.

In previous studies of separating pteridine substances in Drosophila melanogaster, workers have reported separating 10 substances by two-dimensional ascending chromatography (Narayawan and Weir, 1964, Genetics 50:387-392) and 17 substances by two-dimensional descending chromatography (Fox, 1956, Physiological Zoology 29: 288-298). These results were obtained through the use of the squashing technique in which approximately 10 to 15 heads or bodies of the flies were used. By using the whole fly, we have developed a method in which 10 to 23 spots, most of which are pteridines, can be separated in two-dimensional ascending chromatography. We are able to fractionate 200 Ore-R males and obtain 19 spots, and when 400 Ore-R males are used, 23 spots can be observed.

The two hundred whole flies were homogenized in 1 ml of two parts propyl alcohol to one part 1% ammonium hydroxide for thirty minutes in a Virtis 23 homogenizer. The homogenate was then centrifuged for thirty minutes at 4°C. The supernatant was removed and spotted on Whatman #1 chromatographic filter paper with a 1 µl pipette. Approximately 30 µl were used for the spot. The spot was allowed to dry, and the paper was then placed in a chromatographic jar (25 by 12) in a darkened room at 25 ± 1°C. The chromatogram was developed for 18 hours by ascending chromatography and dried at 25 ± 1°C in the dark. The solvent for the one-dimensional development was the standard two parts propyl alcohol to one part 1% ammonium hydroxide. For two-dimensional development, water-saturated collidine (2,4,6, trimethyl pyridine) in the ratio of three parts collidine to one hundred parts distilled water was used. The second development lasted ten hours. In both methods of development 700 ml of solvent was placed in the jar. The fluorescent pteridines on the chromatograms were then studied under an ultra-violet wavelength of 360 µm.

This modification of the standard method has two advantages. It separates substances which are in minute quantities in the fly, and it also produces a sufficient quantity of a particular pteridine in one spot that can be used for further quantitative analysis of the compound. By this method we have been able to separate four spots associated with drosopterin. In previous papers only three have been separated by ascending paper chromatography: drosopterin, isodrosopterin, and neodrosopterin. The fourth is of a darker red-orange fluorescence and has been confirmed by Gregg (personal communication) by descending chromatography and by Throckmorton (1962, The University of Texas Publication G205:415-487) by paper electrophoresis of the testes.

Slizynski, B. M. Edinburgh University, Scotland. Detaching of coverslips from albuminised slides.

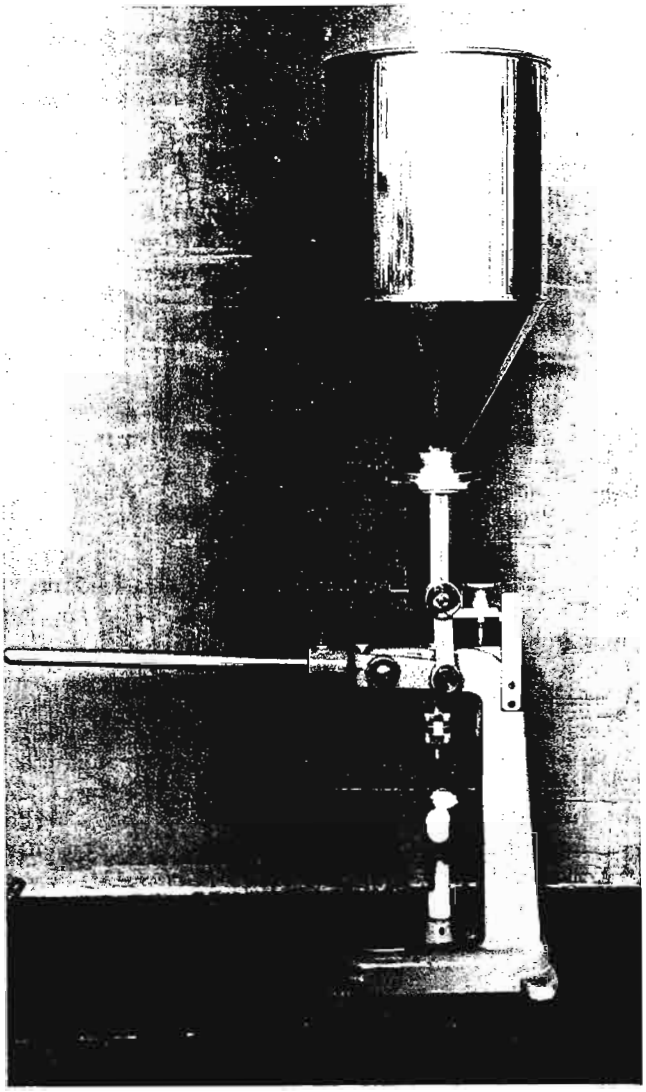
When a squash preparation is made on an albuminised slide, the coverslip usually sticks at the edges of the preparation. It has been found that this can be avoided by making the albuminised area smaller than the coverslip. Albuminisation is done by a pipette: a drop of egg albumen solution is deposited on the slide (about the center) and dried up under cover. The best results (no protruding edges of the albumen) are obtained using a 1:200 solution of egg albumen in distilled water. The coverslip then detaches readily from the slide in 50-60% alcohol.

Sang, J. H. Sussex University, Brighton, England. Clearing Drosophila adults.

Scoring the numbers of melanotic tumors in Drosophila adults by dissecting them out is extremely laborious, and not 100 per cent accurate. On the other hand, the usual clearing procedures involve a complex routine, which is necessary for preserving the tissues but unnecessary for exposing these tumors. A 150 per cent solution of fructose (W/V), with a crystal of thymol per 25 ml. as antiseptic, proved ideal for the purpose, and flies immersed in the solution were completely cleared in 36-48 hours at room temperature. The solution can be used for other purposes where integrity of the tissues is not essential.

Beardmore, J. A. Genetical Institute, Haren, Netherlands. Medium dispensing machine.

Although several dispensers for Drosophila medium have been described, it may be of interest to some workers to know of the existence of a machine, which, without modification, can be used by relatively unskilled personnel. The reservoir holds 15 l of medium and this can be dispensed (lever operation, see accompanying photograph) very accurately and quickly in volumes varying from 1 - 80 ml; resetting of the volume control is very simple. The machine will take media varying widely in viscosity though it may be necessary to change the nose piece to avoid drips or clogging. Provided that it is scrupulously cleaned after use, the chance of appreciable wear or damage with normal use is very small as most moving parts are made of stainless steel.



'Perpetua' filling machine model Hl, Engler Maschinenfabrik, Brunner und Co., Rissaweggasse 12-14, Vienna X/75, Austria; cost in Europe c $230).

McCarron, S. M. J. and K. E. Fuscaldo. Hahnemann Medical College, Philadelphia. Double-diffusion technique for single flies.

The following method has been found satisfactory for double-diffusion tests utilizing a single fly against appropriate antisera. Groundedge cover glasses (2" x 2") are flamed, coated with 2 ml of 0.5% Oxoid Ionagar No. 2 in distilled water, and dried overnight to a film. These precoated slides are then covered with 2 ml of a 1.0% solution of agar in buffered saline (0.005 M. phosphate, pH 7.4, 0.85% Na Cl, 0.1% thimerosal), and allowed to gel in a petri dish provided with a strip of wet filter paper. A central antiserum well and 6 outer antigen wells are cut with the aid of a template and cutter, and the gel is removed from the wells with a small spatula; no sealing gel is necessary.

Single etherized flies are crushed with a glass rod in individual Durham tubes. After about 100 strokes, 0.04 ml of buffered saline is added and homogenization continued to at least 200 strokes. This provides a 0.03 ml antigen dose for one well. The small beaker supporting the Durham tubes is kept in an ice bath until all of the antigen has been delivered to the plate. A total of 0.03 ml of antiserum is added to the central well and the plate is allowed to develop for 4 to 6 days.

If the distance between well centers is made 8 mm and the well diameter 4 mm, the reactants may be delivered in 3 doses of 0.01 ml at approximately 2-hour intervals. If the well geometry is increased to 10 or 13 mm to allow for a well diameter of 6 mm, the complete 0.03 ml of reactants may be delivered at one time.

When precipitation is complete the slide is washed in tap water to remove excess protein from the wells. Unprecipitated protein is eluted by soaking the slide in buffered saline for 24 hours, followed by 2 rinses of 1/2 hour each in distilled water. Slides are stained for 20-30 minutes in Crowle's triple stain (Immunodiffusion, 1961, Academic Press) or in dilute water-soluble nigrosin, destained in 1% acetic acid, and air dried to a film. The finished slide may be used directly in the photographic enlarger to prepare prints, and is in itself a convenient permanent record of the test.

Richardson, R. H. University of Texas.
An improved technique for fecundity and hatchability tests.

A new technique of collecting eggs for fecundity or hatchability tests has been devised, which has the following advantages: homeogeneous egg laying surface resulting in uniform egg distribution, rapidly and easily dispensed medium, medium lacking extraneous food components (such as charcoal), transparent medium allowing scoring of burrowing larvae, and easily cleaned and reused equipment.

The medium consists of 1 g. Bacto-agar, 100 ml. water and 15 ml. white Karo syrup, which is dispensed with an automatic syringe while hot. This medium is then sprayed with a water suspension of bakers yeast immediately before use.

The equipment consists of two variations on the same theme. One variation supplies a black background to facilitate counting. The other presents a transparent background, which allows visual examination of eggs without the removal of the cap from the test bottle.

The test bottles are constructed from 40 dram Plastainer bottles (ca. 2" x 3 1/4") available from Owens-Illinois Glass Co., Toledo, Ohio, at a cost of about $5 per carton (6 dozen). Extra caps are available at about $20 per thousand. The screw caps are made of Teflon and the bottle of clear plastic. A hole is punched in the cap top with a die about 1 1/4" in diameter, and then a piece of plexiglass 1/16" thick is glued to the outer surface of the cap over the hole. The plexiglass may be either black or transparent, giving the two varieties of background. A critical factor in construction is the cement for glueing the cap and the plexiglass. The most satisfactory one tried was Eastman 910 adhesive, available from the Tennessee Eastman Company, Kingsport, Tennessee, at a cost of $8 per bottle. One bottle is sufficient to glue about 400 caps. Also the surface of the cap must be roughened with hardware cloth or a file before glueing. The glue is spread in a very thin band completely around the hole in order to get a water-tight seal. Leaks may be sealed with a band of Duco cement around the external cap-plexiglass junction.

Counting is easily accomplished by marking the agar surface into regions with a blunt needle under about 40X magnification or less. Eggs or larvae may be conveniently transferred to food bottles by transferring agar and eggs or larvae with a small spatula (eg., No. 19240, Curtin Cat. 40) bent at a convenient angle to work inside the cap. Larvae may crawl off the agar surface, but for caps changed every 24 hours or so, it is not a serious problem. Empty egg cases are easily distinguished from unhatched eggs.

An additional advantage of this technique is the practicality of a permanent photographic record of the egg production or hatchability, especially since the eggs are well spread over the surface. The quickest technique using the transparent plexiglass caps in a "contact print" of the cap on photographic paper (available in bulk rolls about 4 1/4" wide) where the shadow of the egg is recorded. Enlargement prints are possible by placing the cap in the film plane of a darkroom enlarger. More detailed records may be made by microfilming the black plexiglass caps with a 35 mm. camera. Examination of the negative either in a microfilm reader or under a dissecting scope allows easy egg counts, hatch scores, or even some egg development scores. It appears counts could even be made by visual scanners in use by automatic data processing systems.

## PERSONAL NEWS

H. Bentley Glass, Professor of Biology at The Johns Hopkins University, is now Vice President for Academic Affairs and Distinguished Professor of Biology at the State University of New York, Stony Brook, Long Island, N.Y.

Hans Laufer, Assistant Professor of Biology at The Johns Hopkins University, is now Associate Professor of Biology at the University of Connecticut, Storrs, Conn.

William J. Welshons has joined the Department of Genetics, Iowa State University, as Head. He will continue his studies on the structure of the Notch locus in Drosophila, and will collaborate in the utilization of existing mouse stocks and facilities for cytogenetic and genetic purposes.

Donald J. Nash has moved from the Department of Zoology, Rutgers University to the Department of Radiology and Radiation Biology, Colorado State University in Fort Collins, Colorado.

Francis M. Butterworth is on an N.I.H. postdoctoral fellowship in the Department of Biology of the University of Virginia in Charlottesville where he will work on the developmental genetics of apterous.

John C. Lucchesi has moved to the Department of Zoology, University of North Carolina, Chapel Hill, North Carolina.

W. J. Peacock has moved to the Genetics Section, Division of Plant Industry, C.S.I.R.O., Canberra, A.C.T., Australia.

Shelia J. Counce has joined the Zoology Department at Duke University, Durham, N. C., as a Research Associate, where she will continue her work on the experimental analysis of insect development.

Nortin M. Hadler, formerly an Undergraduate Research Participant in Biology at Yale, is now at the Harvard Medical School.

Imogene Schneider has moved to the Department of Entomology, Walter Reed Army Research Institute, Washington, D.C., where she is continuing her work on insect tissue culture.

Kugao Oishi is on leave from the National Institute of Genetics, Misima, Japan, to conduct research in the Department of Biology at Yale University.

Roger Milkman will be spending the 1966-67 academic year with Carroll Williams at Harvard. He plans to work on puffing in D. m.

Hans Burla of the University of Zürich is spending a sabbatical year at the University of Oregon.

Anne Chandley has returned to the Bateman Lab., Manchester, England, after 6 months at Cornell University Medical College, New York and 8 months at the Biology Division, Oak Ridge.

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.

Ch. Malogolowkin-Cohen advises correspondents and publishers that reprints and letters to him should be mailed from now on to P. O. Box 1064, Tel-Aviv, Israel.

H. Goldin of the University of Buffalo is spending a year of post-doctoral work at the University of Oregon.

M. Myszewski, formerly of Michigan State University, is spending a year of post-doctoral work at the University of Oregon.

Takashi Narise has returned from the University of Chicago, Illinois, to the National Institute of Genetics, Misima, Shizuoka-Ken, Japan, and is continuing work on the migration and competition of Drosophila.

Sumiko Narise has returned from the University of Chicago, Illinois, to the National Institute of Genetics, Misima, Shizuoka-Ken, Japan, and is continuing work on biochemical genetics.

W. Scharloo has moved from Leiden to join the Drosophila group in Groningen.

B. Wallace is spending six months in Groningen as a guest worker.

M. Kawabe, University of Kobe, Japan, is spending one year from September, 1965, at the Department of Human Genetics, Michigan State University.

A. T. Natarajan has proceeded to Sweden to take up a position in the institute of Radiobiology, Royal University of Stockholm, for a period of two years.

E. Inagaki, Division of Genetics, National Institute of Radiological Sciences, Japan, is spending one year from April 1, 1965, as a Guest Investigator at the laboratory of I. I. Oster, the Institute for Cancer Research, Pennsylvania.

Alexander Sokoloff has taken up a position at the new State College at San Bernardino. Until 1967, when a biology building is completed, he will continue his research on the genetics of Tribolium at Berkeley.

Toshio Shiomi, formerly of the National Institute of Radiological Sciences, Chiba, has moved to the Research Institute for Nuclear Medicine and Biology, Hiroshima University, as Assistant Professor, where he is constructing a new Drosophila Laboratory of Radiation Genetics and continuing studies on radiation mutagenesis and physiological genetics in Drosophila.

Theodore R. F. Wright, formerly Assistant Professor in the Biology Department of The Johns Hopkins University, has joined the faculty of the Department of Biology, University of Virginia, Charlottesville, as an Associate Professor and is continuing his research on the biochemical and developmental genetics of Drosophila.

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## LABORATORY NEWS

As of September 1, 1965, Fenn College in Cleveland was transferred from private to state ownership. It is now known as the Cleveland State University.

The Department of Genetics of the Faculty of Medicine of the University of Chile has been created under the direction of Prof. Danko Brncic. The Department plans to develop research and teaching of Genetics both at the basic as well as at the applied human level. At the moment the two main research projects under way deal with Drosophila and human population genetics.

The stocks of various Drosophila species of Colombia which have been maintained in the laboratory of A. S. Hunter at the Universidad de los Andes in Bogotá are being discontinued. In 1966 new collections will be made and new stocks started at the Centro Experimental de Estudios Superiores in Barquisimeto, Venezuela where the Hunters will be located in a new Department of Biology. As in the past with Colombian Drosophila, we will be glad to supply interested persons with the Drosophila species available in Venezuela.

G. R. Johnson, University of Wyoming, strong allele for bobbed. The gene for bobbed bristles is often useful because of its proximal location on the X-chromosome. Since the expression of bobbed is usually reduced over the years, it might be helpful to know that an allele with strong expression exists in the tu-h stock maintained by the Department of Zoology at Utah State University. Flies with the genotype Df(1)bb/bb(tu-h stock) have bobbed bristles, abnormal abdomens, and increased development time.

K. E. Fuscaldo, Department of Genetics, Hahnemann Medical College, Philadelphia, Pennsylvania, would appreciate receiving stocks with deficiencies for bands 3C1 or 3C2 which may have been obtained in the course of your work.

Alexander Sokoloff would appreciate receiving reprints on current work on Drosophila to supplement the temporarily inadequate library of the new California State College.

Marvin Seiger of the new Dayton Campus of the Ohio State University requests reprints to build up the departmental library.

F. M. Ball, Dept. of Biological Sciences, San Fernando Valley State College, Northridge, California, is maintaining a set of stocks of D. pseudoobscura (see DIS 39:79) and will be happy to furnish them to anyone interested.

J. L. Ménsua, Cátedra de Genética, Universidad de Barcelona (Spain), would appreciate receiving melanogaster stocks selected for high and low number of abdominal bristles, and the same for sternopleural bristles.

M. Dharmarajan of the Department of Animal Genetics, Madras Veterinary College, Madras 7, India, is anxious to build up stocks and would welcome supplies of stocks especially of D. ananassae from different regions.

Sara H. Frye, P.O. Box 267, Irvine, Kentucky, would like to encourage any investigator or investigators (irrespective of sex, nationality, religion, or race) to request the y ac In49 $B^{M1}$ or ac y In49 $B^{M1}$ stock (see Frye, research note, this issue, and Frye, new mutants, this issue) from either the Pasadena or Philadelphia stock center for cytological examination. Any reprints will be appreciated.

## IMPORTANT NOTICE

The stock center supported by the National Science Foundation and presently located in the division of Chemotherapy at the Institute for Cancer Research (Philadelphia) will be transferred to the Department of Biology, Bowling Green State University, Bowling Green, Ohio, 43402. It will continue under the direction of I. I. Oster, who has accepted a position as Associate Professor at Bowling Green State University. All stock requests should be sent to the Ohio address after February 1, 1966. The other center, under the direction of E. B. Lewis, will continue to be maintained at Pasadena.

# GEOGRAPHICAL DIRECTORY

## ARGENTINA

Buenos Aires: Argentine Atomic Energy Commission, Department of Radiobiology, Tel. No. 70-7711, Ext. 59.

Kirschbaum, Werner F. B.Sc.Agr. Research Associate. Salivary cytology.
Leon, Williams N. Technical Assistant.
de Marinic, Susana E. (Mrs.) Research Assistant.
Mazar Barnett, Beatriz (Mrs.) Doctora en Ciencias Naturales. Chemical induction of mutations.
Muñoz, Enzo R. M.D. Research Associate. Radiation genetics.
Paz, Carmen (Miss) Research Assistant. Curator of stocks.
Pereyra, Edith (Miss) Research Assistant.
Valencia, Ruby M. (Mrs.) Ph.D. Chief of Genetics Division. Radiation genetics.

Buenos Aires: Universidad de Buenos Aires, Facultad de Ciencias Exactas y Naturales, Laboratorio de Genetica I. (DIS 40)

## AUSTRALIA

Adelaide, South Australia: University of Adelaide, Department of Genetics.

Hayman, D. L. Ph.D. Lecturer. Recombination, melanogaster.
Levy, Anne (Miss) M.Sc. Demonstrator. Population studies, melanogaster.
Mayo, M. Jean (Mrs.) Ph.D. Lecturer.

Adelaide, South Australia: University of Adelaide, School of Biological Sciences at Bedford Park.

Boettcher, B. B.Sc. Biochemical genetics, immunogenetics.
Clark, A. M. Professor. Mutation.
Clark, E. G. (Mrs·) Curator of Stocks.
Lloyd, B. (Miss) Technical Assistant.

Brisbane, Queensland: University of Queensland, Department of Zoology, Genetics Laboratory.

Angus, D. B.Sc.(Hons.) Graduate student. Chromosomal polymorphism, speciation.
Baimai, V. B.Sc. Graduate student.
Bock, I.R. B.Sc. Graduate student.
Butler, Sheridan Research Assistant.
Dines, Janice Research Assistant.
Kahn, F. M. M.Sc. Graduate student (Pakistan). Cytogenetics.
Mather, Wharton B. Ph.D. Reader. Chromosomal polymorphism, speciation.
Spurway, Rosalyne Research Assistant.

Hobart, Tasmania: University of Tasmania, Department of Botany. (DIS 40)

Melbourne, Victoria: Monash University, Department of Zoology. (DIS 40)

Melbourne, Victoria: University of Melbourne, Departments of Zoology and Genetics, Tel. No. 34-0484, Ext. 2472.

Gunson, Mary M. (Miss) M.Sc. Lecturer. Salivary gland chromosomes.
Hosgood, Sally M. (Miss) B.Sc. Research Student. Ecological genetics.
Kaul, Dipika (Miss) M.Sc. Research Student. Population genetics of D. pseudoobscura.
McBean, I. B.Sc. Research Student. Behavioral genetics.
Parsons, P. A. Ph.D. Reader. Population and behavioral genetics.
Strangio, V. A. Ph.D. Assistant Lecturer. Radiation genetics.
Thomson, J. A. Ph.D. Senior Lecturer. Insect cell culture.

Sydney, New South Wales: Commonwealth Scientific and Industrial Research Organization, Animal Genetics Division, North Ryde.

Bong Kug Ohh B.Sc., M.S. (Seoul University), M.S. (Minn.). Research Student. Canalization, animal breeding.
Finlay, D. B.Sc.Agr. Canalization.
Kindred, B. M (Miss) B.Sc., M.Sc. Canalization.
Rendel, J. M. B.Sc., Ph.D. (Chief). Population genetics, canalization.
Sheldon, B. L B.Sc.Agr., Ph.D. Quantitative genetics, canalization, mutation studies.
Young, S. S. Y. B.Sc., Ph.D. Quantitative genetics, canalization studies.

Sydney, New South Wales: University of Sydney, Department of Animal Husbandry, Tel. No. 68-0522, Ext. 2184.

Barker, J. S. F. B.Agr.Sc., Ph.D. Senior Lecturer in Animal Genetics. Quantitative genetics, population genetics.
Frankham, R. B.Sc.Agr. Research Student. Quantitative genetics.
Hollingdale, B. J. (Miss) B.Sc.Agr. Research Student. Quantitative genetics.
Jones, L. P. B.Sc. Research Student. Quantitative genetics.
Podger, R. N. B.Agr.Sc. Research Assistant. Population Genetics.
Rathie, K. A. B.Sc.Agr. Research Student. Quantitative genetics.
Sheridan, A. K. M.Sc.Agr. Research Student. Quantitative genetics.

Sydeny, New South Wales: University of Sydney, Department of Agriculture.

Baker, E. P. B.Sc.Agr., Ph.D. Senior Lecturer. Population genetics.
McKinley, A. B. Sc.Agr. Teaching Fellow. Teaching.

## AUSTRIA

Vienna: University of Vienna, Department for General Biology, Tel. No. 42-27-67.

Karlik, Anna (Miss) Ph.D. Melanogaster, population genetics.
Kunze-Mühl, Elfriede (Mrs.) Ph.D. Cytogenetics.
Mainx, Felix Ph.D., M.D. Professor, Head of Department.
Parkash, Om Ph.D., M.D. Radiation genetics.
Ruderer-Doschek, Elfriede (Mrs.) Ph.D. Megaselia genetics.
Sperlich, Diether Ph.D. Population genetics, Subobscura cytogenetics.
Springer, Robert Ph.D. Megaselia genetics, Subobscura sexual behavior.

## BELGIUM

Louvain: The University, Parc d'Arenberg, F. A. Janssens Laboratory for Genetics. (DIS 40)

## BRAZIL

Pôrto Alegre: Universidade do Rio Grande do Sul, Departamento de Genética, Caixa Postal 1953.

Araujo, A. M. de Graduate student. Fellow of Universtiy C. of Res. Human Genetics.
Castro, Ignes de Research Assistant. Human genetics, haptoglobins.
Cordeiro, E. R. Technician.
Fernandes, Maria I. de M. Bc.Sc.Lic. Research Assistant, Head of the Laboratory of Vegetal Genetics.
Ferreira, Therezinha F. Agron.Eng. Research Assistant. Vegetal genetics.
Gomes, Norma B. Graduate student. Fellow of Brazil C. of Res. (CNPq). Drosophila populations.
Kalisz, Alice Graduate student. Fellow of Brazil C. of Res. (CNPq). Disruptive selection, Drosophila populations.
Leães, Anamaria P. Graduate student. Fellow of Brazil C. of Res. (CNPq). Cytology, Drosophila.
Lewgoy, F. Chem. Eng. Research Assistant. Biophysical genetics.
Ludwig, Maria da R. Technician.
Ludwig, Nilda C. Administrative Assistant.
Machado, Dinarcy M. Administrative Assistant.
Mallmann, Maria Clara Graduate student. Fellow of University C. of Res. Human. Cytology.
Marques, E. K. Bc.Sc.Lic. Head of the Animal Genetics Laboratory. Genetic effects of radiation on Drosophila populations.
Mendez, Heirie Graduate student. Biophysical genetics.
Napp, Marly Bc.Sc.Lic. Research Assistant. Polymorphism, disruptive selection, Drosophila populations.
Nardon, Rozmary Graduate student. Fellow of Brazil C. of Res. (CNPq). Disruptive selection, Drosophila populations.
Pereira, Sirlei Graduate student. Fellow of Brazil C. of Res. (CNPq). Genetic analysis, radiation, Drosophila populations.
Peres, Agueda P. Technician.
Possani, L Graduate student. Fellow of University C. of Res. Biophysical genetics.
Ramila, D. Technician. Foodmaker.
Rocha, F. J. da Research Assistant. Human genetics, hemoglobins.
Rocha, Zita M. A. da Technician.
Salzano, F. M. Ph.D. Head of the Department of Genetics. Human blood groups, Indian Population genetics.
Santos, Tânia M. M. dos Graduate student. Fellow of Brazil C. of Res. (CNPq). Drosophila ecology.
Silva, L. C. da Technician Electronics.
Silva, M. M. da Administrative Assistant.
Silveira, Francisca M. da Graduate student. Cytotaxonomy.
Simões, C. V. Technician.
Simões, G. V. Technician. Field worker. Human genetics.
Simões, Neiva Technician. Vegetal genetics.
Soares, Beloni Technician.
Suñe, Margarete V. Bc.Sc.Lic. Research Assistant. Cytology, human genetics.
Thedy, O. P. Technician. Drosophila populations.
Tondo, C. V. E.E.Bc.Sc. Head of Biophysical Laboratory. Hemoglobins.
Veiga-Neto, A. J. Graduate student. Fellow of University C. of Res. Ecology, Drosophila populations.
Xavier, Juracy Technician.

São José do Rio Prêto, São Paulo: Govêrno do Estado de São Paulo, Faculdade de Filosofia, Ciências e Letras, Departamento de Biologia Geral. (DIS 40)

## CANADA

Vancouver, British Columbia: University of British Columbia, Department of Zoology, Tel. No. (604) 228-2131, Ext. 54.

Astell, Caroline (Miss) B.S. Graduate student. Cytology of chromosome structure.
Baillie, David Undergraduate honors. Chromosome mechanics.
Dudley, Mary (Miss) Undergraduate honors. Chromosome mechanics.
Garland, Maureen (Mrs.) B.S. Graduate student. Chromosome mechanics.
Giroux, Joan (Miss) Undergraduate. Chemical mutagenesis.
Haddow, Douglas Undergraduate honors. Chromosome mechanics.
Hayashi, Susi (Miss) B.S. Research assistant.
Hoar, David. Undergraduate honors. Cytogenetics.
Korinek, Ella (Mrs.) Research technician.
Piternik, Leonie (Mrs.) Ph.D. Gene action.
Rigby, Brian Honors student. Chromosome mechanics.
Scholefield, Jane (Miss) B.S. Graduate student. Chromosome mechanics.
Suzuki, David Ph.D. Associate Professor. Chromosome mechanics.
Takahashi, Judy (Miss) Undergraduate honors. Chemical mutagenesis.

## CHILE

Santiago: Universidad de Chile, Departamento de Genética, Instituto de Biología "Juan Noé", Av. Zañartu 1042.

Barrera, R. Research Assistant. Human genetics.
Brncic, D. Professor. Population genetics.
Covarrubias, Edmundo M.D. Research Associate. Human genetics.
Del Solar, Eduardo Research Assistant. Ecological genetics. (now at Rockefeller Institute, New York, U.S.A.)
Fenner, Hertha (Mrs.) Technical Assistant
Fernández, Raúl Research Assistant. Cytogenetics.
Iturra, Patricia (Miss) Technical Assistant.
Koref-Santibañez, Susi (Mrs.) M.D. Research Associate. Population genetics, isolating mechanisms.
Palomino, Hernán Graduate student. Ecological genetics.
Rojas, Eliana (Miss) Technical Assistant, Secretary.

## COLOMBIA

Bogotá D. E.: Universidad de Los Andes, Instituto de Genetica.

Bautista, E. B.Sc. Research Associate. Biochemistry of systematics.
Castro, L. Research Assistant. Population genetics.
Cortés, B. I. Technician. Biochemistry of systematics.
Chejne, A. J. Research Assistant. Biochemistry of systematics.
German, E. Hortobay Research Assistant. Population genetics.
Granobles, L. A. B.Sc. Research Assistant. Population genetics
Hoenigsberg, H. F. B.A., Ph.D. Population genetics.

Medina, L. Technician. Biochemistry of systematics.
Mojica, T. Technician. Population genetics.
Navas, Y. G. B.Sc. Research Assistant. Population genetics.
Lavende, A. Technician. Population genetics.
Romoro, I. F. Technician. Biochemistry of systematics.
Marin, L. M. Stock curator.

## CZECHOSLAVAKIA

Prague 6: Institute of Experimental Botany, Flemming's Square 2.

Landa, Z. Research worker. Chemical mutagenesis.
Ondřej, M. Research student. Crossing over, mutagenesis.

## DENMARK

Copenhagen: University of Copenhagen, Institute of Genetics, 2A Øster Farimagsgade, K. Tel. No. PA 9255.

Anderson, B. (Mrs.) Technical Assistant
Bahn, Erik Ph.D. Amylase variants.
Frydenberg, Ove Ph.D. Population genetics.
Sick, Knud Ph.D. Population genetics.
Wettstein, D. von Professor. Head of Institute.

## FINLAND

Helsinki: University of Helsinki, Institute of Genetics, P. Rautatiekatu 13, Tel. No. 444562.

Hackman, Robin B.A. Research Associate. Developmental genetics.
Lakovaara, Seppo Ph.D. Assistant Teacher. Developmental genetics, population genetics.
Suomalainen, Esko Ph.D. Professor. Head of Department.
Tiivola, Airi (Mrs.) Technical Assistant, Curator of Stocks.

Turku: University of Turku, Department of Genetics.

Arajärvi, Pirkko (Mrs.) Cand. Nat. Sc. Research Assistant. Melanogaster: mutations, salivaries.
Hannah-Alava, Aloha (Mrs.) Ph.D. Research Associate. Melanogaster: developmental genetics, mutations.
Oksala, T. A. Ph.D. Professor, Head of Department. Melanogaster: mechanism of segregation, interchromosomal effects.
Puro, J. Lic. Phil. Assistant teacher. Melanogaster: mutations.
Portin, P. Mag. Phil. Melanogaster: mechanism of segregation.
Savolainen, Salme (Mrs.) Technical Assistant.
Savontaus, Marja-Liisa (Mrs.) Mag. Phil. Melanogaster: mechanism of segregation.

## FRANCE

Clermont-Ferrand, Puy de Dôme: Laboratoire de Zoologie, Faculté des Sciences, 1 Avenue Vercingétorix.

Perrin-Waldemer, Cl. Assistant. Biology of spermatozoa in Drosophila.

Gif-Sur-Yvette: Centre National de la Recherche Scientifique, Laboratoire de Génétique des Virus.

Bernard, J (Miss) Assistant. $CO_2$ sensitivity in Drosophila.
Gay, P. Attaché de recherches. $CO_2$ sensitivity in Drosophila.
Herreng, F. (Miss) Assistant. Multiplication of Sindbis virus in D. melanogaster.
L'Heritier, Ph. Professor. Head of the department. $CO_2$ sensitivity in Drosophila.
Ohanessian-Guillemain, A. (Mrs.) Chargée de recherches. $CO_2$ sensitivity in Drosophila. Tissue cultures of D. melanogaster.
Plus, N. (Mrs.) Maître de recherches. $CO_2$ sensitivity in Drosophila. Biochemical properties of Drosophila virus.
Printz, P. Assistant. $CO_2$ sensitivity in Drosophila.

Lyon, Rhône: Faculty of Sciences, Laboratoire de Zoologie générale, Tel. No. 72 05 45.

(only the personnel working with Drosophila are listed)
Clavel, M. F. (Miss) Technician. Temperature effects.
David, J. R. Maître de Conférences. Nutrition and physiology of adults.
Javellot, M. P. (Miss) Graduate student. Physiology of vestigial.
Merle, J. (Miss) Graduate student. Physiology of virgin females.

91-Orsay: Université de Paris, Institut de Biologie expérimentale.

Brun, G. Professor. $CO_2$ sensitivity in Drosophila.
Bussereau, F. (Miss) Assistant. $CO_2$ sensitivity in Drosophila.
Proust, J. (Mrs.) Chargée de recherches. UV mutagenesis in Drosophila.
Prudhommeau, C. Assistant. $CO_2$ sensitivity in Drosophila.
Teninges, D. (Mrs.) Attachée de recherches. UV mutagenesis in Drosophila.
Vigier, Ph. Maître-assistant. $CO_2$ sensitivity in Drosophila.

91-Orsay: Université de Paris, Faculté des Sciences, Laboratoire de Biologie générale.

Bregliano, J. C. Maître-assistant. $CO_2$ sensitivity in Drosophila.

Paris: Université de Paris, Faculté des Sciences, Laboratoire de Zoologie, 7 quai Saint-Bernard, Tel. No. DAN 07 25. (DIS 40)

Strasbourg (Bas-Rhin): Faculté des Sciences, Laboratoire de Zoologie. (DIS 40)

Lyon, Villeurbanne: Faculté des Sciences, Laboratoire de Zoologie Experimentale, 43, Boulevard de l'Hippodrome.

Bouzenot, M. J. (Miss) Contractuelle C. E. A. Unfavorable mutations in Diptera.
Brun, J. Maître-Assistant. Cytology and Genetics of Nematodes.
Daillie, J. Maître-Assistant. Nucleic acid metabolism.
Fourche, J. Maître-Assistant. Respiratory metabolism in Drosophila.
Gillot, S. (Mrs.) Assistant. Ovogenesis in insects.
Godet, J (mrs.) Maître-Assistant. Cellular differentiation.
Guerrier, P. Maître-Assustant. Cytology of Nematodes.
Legay, J. M. Professor. Physiology and genetics of phytophagus insects.
Mourgues, C. (Mrs.) Assistant. Heritability in Drosophila.
Neulat, M. M. (Miss) Attachée de Recherches. Nucleic acid metabolism.
Nigon, V. Professor, Head of Department. Nucleic acid metabolism.
Ploye, H. Assistant. Genetics of Lebistes.
De Reggi, L. Assistant. Quantitative inheritance.

## GERMANY

Berlin 33 (Dahlem): Institut für Genetik der Freien Universität Berlin, Rudeloffweg 9, Tel. No. 76 90 640.

Bartelt, Jutta Technical Assistant. Melanogaster: radiation genetics.
Belitz, Hans-Joachim (Dr.) Research Assistant. Melanogaster: induced mutations.
Haller, Hans Jürgen Graduate student. Melanogaster; radiation genetics.
Lüers, Herbert Prof. Dr. Director. Comparative genetics; mutagens.
Lüers, Thea (Mrs.,Dr.) Guest Associate. Drosophila neurology.
Nöthel, Horst (Dr.) Research Assistant. Melanogaster: radiation genetics.
Obe, Günter Graduate student. Cytogenetics.
Schereiks, Gisela (Miss) Technical Assistant. Melanogaster: chemogenetics.
Sommerau, Ingeborg (Miss) Technical Assistant. Insects: cytology.
Sperling, Karl Graduate student. Cytogenetics.
Struck, Eva (Mrs.,Dr.) Research Assistant. Insects: cytology.
Wieczorek-Bochnig, Veronika (Dr.) Research Assistant. Curator of stocks. Melanogaster: physiological genetics, radiation genetics.
Wolf, Eroch (Dr.) Associate. Insects: cytology.

78 Freiburg: Zoologisches Institut der Universität.

Anders, Liesel (Mrs.) Technician.
Hess, Oswald Structural modifications of the Y chromosome in D. hydei and their relations to gene activity.
Sander, Klaus Embryology, experimental analysis of development.

Hamburg 13: Zoologishes Staatsinstitut und Zoologisches Museum, von Melle Park 10. (DIS 40)

Karlsruhe: Kernforschungszentrum, Strahlenbiologie. (DIS 40)

München 2: Zoologisches Institut der Universität, Lusienstrasse 14, Tel. No. 55 79 76, Ext. 359.

Becker, Gweneth L. (Mrs.) Lethals.
Becker, Hans Joachim Puffing and variegation.
Haendle, Jutta (Mrs.) Somatic crossing over.
Janning, Wilfried Position-effect variegation.
Kalisch, Wolf-Ekkehard Pteridines.
Korge, Günter Puffing.
Moritz, Karl General.
Perschmann, Brigitte (Miss) Curator of Stocks.

Münster (Westf.): Universität Münster, Institut für Strahlenbiologie.

Dittrich, Wolfgang Prof. Dr. Radiation biology.
Mertens, Ruth (Miss) Technician.
Traut, Anneliese (Mrs.) Technician.
Traut, Horst Dr. Radiation genetics.
Wind, Heinz Dipl. Biol. Radiation genetics.

Tübingen: Max Planck-Institut für Biologie, Abt. Beermann, Tel. No. 32-47.

Arcos, Laura (Miss) Graduate student.
Beermann, Wolfgang Physiology of salivary gland chromosomes.
Bromberg, Renate (Miss) Technician.
Hennig, Wolfgang Graduate student.
Hilk, Ingrid (Miss) Technician.

Meyer, Günther F. Gametogenesis, light and electron microscopy; fine structure of chromosomes.
Wolstenholme, David R. Ultrastructure of genetic systems.

## GREAT BRITIAN

Birmingham, England: The University, Department of Genetics, Tel. No. SEL. 1301 Ext. 631.

Barnes, B. W. B.Sc. Research student. Environment and selection.
Gale, J. S. Ph.D. Senior Scientific Officer. Competition.
Jinks, J. L. D.Sc. Professor. Psychogenetics, extrachromosomal inheritance.
Kearsey, M. J. Ph.D. Lecturer. Competition, chromosome assays.
Lawrence, M. J. Ph.D. Lecturer. Recombination and selection.
Wilkinson, Patricia. Technical Assistant. Drosophila stocks.

Brighton, Sussex, England: University of Sussex, School of Biology.

Atherton, June (Mrs.) Stock keeper.
Bond, P. A. Ph.D. Research Fellow. Biochemistry of Melanotic tumors.
Bryant, P. J. M.Sc. Research student. Melanotic tumors.
Collett, Janet (Mrs.) M.S. Research Assistant. Physiology of aging.
Cook, Johnathan B.Sc. Research student. Melanotic tumors.
Hastings, Muriel (Miss) B.Sc. Research assistant. D. Nutrition.
Huot, L. Professor. Visiting worker.
Sang, J. H. Professor. Ph.D., FRSE. Physiological genetics, nutrition and tumors.
Shields, Glen M.Sc. Junior Research Fellow. Tissue culture.
Smith, J. Maynard Professor. B.A., B.Sc. Aging and selection studies.

Cambridge, England: University of Cambridge, Department of Genetics, Tel. No. 58694.

Alderson, T. Ph.D. Research worker. Mutagenesis.
Ashburner, M. Research student. Salivary glands.
Davies, W. Research student. Quantitative and Developmental genetics.
Gibson, J.B. Ph.D. Senior Assistant in Research. Quantitative and developmental genetics.
Hudson, B. Laboratory Assistant.
Khan, A. H. Research student. Irradiation, chemical mutagenesis.
Thoday, J. M. Ph.D. Professor. Selection.
Whittle, J. R. S. Research student. Quantitative and developmental genetics.

Chalfont St. Giles, Bucks., England: Chester Beatty Research Institute, Institute of Cancer Research, Pollards Wood, Department of Genetics, Tel. No. Little Chalfont 2530.

Fahmy, Myrtle J. (Mrs.) Ph.D. Mutagenesis
Fahmy, O. G. M.Sc., Ph.D. Cytogenetics.
Lees, R. K. (Miss) B.Sc. Research Assistant.
Lovell, D. M. (Miss) Technical Assistant.
Massasso, J. (Miss) B.Sc. Research student.
de Vrye, C. E. (Mrs.) B.Sc. Research student.
Wilson, F. (Miss) B.Sc. Research Assistant.

Edinburgh 9, Scotland: Institute of Animal Genetics, West Mains Road, Tel. No. NEWington 1011.

Auerbach, C. A. (Miss) D.Sc., F.R.S. Reader. Mutagenesis.
Bartjelmess, I. (Miss) Ph.D. Guest investigator. Quantitative variation in pteridines.

Basden, E. B. Research Assistant. Wild species.
Brink, N. G. Ph.D. Guest investigator. Mosaicism.
Chipchase Birnstiel, M. (Mrs.) Drosophila nucleic acids.
Chopra, U. L. B.Sc. (Hons) Assoc. I.A.R.I. Graduate student. Genetical effect of irradiated or chemically treated medium.
Clayton, G. Lecturer. Selection.
Futch, D. Ph.D. Guest investigator. Ecological genetics and speciation.
Knight, G. R. Research Assistant. Subobscura salivaries.
Perry, M. (Miss) Research Assistant. Ultrastructure of eggs.
Ratnayake, W. C. B.Sc. Graduate student. Chemical mutagenesis.
Robertson, A. D.Sc., F.R.S. Quantitative genetics.
Robertson, E. (Mrs.) Research Assistant. In vitro and in vivo culture of eye-buds, effects of antibiotics.
Robertson, F. W. D.Sc. Population and physiological genetics.
Slizynska, H. (Mrs.) Ph.D. Cytological analysis.
Slizynski, B. M. Ph.D. Salivaries.
Strachan, K. (Miss) Stock-keeper.
Waddington, C. H. Sc.D., F.R.S. Head of Department. Evolutionary and developmental genetics.

Harwell, Berks., England: Radiobiological Research Unit, Department of Drosophila Genetics, Tel. No. Rowstock 393, Ext. 57.

Calder, J. E. (Mrs.) Technical Assistant.
Cox, J. T. (Miss) Technical Assistant.
Dyer, K. F. B.Sc. Research Assistant.
Lamb, M. J. (Miss) Ph.D. Radiation and aging.
McSheehy, T. W. B.Sc. Radiation genetics.
Pearce, S. (Miss) Technical Assistant.
Purdom, C. E. Ph.D. Radiation genetics.

Hertford, England: John Innes Institute, Department of Genetics, Tel. No. 3191.

Harrison, B. J.

Keele, Staffordshire, England: University of Keele, Department of Biology, Tel. No. Keele Park 371.

Goodway, K. M. Dr. Ecological genetics.

Lancaster: The University, Department of Biology.

Paxman, G. J. Ph.D. Senior Lecturer in Genetics.

Leicester, England: Leicester University, Department of Genetics, Tel. No. 50000, Ext. 146.

Cronenwett, Carolyn S. (Mrs.) Ph.D. Lecturer. Microbial genetics.
Hedges, R. W. B.A. Oxon. Assistant Lecturer. Microbial genetics.
Holland, I. B. Ph.D. Lecturer. Microbial genetics.
Pritchard, R. H. Ph.D. Professor. Microbial genetics.

Liverpool 3: The University, Department of Genetics.

Batten, J. L. Assistant Lecturer. Recombinational lethals in D. melanogaster.
Faulkner, B. Ph.D. Lecturer. Extrachromosomal inheritance in fungi.
Gill, J. J. B. Assistant Lecturer. Plant cytology.
Sheppard, P. M. D.Phil., F.R.S. Professor. Population and medical genetics.
Walker, S. Ph.D. Senior Lecturer. Plant and human cytology.

London E.C.I., England: St. Bartholomew's Hospital Medical College, University of London, Charterhouse Square, Department of Zoology, Tel. No. CLErkenwell 9237.

Hollingsworth, M. J. Ph.D. Lecturer. Physiology of aging and temperature acclimatization

in Drosophila.

London, England: University College, Department of Biometry, Eugenics and Genetics, (DIS 40)

London, England: University College, Department of Zoology. (DIS 40)

Manchester 20, England: Paterson Laboratories, Department of Cytogenetics, Tel. No. DIDsbury 8123, Ext. 383.

Bateman, Angus J. Ph.D., D.Sc. Mutation and recombination.
Chandley, Ann C. (Miss) M.Sc. Oogenesis and recombination.

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## ISRAEL

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Ehrman, Lee (Mrs.) Assistant Professor. Reproductive isolating mechanisms: paulistorum.
Kastritsis, Costas Ph.D. Research Associate. Chromosomal evolution: paulistorum.
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(continued on page 8)

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Bender, H. A. Notre Dame, Indiana
Bennett, J. DeKalb, Illinois
Bennett, K. W. DeKalb, Illinois
Berendes, H. D. Netherlands, Leiden
Berger, E. Syracuse, New York
Berger, H. Switzerland, Zürich
Bernard, J. France, Gif-sur-Yvette
Bernstein-Barzilay, N. Israel, Jerusalem
Bie, V. Netherlands, Leiden
Biggin, W. Philadelphia, Pennsylvania
Blaylock, B. G. Oak Ridge, Tennessee
Bleecker, W. M. Ames, Iowa
Blount, J. L. Alliance, Ohio
Boam, T. B. England, Sheffield
Bock, I. R. Australia, Brisbane
Bodenstein, D. Charlottesville, Virginia
Boettcher, B. Australia, Adelaide
Bogdanovich, M. St. Louis, Missouri
Bond, P. A. England, Sussex
Bong Kug Ohh Australia, Sydney
Boshes, R. Chicago, Illinois
Bosma, A. Netherlands, Utrecht
Bouzenot, M. J. France, Lyon
Bowler, J. A. Philadelphia, Pennsylvania
Bowman, J. T. Logan, Utah
Boyd, E. Raleigh, North Carolina
Boyd, T. Detroit, Michigan
Brand, J. W. Netherlands, Leiden
Brändle, E. Switzerland, Zürich
Bregliano, J. C. France, Orsay
Brewster, T. Lincoln, Nebraska
Briggs, J. D. Columbus, Ohio
Brink, N. G. Scotland, Edinburgh
Brncic, D. Chile, Santiago
Bromberg, R. Germany, Tübingen
Brooks, G. T. Columbus, Ohio
Brosseau, G. E. Iowa City, Iowa
Brown, D. Riverside, California
Brown, E. H. Urbana, Illinois
Brown, J. Raleigh, North Carolina
Brown, M. Syracuse, New York
Brown, W. P. Marietta, Ohio
Bruck, D. L. Chicago, Illinois
Brun, G. France, Orsay
Brun, J. France, Lyon
Bryant, P. J. England, Sussex
Buchanan, J. S. Cold Spring Harbor, N. Y.
Bull, A. L. Roanoke, Virginia
Bunker, M. C. Bar Harbor, Maine
Burckhardt, B. Switzerland, Zürich
Burdick, A. B. Lebanon, Beirut

Burkholder, N. Duarte, California
Burla, H. Eugene, Oregon
Burnet, B. England, Sheffield
Bussereau, F. France, Orsay
Butler, S. Australia, Brisbane
Butterworth, F. M. Charlottesville, Virginia
Byers, H. M. Chapel Hill, North Carolina
Calder, J. E. England, Harwell
Cama, J. Spain, Barcelona
Campbell, R. Philadelphia, Pennsylvania
Cantafio, J. Detroit, Michigan
Canuti, N. Italy, Rome
Cargile, M. A. Philadelphia, Pennsylvania
Carlin, L. A. Baltimore, Maryland
Carlson, E. A. Los Angeles, California
Carlson, R. Los Angeles, California
Carmody, G. New York, New York
Carpenter, J. M. Lexington, Kentucky
Carson, H. L. St. Louis, Missouri
Carfagna, M. Italy, Naples
Castro, I. de Brazil, Pôrto Alegre
Castro, L. Colombia, Bogotà
Cates, M. Chapel Hill, North Carolina
Cattani, C. East Lansing, Michigan
Chandley, A. C. England, Manchester
Chang, D. H. Korea, Seoul
Chatterjee, K. India, Kalyani
Chejne, A. J. Colombia, Bogotà
Chen, P. S. Switzerland, Zürich
Chigusa, S. Lafayette, Indiana
Childress, D. Eugene, Oregon
Chipchase Birnstiel, M. Scotland, Edinburgh
Cho, S. H. Korea, Seoul
Choi, C. G. Korea, Kwangju
Choo, J. K. Korea, Seoul
Chopra, U. L. Scotland, Edinburgh
Chovnick, A. Storrs, Connecticut
Chun, W. S. Korea, Seoul
Chung, J. Y. Korea, Seoul
Chung, O. K. Korea, Seoul
Clancy, C. W. Eugene, Oregon
Clancy, E. B. Eugene, Oregon
Clark, A. M. Australia, Adelaide
Clark, E. G. Australia, Adelaide
Clark, J. O. Chapel Hill, North Carolina
Clausing, J. A. Ridgefield, Connecticut
Clavel, M. F. France, Lyon
Clayton, F. E. Fayetteville, Arkansas
Clayton, G. Scotland, Edinburgh
Clise, R. Cleveland, Ohio
Cockerham, L. Berkeley, California
Collett, J. England, Sussex
Collins, J. F. Chapel Hill, North Carolina
Conduff, V. Johnson City, Tennessee
Conley, C.A.M. Madison, Wisconsin
Cook, J. England, Sussex
Coomber, R. Logan, Utah
Cooper, D. M. New York, New York
Cordeiro, E. R. Brazil, Pôrto Alegre
Cortés, B. I. Colombia, Bogotà
Corwin, H. Los Angeles, California
Costantino, R. F. Lafayette, Indiana
Coughlin, J. St. Louis, Missouri
Counce, S. J. Durham, North Carolina
Council, S. Raleigh, North Carolina
Counts, C. P. Johnson City, Tennessee
Courtright, J. B. Baltimore, Maryland
Covarrvbias, E. Chile, Santiago
Cox, J. T. England, Harwell
Cronewett, C. S. England, Leicester
Crow, J. F. Madison, Wisconsin
Crumpacker, D. W. New York, New York
Cullen, M. U. New Haven, Connecticut
Cummings, M. Evanston, Illinois
Curry, S. Syracuse, New York
Daillie, J. France, Lyon
Dalebroux, M. Raleigh, North Carolina
Daniel, G.M.J. Madison, Wisconsin
Datta, R. K. India, Calcutta
David, J. R. France, Lyon
Davies, W. England, Cambridge
Dawson, H. Duarte, California
Day, J. W. Oak Ridge, Tennessee
Day, M. H. Charlottesville, Virginia
Dean, M. R. Ithaca, New York
Dearden, M. England, Manchester
De Cindio, O. Italy, Naples
DeForest, D. Detroit, Michigan
Deland, M. Storrs, Connecticut
Del Campo, G. Pasadena, California
De Lerma, B. Italy, Naples
Del Grande Kravina, A. M. Italy, Milan
Del Solar, E. Chile, Santiago
DeMarco, P. Pittsburgh, Pennsylvania
DeMarinis, F. Cleveland, Ohio
Dempsey, T. P. Philadelphia, Pennsylvania
Denniston, C. L. Madison, Wisconsin
Deppe, C. Gainesville, Florida
De Reggi, L. France, Lyon
Dewees, A. A. Lafayette, Indiana
Dharmarajan, M. India, Madras
Dick, J. L. Emporia, Kansas
Dickerman, R. C. Argonne, Illinois
Di Lorenzo, M. Italy, Naples
Dines, J. Australia, Brisbane
Di Pasquale, A. Italy, Milan
Di Sangro, G. Italy, Naples
Dittmar, S. Lafayette, Indiana
Dittrich, W. Germany, Münster
Doane, W. W. New Haven, Connecticut
Dobzhansky, Th. New York, New York
Dolfini, S. Italy, Milan
Dolwick, M. F. Lexington, Kentucky
Donady, J. Iowa City, Iowa
Druger, M. Syracuse, New York
Dübendorfer, A. Switzerland, Zürich
Dudley, M. Canada, Vancouver
Duggal, K. K. India, Punjab
Duggleby, W. F. Madison, Wisconsin
Duke, E. J. Chapel Hill, North Carolina
Du Pui, M.L.L. Netherlands, Haren
Dutta Gupta, A. K. India, Calcutta
Duyn-Goedhart, A. Netherlands, Leiden
Dyer, K. F. England, Harwell
Ebersole, L. A. Columbus, Ohio
Ebitani, N. Japan, Tokyo
Edney, E. Riverside, California
Ehrlich, E. Eugene, Oregon

Ehrman, L. New York, New York
Eiche, A. Sweden, Stockholm
Ellgaard, E. Iowa City, Iowa
Ellis, J. F. Clinton, New York
Elzen, H. van der Netherlands, Utrecht
Engel, J. Eugene, Oregon
Englert, D. C. Carbondale, Illinois
English, D. S. Ames, Iowa
Epler, J. L. Oak Ridge, Tennessee
Erickson, J. Bellingham, Washington
Erk, F. C. Stony Brook, New York
Erway, L. Davis, California
Fahmy, M. J. England, Buckinghamshire
Fahmy, O. G. England, Buckinghamshire
Falk, R. Israel, Jerusalem
Falke, E. V. Ithaca, New York
Farley, B. Bar Harbor, Maine
Farnsworth, M. W. Buffalo, New York
Faulkner, B. England, Liverpool
Faulkner, K. Canberra, Australia
Feher, P. G. Corvallis, Oregon
Félix, R. Mexico, Mexico City
Felsenstein, J. Chicago, Illinois
Fenner, H. Chile, Santiago
Fernandes, M. I. de M. Brazil, Pôrto Alegre
Fernández, R. Chile, Santiago
Ferreira, T. F. Brazil, Pôrto Alegre
Finlay, D. Australia, Sydney
Finnerty, V. G. Storrs, Connecticut
Folkers, W. Netherlands, Haren
Forger, J. Netherlands, Leiden
Forsyth, T. B. Fayetteville, Arkansas
Forward, K. Ann Arbor, Michigan
Fourche, J. France, Lyon
Fox, A. S. Madison, Wisconsin
Fradkin, M. College Park, Maryland
Frankel, A. Iowa City, Iowa
Frankham, R. Australia, Sydney
Fraser, A. S. Davis, California
Fraser, T. Philadelphia, Pennsylvania
Freeborn, J. Ridgefield, Connecticut
Freland, M. Raleigh, North Carolina
Friedberg, E. Israel, Jerusalem
Friedman, L. D. Hiram, Ohio
Fritz, M. Sweden, Stockholm
Froines, A. New Haven, Connecticut
Frydenberg, O. Denmark, Copenhagen
Fuchs, M. S. Madison, Wisconsin
Fujii, S. Japan, Kobe
Fujio, Y. Japan, Anzyo
Fukushi, Y. Japan, Osika
Fuscaldo, K. E. Philadelphia, Pennsylvania
Fusté, M. Spain, Barcelona
Futch, D. Scotland, Edinburgh
Gale, J. S. England, Birmingham
Gardner, E. J. Logan, Utah
Garland, M. Canada, Vancouver
Gartner, L. P. Newark, New Jersey
Gay, H. Ann Arbor, Michigan
Gay, P. France, Gif-sur-Yvette
Geer, B. W. Galesburg, Illinois
Gehring, W. Switzerland, Zürich
German, E. H. Colombia, Bogotà
Gersh, E. S. Philadelphia, Pennsylvania
Gethmann, R. C. Corvallis, Oregon
Geum, C. S. Korea, Seoul
Giacalone, J. New York, New York
Gibson, J. B. England, Cambridge
Giesel, B. J. Eugene, Oregon
Gill, J.J.B. England, Liverpool
Gillot, S. France, Lyon
Gilmore, G. Iowa City, Iowa
Girard, P. Los Angeles, California
Giroux, J. Canada, Vancouver
Glass, H. B. Stony Brook, New York
Glass, S. S. Stony Brook, New York
Glassman, E. Chapel Hill, North Carolina
Gleditsch, A. Norway, Oslo
Gloor, H. J. Netherlands, Leiden
Godet, J. France, Lyon
Goetz, W. Switzerland, Zürich
Goldberg, C. College Park, Maryland
Goldin, H. Eugene, Oregon
Goldschmidt, E. Israel, Jerusalem
Gomes, N. B. Brazil, Pôrto Alegre
Gonzalez, F. W. Madison, Wisconsin
Goodway, K. M. England, Keele
Gotborn, L. Sweden, Uppsala
Gottlieb, D. J. Knoxville, Tennessee
Gottlieb, F. J. Pittsburgh, Pennsylvania
Gowen, J. W. Fort Collins, Colorado
Grabicki, E. New Haven, Connecticut
Grace, D. Los Angeles, California
Granobles, L. A. Colombia, Bogotà
Grant, B. Raleigh, North Carolina
Green, C. P. Evanston, Illinois
Green, M. M. Davis, California
Gregg, T. G. Oxford, Ohio
Grell, E. H. Oak Ridge, Tennessee
Grell, R. F. Oak Ridge, Tennessee
Griffen, A. B. Bar Harbor, Maine
Grimwood, B. DeKalb, Illinois
Groot-van Straler, C. Netherlands, Leiden
Grubbs, C. G. Richmond, Virginia
Guerrier, P. France, Lyon
Guest, W. C. Fayetteville, Arkansas
Gunson, M. M. Australia, Melbourne
Gupta, J. P. India, Varanasi
Gustafson, K. Sweden, Stockholm
Gutenmann, H. Ithaca, New York
Haalman, P. Pittsburgh, Pennsylvania
Hackman, R. Finland, Helsinki
Haddow, D. Canada, Vancouver
Hadorn, E. Switzerland, Zurich
Haefele, J. Corvallis, Oregon
Haendle, J. Germany, Munich
Hale, G. New York, New York
Halfer, C. Italy, Milan
Haller, H. J. Germany, Berlin
Hancock, N. Lafayette, Indiana
Hanimann, F. Switzerland, Zürich
Hanks, G. D. Salt Lake City, Utah
Hannah-Alava, A. Finland, Turku
Harrison, B. J. England, Hertford
Hartl, D. L. Madison, Wisconsin
Hartman, A. Chicago, Illinois
Hartmann-Goldstein, I. J. England, Sheffield
Hartshorne, J. N. England, Manchester

Hastings, M. England, Sussex
Havin, E. Norway, Oslo
Hawkes, N. R. Logan, Utah
Hawkins, E. Los Angeles, California
Hayashi, K. Japan, Osaka
Hayashi, S. Canada, Vancouver
Hayman, D. L. Australia, Adelaide
Hebb, S. Emporia, Kansas
Hedges, R. W. England, Leicester
Heed, W. Tucson, Arizona
Heeke, C. Emporia, Kansas
Heerema, N. Iowa City, Iowa
Heller, F. New York, New York
Henderson, A. S. Chapel Hill, North Carolina
Hendrickson, R. Los Angeles, California
Hennig, W. Germany, Tübingenn
Herforth, R. S. Lincoln, Nebraska
Herman, H. T. Davis, California
Hernández, E. Spain, Barcelona
Herreng, F. France, Gif-sur-Yvette
Hess, O. Germany, Freiburg
Hildreth, P. Berkeley, California
Hilk, I. Germany, Tübingen
Hillman, N. W. Philadelphia, Pennsylvania
Hillman, R. Philadelphia, Pennsylvania
Himoe, E. Madison, Wisconsin
Hinton, C. W. Athens, Georgia
Hiraizumi, Y. Japan, Misima
Hirara, H. Japan, Tokyo
Hirosé, Y. Japan, Kobe
Hiroyoshi, T. Japan, Osaka
Hiziguro, S. Japan, Osaka
Hoar, D. Canada, Vancouver
Hochman, B. Knoxville, Tennessee
Hoenigsberg, H. F. Colombia, Bogotà
Hoffman, F. Syracuse, New York
Holland, I. B. England, Leicester
Hollander, W. F. Ames, Iowa
Hollingdale, B. J. Australia, Sydney
Hollingsworth, M. J. England, London
Holm, D. G. Storrs, Connecticut
Holzman, H. E. Chambersburg, Pennsylvania
Hon, P.-Y. Madison, Wisconsin
Horikawa, M. Madison, Wisconsin
Hosgood, S. M. Australia, Melbourne
House, V. L. Columbus, Ohio
Hubby, J. L. Chicago, Illinois
Huck, R. St. Louis, Missouri
Hudson, G. England, Cambridge
Hunt, D. M. England, Sheffield
Hunter, A. S. Venezuala, Barquisimeto
Huot, L. England, Sussex
Huston, L. Pittsburgh, Pennsylvania
Ichida, H. Japan, Tokyo
Ikeda, H. Japan, Tokyo
Imaizumi, Y. Japan, Chiba
Imberski, R. Baltimore, Maryland
Inagaki, E. Bowling Green, Ohio
Iturra, P. Chile, Santiago
Iyama, S. Japan, Misima
Jackson, D. Chapel Hill, North Carolina
Jacobs, M. E. Goshen, Indiana
Jacobson, C. Berkeley, California
Janning, W. Germany, Munich
Javellot, M. P. France, Lyon
Jeavons, H. New Haven, Connecticut
Jeffery, D. Berkeley, California
Jenkins, J. Los Angeles, California
Jensen, R. Tucson, Arizona
Jhin, H. S. Korea, Seoul
Jinks, J. L. England, Birmingham
Johnson, G. R. Laramie, Wyoming
Johnson, M. B. Richmond, Virginia
Johnston, F. Storrs, Connecticut
Jones, J. K. England, Reading
Jones, L. P. Australia, Sydney
Jost, P. Eugene, Oregon
Jozwiak, J. Buffalo, New York
Juner, J. New York, New York
Jungen, H. Switzerland, Zürich
Kahn, F. M. Australia, Brisbane
Kaji, S. Japan, Kobe
Kale, P. G. India, Varanasi
Kalisch, W.-E. Germany, Munich
Kalisz, A. Brazil, Pôrto Alegre
Kan, J. Madison, Wisconsin
Kanehisa, T. Japan, Kobe
Kanellis, A. Greece, Thessaloniki
Kang, S. H. Madison, Wisconsin
Kang, Y. S. Korea, Seoul
Kantor, P. Syracuse, New York
Kaplan, M. Eugene, Oregon
Kaplan, M. New York, New York
Kaplan, W. D. Duarte, California
Karlik, A. Austria, Vienna
Kasnic, G. Philadelphia, Pennsylvania
Kastritsis, C. New York, New York
Kataoka, Y. Japan, Tokyo
Kaufmann, B. P. Ann Arbor, Michigan
Kaul, D. Australia, Melbourne
Kawabe, M. Japan, Kobe
Kawonishi, M. Japan, Misima
Kearsey, M. J. England, Birmingham
Keith, A. Eugene, Oregon
Keller, E.C.,Jr. College Park, Maryland
Keller, H. College Park, Maryland
Kelly, A. K. Syracuse, New York
Kempen, C. Ann Arbor, Michigan
Kenyon, A. Athens, Georgia
Kernaghan, R. P. Stony Brook, New York
Kesavar, P. C. India, New Delhi
Khan, A. H. England, Cambridge
Khan, F. M. Australia, Brisbane
Kidwell, J. F. Providence, Rhode Island
Kieft, P. Netherlands, Leiden
Kikkawa, H. Japan, Osaka
Kim, K. W. Korea, Kwangju
Kim, S. H. Korea, Seoul
Kim, Y. J. Korea, Seoul
Kimura, M. Japan, Misima
Kindred, B. M. Australia, Sydney
King, A. L. Salt Lake City, Utah
King, R. C. Evanston, Illinois
Kircher, H. Tucson, Arizona
Kiriazis, W. C. Hiram, Ohio
Kirschbaum, W. F. Argentina, Buenos Aires
Kitagawa, O. Japan, Tokyo
Kitazume, Y. Japan, Kobe

Klepper, C. Netherlands, Leiden
Klerk, T. H. Netherlands, Leiden
Klingele, M. B. Notre Dame, Indiana
Knight, G. R. Scotland, Edinburgh
Kobayashi, S. Japan, Misima
Kobel, H. R. Netherlands, Leiden
Koch, E. A. Evanston, Illinois
Koch, R. Switzerland, Zürich
Kochanski, Z. New York, New York
Koepfer, R. New York, New York
Kohl, J. C. Ann Arbor, Michigan
Kohne, D. College Park, Maryland
Kojima, K. Raleigh, North Carolina
Komma, D. J. New York, New York
Kondo, K. Japan, Anzyo
Kono, S. Japan, Osaka
Kooistra, J. Netherlands, Haren
Koref-Santibañez, S. Chile, Santiago
Korge, G. Germany, Munich
Korinek, E. Canada, Vancouver
Kosuda, K. Japan, Tokyo
Kranenburg, E. J. Netherlands, Haren
Krause, C. Storrs, Connecticut
Krauss, M. I. Storrs, Connecticut
Kret, M. Netherlands, Leiden
Krimbas, C. B. Greece, Athens
Kroeger, H. Switzerland, Zürich
Kroman, R. Long Beach, California
Kubli, E. Switzerland, Zürich
Kung, H. H. Chicago, Illinois
Kunze-Mühl, E. Austria, Vienna
Kuroda, Y. Japan, Osaka
Kurokawa, H. Japan, Tokyo
Kusyk, C. Chapel Hill, North Carolina
Kvelland, F. Norway, Oslo
Lakovaara, S. Finland, Helsinki
Lamb, M. J. England, Harwell
Lamprecht, J. Switzerland, Zürich
Landa, Z. Czechoslavokia, Prague
Lange, E. L. Lafayette, Indiana
Langer, B. Pittsburgh, Pennsylvania
Lansky, L. Philadelphia, Pennsylvania
Latter, B.D.H. Australia, Canberra
Laughnan, J. R. Urbana, Illinois
Lavende, A. Colombia, Bogota
Lawrence, M. J. England, Birmingham
Lawrence, R. DeKalb, Illinois
Lawser, J. Ann Arbor, Michigan
Leães, A. P. Brazil, Pôrto Alegre
Lee, B. W. Korea, Seoul
Lee, C. C. Korea, Seoul
Lee, C. S. Korea, Seoul
Lee, T. J. Korea, Seoul
Lees, R. K. England, Buckinghamshire
LeFever, H. M. Emporia, Kansas
Lefevre, G. Northridge, California
Legay, J.-M. France, Lyon
Leigh, B. Netherlands, Leiden
Leon, W. N. Argentina, Buenos Aires
Lesseps, R. J. Woodstock, Maryland
Levene, H. New York, New York
Leventhal, E. New Haven, Connecticut
Levine, L. New York, New York
Levitan, M. Philadelphia, Pennsylvania
Levy, A. Australia, Adelaide
Lewgoy, F. Brazil, Pôrto Alegre
Lewis, E. B. Pasadena, California
Lewis, H. W. Washington, D. C.
Lewis, K. G. Woodstock, Maryland
Lewontin, R. C. Chicago, Illinois
Lezzi, M. Switzerland, Zürich
L'Heritier, P. France, Gif-sur-Yvette
Lifschytz, E. Israel, Jerusalem
Lindsay, D. W. Rexburg, Idaho
Lindsley, D. L. Oak Ridge, Tennessee
Liner, E. C. College Park, Maryland
Ling, L.-N.L. Madison, Wisconsin
Lloyd, B. Australia, Adelaide
Locatelli, F. Italy, Milan
Lommerse, M.A.H. Netherlands, Leiden
Long, T. Netherlands, Haren
Lopez, C. A. Mexico, Mexico City
Lovell, D. M. England, Buckinghamshire
Lowenstein, L. L. Madison, Wisconsin
Lucchesi, J. C. Chapel Hill, North Carolin
Luce, W. M. Urbana, Illinois
Luchowski, E. Buffalo, New York
Ludwig, M. da R. Brazil, Pôrto Alegre
Ludwig, N. C. Brazil, Pôrto Alegre
Lüers, H. Germany, Berlin
Lüers, T. Germany, Berlin
Lundberg, D. Sweden, Uppsala
Lundin, D. J. Ridgefield, Connecticut
Lüning, K. G. Sweden, Stockholm
Lupo, R. Israel, Jerusalem
Machida, I. Japan, Chiba
Machado, D. M. Brazil, Pôrto Alegre
MacIntyre, R. J. Ithaca, New York
MacKinney, D. Chapel Hill, North Carolina
Maeda, Y. Japan, Kobe
Magnelli, N. Madison, Wisconsin
Mahowald, A. P. Woodstock, Maryland
Mainx, F. Austria, Vienna
Majoral, J. Spain, Barcelona
Malecky, H. Australia, Canberra
Mallmann, M. C. Brazil, Pôrto Alegre
Mampell, K. Davis, California
Mandia, R. M. Lexington, Kentucky
Manna, G. K. India, Kalyani
Marien, D. New York, New York
Marin, L. M. Colombia, Bogotà
Marinic, S.E.de Argentina, Buenos Aires
Marinkovic, D. New York, New York
Markowitz, E. Madison, Wisconsin
Marques, E. K. Brazil, Pôrto Alegre
Maruyama, T. Madison, Wisconsin
Masdevall, J. M. Spain, Barcelona
Massasso, J. England, Buckinghamshire
Massimillo, L. Bowling Green, Ohio
Masuda, H. Japan, Misima
Mather, W. B. Australia, Brisbane
Mats-Ers, C. Sweden, Uppsala
Matsudaira, Y. Bowling Green, Ohio
Maxwell, D. College Park, Maryland
Mayeda, K. Detroit, Michigan
Mayo, M. J. Australia, Adelaide
Mazar-Barnett, B. Argentina, Buenos Aires
McBean, I. Australia, Melbourne

McCampbell, C. M. Knoxville, Tennessee
McCarron, M. J. Philadelphia, Pennsylvania
McCarthy, P. Detroit, Michigan
McClure, S. Ann Arbor, Michigan
McCrady, W. B. Arlington, Texas
McDonald, J. A. Corvallis, Oregon
McDonald, K. H. Pullman, Washington
McFarlane, J. L. Riverside, California
McKinley, A. Australia, Sydney
McNary, H. W. Lafayette, Indiana
McNew, R. W. Lafayette, Indiana
McReynolds, M. Chicago, Illinois
McSheehy, T. W. England, Harwell
Mead, C. G. Oak Ridge, Tennessee
Medina, L. Colombia, Bogotà
Megna, F. Italy, Naples
Mendez, H. Brazil, Pôrto Alegre
Ménsua, J. L. Spain, Barcelona
Merle, J. France, Lyon
Merriam, J. R. Seattle, Washington
Mertens, R. Germany, Münster
Metcalfe, J. England, Newcastle Upon Tyne
Mitchell, A. Pasadena, California
Mitchell, H. K. Pasadena, California
Mettler, L. E. Raleigh, North Carolina
Meyer, G. F. Germany, Tubingen
Meyer, H. U. Madison, Wisconsin
Micheli, A. Italy, Rome
Mickey, G. H. Ridgefield, Connecticut
Milkman, R. Syracuse, New York
Miller, D. D. Lincoln, Nebraska
Miller, L. Lebanon, Beirut
Miramori, S. Japan, Hiroshima
Mindek, G. Switzerland, Zürich
Mittler, J. E. DeKalb, Illinois
Mittler, S. DeKalb, Illinois
Mizushima, T. Japan, Osaka
Moberly, B. J. Ann Arbor, Michigan
Mohler, B. Corvallis, Oregon
Mohler, J. D. Corvallis, Oregon
Mojica, T. Colombia, Bogotà
Monclús, M. Spain, Barcelona
Montalenti, G. Italy, Rome
Montelius, I. Sweden, Stockholm
Moon, H. M. Chapel Hill, North Carolina
Moree, R. Pullman, Washington
Moritz, C. Germany, Munich
Moriwaki, D. Japan, Tokyo
Morton, M. Charlottesville, Virginia
Moskwinski, T. Notre Dame, Indiana
Mossige, J. C. Norway, Oslo
Mourgues, C. France, Lyon
Mukai, Y. Japan, Misima
Mukherjee, A. S. India, Calcutta
Mukherjee, R. Netherlands, Leiden
Mulkay, L. M. Rexburg, Idaho
Muller, H. J. Madison, Wisconsin
Muñoz, E. R. Argentina, Buenos Aires
Murata, I. Japan, Chiba
Murphy, C. Berkeley, California
Murty, B. R. India, New Delhi
Myren, J. Tucson, Arizona
Myszewski, M. E. Eugene, Oregon
Nadal, A. Spain, Barcelona
Nagle, J. Raleigh, North Carolina
Nair, P. S. St. Louis, Missouri
Nakai, S. Japan, Chiba
Nakanishi, H. Japan, Chiba
Nakao, Y. Japan, Chiba
Nam, H. W. Korea, Seoul
Napp, M. Brazil, Pôrto Alegre
Narayana, R. India, Madras
Narda, R. D. India, Punjab
Nardon, R. Brazil, Pôrto Alegre
Narise, S. Japan, Misima
Narise, T. Japan, Misima
Nash, D. J. Fort Collins, Colorado
Nash, W. G. Ann Arbor, Michigan
Navas, Y. G. Colombia, Bogotà
Nawa, S. Japan, Misima
Neeley, J. C. Corvallis, Oregon
Nei, M. Japan, Chiba
Neulat, M. M. France, Lyon
Nicoletti, B. Italy, Rome
Niederkorn, J.A., Jr. St. Louis, Missouri
Nienhaus, A. J. Netherlands, Groningen
Nigon, V. France, Lyon
Nilsson, B. Northridge, California
Nilsson, L. R. Sweden, Uppsala
Nobuki, R. Japan, Osaka
Norton, I. L. Oak Ridge, Tennessee
Nöthel, H. Germany, Berlin
Nöthiger, R. Switzerland, Zürich
Novitski, E. Eugene, Oregon
Nozawa, K. Japan, Anzyo
Obe, G. Germany, Berlin
Oftedal, P. Norway, Oslo
Ohba, S. New York, New York
Ohta, T. Raleigh, North Carolina
Oishi, K. New Haven, Connecticut
O'Neal, L. Newark, New Jersey
Orem, J. Seattle, Washington
Oster, I. I. Bowling Green, Ohio
Oster, P. Bowling Green, Ohio
Owens, S. DeKalb, Illinois
Ogita, Z. Japan, Osaka
Ohanessian-Guillemain, A. France, G-s-Y.
Ohba, S. Japan, Tokyo
Ohlendorff, H. Sweden, Uppsala
Ohnishi, E. Japan, Tokyo
Oishi, K. Japan, Misima
Okada, T. Japan, Tokyo
Oksala, T. A. Finland, Turku
Okuno, T. Japan, Osaka
Olivieri, G. Italy, Rome
Olivieri Mancini, A. Italy, Rome
Olvera, R. O. Mexico, Mexico City
Ondres, M. Czechoslavakia, Prague
Oshima, C. Japan, Misima
Paik, Y. K. Korea, Seoul
Paika, I. India, Punjab
Pakonen, C. Z. Pullman, Washington
Palomino, H. Chile, Santiago
Park, B. S. Korea, Seoul
Park, M. S. Korea, Kwangju
Parkash, O. Austria, Vienna
Parshad, R. India, Punjab
Parker, D. R. Riverside, California

Parrish, J. L. Chapel Hill, North Carolina
Parsons, P. A. Australia, Melbourne
Parzen, S. D. Madison, Wisconsin
Pasternak, M. Duarte, California
Pasztor, L. Eugene, Oregon
Patton, J. Tucson, Arizona
Pavlovsky, O. New York, New York
Paxman, G. J. England, Lancaster
Paz, C. Argentina, Buenos Aires
Peach, F. Woodstock, Maryland
Peacock, W. J. Australia, Canberra
Pearce, S. England, Harwell
Pearson, L. C. Rexburg, Idaho
Pearson, M. England, Sheffield
Pelecanos, M. Greece, Thessaloniki
Pentzou-Daponte, A. Greece, Thessaloniki
Pereira, S. Brazil, Pôrto Alegre
Peres, A. P. Brazil, Pôrto Alegre
Pereyra, E. Argentina, Buenos Aires
Perez, R. Duarte, California
Perez-Chiesa, Y. Chicago, Illinois
Perkins, J. C. Knoxville, Tennessee
Perreault, W. Ann Arbor, Michigan
Perrin, M. Switzerland, Zürich
Perrin-Waldemer, C. France, Clermont-Ferrand
Perry, M. Scotland, Edinburgh
Perschmann, B. Germany, Munich
Persson, K. Sweden, Uppsala
Petermann, U. Switzerland, Zürich
Peters, W. Chicago, Illinois
Peterson, K. Ann Arbor, Michigan
Petri, G. F. Logan, Utah
Peyton, K. Long Beach, California
Philip, U. England, Newcastle Upon Tyne
Phillips, D. Syracuse, New York
Phillips, J. P. Logan, Utah
Piekaar, A. M. Netherlands, Leiden
Pierce, H. Madison, Wisconsin
Pieters, J. Netherlands, Haren
Pijnacker, L. P. Netherlands, Haren
Pipkin, S. B. Baltimore, Maryland
Piternik, L. Canada, Vancouver
Plaine, H. L. Columbus, Ohio
Plaut, W. S. Madison, Wisconsin
Plimpton, A. S. Evanston, Illinois
Ploye, H. France, Lyon
Plus, N. France, Gif-sur-Yvette
Podger, R. N. Australia, Sydney
Popper, J. Eugene, Oregon
Portin, P. Finland, Turku
Possani, L. Brazil, Pôrto Alegre
Potthoff, R. F. Chapel Hill, North Carolina
Poulson, D. F. New Haven, Connecticut
Powers, L. M. Baltimore, Maryland
Prakash, S. St. Louis, Missouri
Pratt, C. W. Corvallis, Oregon
Prevosti, A. Spain, Barcelona
Printz, P. France, Gif-sur-Yvette
Pritchard, R. H. England, Leicester
Proust, J. France, Orsay
Prout, T. Riverside, California
Prudhommeau, C. France, Orsay
Purdom, C. E. England, Harwell
Puro, J. Finland, Turku
Rakha, A. Ames, Iowa
Ramanamurthy, C. V. Notre Dame, Indiana
Ramamurthy, G. Berkeley, California
Ramel, C. Sweden, Stockholm
Ramila, D. Brazil, Pôrto Alegre
Ramirez, L. Mexico, Mexico City
Rapport, E. Ann Arbor, Michigan
Rasmuson, B. Sweden, Uppsala
Rasmuson, M. Sweden, Uppsala
Rathie, K. A. Australia, Sydney
Ratnayake, W. C. Scotland, Edinburgh
Ratty, F. J. San Diego, California
Rau, C. G. St. Louis, Missouri
Ray-Chaudhuri, S. P. India, Varanasi
Rayle, R. Urbana, Illinois
Remensberger, P. Switzerland, Zürich
Remondini, D. Logan, Utah
Rendel, J. M. Australia, Sydney
Rezzonico Raimondi, G. Italy, Milan
Ribó, G. Spain, Barcelona
Ricker, J. Galesburg, Illinois
Rigby, B. Canada, Vancouver
Rim, N. R. Korea, Seoul
Rimbey, M. H. Columbus, Ohio
Rinehart, R. R. San Diego, California
Rizki, R. M. Ann Arbor, Michigan
Rizki, T. M. Ann Arbor, Michigan
Roberts, B. L. Charlottesville, Virginia
Roberts, P. A. Oak Ridge, Tennessee
Robertson, A. Scotland, Edinburgh
Robertson, E. Scotland, Edinburgh
Robertson, F. W. Scotland, Edinburgh
Rocha, F. J. da Brazil, Pôrto Alegre
Rocha, Z. M. A. da Brazil, Pôrto Alegre
Roeder, C. Switzerland, Zürich
Rojas, E. Chile, Santiago
Romero, I. F. Colombia, Bogotà
Ronen, A. Israel, Jerusalem
Rosenfeld, A. Seattle, Washington
Rosin, S. Switzerland, Bern
Rothloff, L. Philadelphia, Pennsylvania
Ruderer-Doschek, E. Austria, Vienna
Rüegg, M. Switzerland, Zürich
Rundell, C. Davis, California
Russell, D. Emporia, Kansas
Russell, J. Tucson, Arizona
Saeki, T. Japan, Chiba
Sakaguchi, B. Japan, Misima
Sakai, K. I. Japan, Misima
Sakai, T. Japan, Anzyo
Sakata, Y. Japan, Hiroshima
Salceda, S. U. Mexico, Mexico City
Salceda, V. M. New York, New York
Salverson, H. Madison, Wisconsin
Salzano, F. M. Brazil, Pôrto Alegre
Sander, K. Germany, Freiburg
Sanders, L. Ames, Iowa
Sanders, N. Emporia, Kansas
Sandler, A. Philadelphia, Pennsylvania
Sandler, L. Seattle, Washington
Sang, J. H. England, Sussex
Sankaranarayanan, K. Netherlands, Leiden
Santell, F. College Park, Maryland
Santos, T. M. M. dos Brazil, Pôrto Alegre

Sanyal, C. India, Calcutta
Sari Gorla, M. Italy, Milan
Sarkar, D. N. India, Varanasi
Sasaki, F. Japan, Tokyo
Savolainen, S. Finland, Turku
Savontaus, M.-L. Finland, Turku
Schalet, A. Storrs, Connecticut
Scharloo, W. Netherlands, Haren
Scheidt, G. East Lansing, Michigan
Scheinberg, E. Raleigh, North Carolina
Schereiks, G. Germany, Berlin
Schertz, G. East Lansing, Michigan
Schewe, M. J. Chapel Hill, North Carolina
Schmid, V. Switzerland, Zürich
Schmitz, T. H. Carbondale, Illinois
Schneider-Minder, A. Chapel Hill, North Carolina
Scholefield, J. Canada, Vancouver
Schouten, S.C.M. Netherlands, Utrecht
Schubiger, G. Switzerland, Zürich
Schwarz, R. Bowling Green, Ohio
Schwinck, I. Storrs, Connecticut
Scowcroft, W. R. Australia, Canberra
Sederoff, R. Los Angeles, California
Seecof, R. Duarte, California
Seegmiller, R. Salt Lake City, Utah
Seeley, B. A. Madison, Wisconsin
Seiger, M. B. Dayton, Ohio
Seki, T. Japan, Osaka
Sena, E. Ithaca, New York
Sessi, J. Pittsburgh, Pennsylvania
Shamay, E. Israel, Jerusalem
Shapiro, P. Medford, Massachusetts
Sharma, G. P. India, Punjab
Sharma, R. P. India, New Delhi
Shaw, B. A. Bowling Green, Ohio
Shaw, R. Detroit, Michigan
Sheldon, B. L. Australia, Sydney
Shelton, E. E. Logan, Utah
Sheppard, P. M. England, Liverpool
Sherer, G. Philadelphia, Pennsylvania
Sheridan, A. K. Australia, Sydney
Shermoen, A. Los Angeles, California
Sherwin, R. M. Pittsburgh, Pennsylvania
Sherwood, E. R. Berkeley, California
Shideler, D. Lafayette, Indiana
Shields, G. England, Sussex
Shillcock, J. Neward, New Jersey
Shinoda, T. Chapel Hill, North Carolina
Shiomi, M. Japan, Hiroshima
Shiomi, T. Japan, Hiroshima
Shoham, Y. Israel, Jerusalem
Short, T. St. Louis, Missouri
Shoup, J. Chicago, Illinois
Sick, K. Denmark, Copenhagen
Silva, L.C.da Brazil, Pôrto Alegre
Silva, M.M.da Brazil, Pôrto Alegre
Silveira, F.M.da Brazil, Pôrto Alegre
Simmons, J. R. Logan, Utah
Simões, C. V. Brazil, Pôrto Alegre
Simões, N. Brazil, Pôrto Alegre
Simpson, B. Bellingham, Washington
Singer, K. Storrs, Connecticut
Singh, A. India, Punjab
Singh, M. Chicago, Illinois
Slatis, H. M. East Lansing, Michigan
Slizynska, H. Scotland, Edinburgh
Slizynski, B. M. Scotland, Edinburgh
Smit, A. Pasadena, California
Smith, C. F. Chapel Hill, North Carolina
Smith, D. Raleigh, North Carolina
Smith, P. A. Evanston, Illinois
Smith, P. D. Chapel Hill, North Carolina
Smith, S. Ann Arbor, Michigan
Smith, J. M. England, Sussex
Soares, B. Brazil, Pôrto Alegre
Sobels, F. H. Netherlands, Leiden
Soliman, A. M. Ann Arbor, Michigan
Sommerau, I. Germany, Berlin
Sondhi, G. Newark, New Jersey
Sondhi, K. C. Newark, New Jersey
Sonnenblick, B. P. Newark, New Jersey
Spassky, B. New York, New York
Sperlich, D. Austria, Vienna
Sperling, K. Germany, Berlin
Spieler, R. A. Chicago, Illinois
Spiess, E. B. Pittsburgh, Pennsylvania
Spiess, L. D. Pittsburgh, Pennsylvania
Spieth, H. T. Davis, California
Spofford, J. B. Chicago, Illinois
Spring, H. Switzerland, Zürich
Springer, R. Austria, Vienna
Spurway, R. Australia, Brisbane
Stadler, J. Fort Collins, Colorado
Stalker, H. D. St. Louis, Missouri
Stalker, S. E. Lexington, Kentucky
Staub, M. Switzerland, Zürich
Stauffer, H. Berkeley, California
Stern, C. Berkeley, California
Stevenson, R. Johnson City, Tennessee
Stewart, M. C. Oak Ridge, Tennessee
Strachan, K. Scotland, Edinburgh
Strangio, V. A. Australia, Melbourne
Strickberger, M. W. St. Louis, Missouri
Strickland, B. C. Knoxville, Tennessee
Strömnoes, O. Norway, Oslo
Struck, E. Germany, Berlin
Sturtevant, A. H. Pasadena, California
Suárez, M. Spain, Barcelona
Sugimoto, K. Japan, Hiroshima
Sugisaki, R. Japan, Misima
Suguna, S. G. India, Madras
Suñe, M. U. Brazil, Pôrto Alegre
Suomalainen, E. Finland, Helsinki
Suzuki, D. Canada, Vancouver
Swaminathan, M. S. India, New Delhi
Swatek, J. Chicago, Illinois
Taira, T. Japan, Misima
Takahashi, J. Canada, Vancouver
Takaya, H. Japan, Kobe
Tanaka, Y. Japan, Tokyo
Tantawy, A. O. Chicago, Illinois
Tartof, K. Ann Arbor, Michigan
Tasca, R. Philadelphia, Pennsylvania
Tates, A. D. Netherlands, Leiden
Tatsukawa, S. Japan, Hiroshima
Teitge, J. Galesburg, Illinois
Temin, R. G. Madison, Wisconsin
Teninges, D. France, Orsay

Thedy, O. P. Brazil, Pôrto Alegre
Thoday, J. M. England, Cambridge
Thompson, C.F.,Jr. Pittsburgh, Pennsylvania
Thompson, P. E. Ames, Iowa
Thompson, S. R. Corvallis, Oregon
Thomson, J. A. Australia, Melbourne
Thorsen, E. A. Evanston, Illinois
Throckmorton, L. H. Chicago, Illinois
Tüvola, A. Finland, Helsinki
Tillinghast, B. Storrs, Connecticut
Tjoa, F.H.B. Netherlands, Leiden
Tobari, I. Japan, Chiba
Tobari, Y. Japan, Tokyo
Tobler, H. Switzerland, Zürich
Tokuda, T. Japan, Misima
Tokunaga, C. Berkeley, California
Tomita, T. Japan, Anzyo
Tondo, C. V. Brazil, Pôrto Alegre
Tonzetich, J. Durham, North Carolina
Towne, J. Emporia, Kansas
Townsend, J. I. Richmond, Virginia
Traut, A. Germany, Münster
Traut, H. Germany, Münster
Trosko, J. E. Oak Ridge, Tennessee
Trout, W. E., III Oak Ridge, Tennessee
Tsacas, S. Greece, Athens
Tsukamoto, M. Japan, Osaka
Tuinstra, E. J. Netherlands, Utrecht
Turoczi, L. Newark, New Jersey
U, R. DeKalb, Illinois
Ulrich, H. Switzerland, Zürich
Ulrich, V. Pittsburgh, Pennsylvania
Ulrichs, P. C. Berkeley, California
Ursprung, H. Baltimore, Maryland
Valencia, R. M. Argentina, Buenos Aires
Van Delden, W. Netherlands, Haren
Vander Mey, K. Bellingham, Washington
Vandling, W. H. Eugene, Oregon
Vann, E. Evanston, Illinois
Van Rhijn, J. G. Netherlands, Haren
Van Valen, L. New York, New York
Vasistha, H. C. India, Varanasi
Vaux, P. College Park, Maryland
Velga-Neto, A. J. Brazil, Pôrto Alegre
Vigier, P. France, Orsay
Vlist, J. van der Netherlands, Utrecht
Voelker, R. Lincoln, Nebraska
Volkers, W. Netherlands, Leiden
von Borstel, R. C. Oak Ridge, Tennessee
Von Halle, E. S. Oak Ridge, Tennessee
Urye, C.E.de England, Buckinghamshire
Waddington, C. H. Scotland, Edinburgh
Wahrman, J. Israel, Jerusalem
Waldner, R. Switzerland, Zürich
Walker, S. England, Liverpool
Wallace, B. Ithaca, New York
Wallbrunn, H. M. Gainesville, Florida
Wallis, B. Madison, Wisconsin
Walsh, M. M. DeKalb, Illinois
Ward, C. L. Durham, North Carolina
Wassenaar, M. C. Netherlands, Leiden
Wasserman, M. New York, New York
Watanabe, I. Japan, Chiba
Watanabe, T. Japan, Kobe
Watanabe, T. Japan, Misima
Watanabe, T. K. Japan, Misima
Watson, W.A.F. Netherlands, Leiden
Webb, H. Salt Lake City, Utah
Weber, U. Pasadena, California.
Wehman, H. J. Baltimore, Maryland
Wei, I. St. Louis, Missouri
Weisbrot, D. R. Medford, Massachusetts
Welshons, W. J. Ames, Iowa
Wendell, S. Stony Brook, New York
Westphal, N. J. Lincoln, Nebraska
Wettstein, D. von Denmark, Copenhagen
White, G. J. Ann Arbor, Michigan
Whitmire, S. D. Chicago, Illinois
Whitten, M. J. Australia, Canberra
Whittinghill, M. Chapel Hill, North Carol
Whittle, J.R.S. England, Cambridge
Widmayer, D. Wellesley, Massachusetts
Wieczorek-Bochnig, U. Germany, Berlin
Wiesner, W. Syracuse, New York
Wildermuth, H. Switzerland, Zürich
Wilkerson, R. D. Oak Ridge, Tennessee
Wilkins, M. S. Lexington, Kentucky
Wilkinson, P. England, Birmingham
William, D. L. Philadelphia, Pennsylvania
Williams, E. Lebanon, Beirut
Williams, J. Raleigh, North Carolina
Williams, S. Ithaca, New York
Williamson, J. H. Athens, Georgia
Willott, G. M. England, Sheffield
Willson, A. Raleigh, North Carolina
Wilson, F. England, Buckinghamshire
Wilson, J. Riverside, California
Wilson, L. P. Wellesley, Massachusetts
Wind, H. Germany, Münster
Wing, M. Raleigh, North Carolina
Winters, V. Syracuse, New York
Wittenberg, T. Eugene, Oregon
Woldring-Steensma, K. T. Netherlands, Gr.
Wolf, E. Germany, Berlin
Wolff, M. L. Washington, D.C.
Wolstenholme, D. R. Germany, Tübingen
Wong, P.T.C. Corvallis, Oregon
Wood, B. Ithaca, New York
Workman, P. L. England, Cambridge
Wright, T.R.F. Charlottesville, Virginia
Wu, D. Detroit, Michigan
Wu, S. Pullman, Washington
Wui, I. S. Korea, Kwangju
Würgler, F. E. Switzerland, Zürich
Wylie, A. P. New Zealand, Duredin
Xavier, J. Brazil, Pôrto Alegre
Yamaguchi, E. Japan, Chiba
Yamaguchi, H. Japan, Misima
Yamazaki, T. Ames, Iowa
Yanders, A. F. East Lansing, Michigan
Yarbrough, K. Raleigh, North Carolina
Yoon, S. B. Madison, Wisconsin
Yoshikawa, I. Japan, Chiba
Young, S.S.Y. Australia, Sydney
Young, W. J. Baltimore, Maryland
Ytterborn, K. Sweden, Stockholm
Yuki, S. Japan, Kobe
Zambruni, L. Italy, Milan
Zouros, E. Greece, Athens