# DROSOPHILA

## Information Service

# 47

## July 1971

Material Contributed by
DROSOPHILA WORKERS

and arranged by
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# DROSOPHILA INFORMATION SERVICE

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RESEARCH NOTES

TECHNICAL NOTES



TEACHING NOTE

## PASADENA, CALIFORNIA: CALIFORNIA INSTITUTE OF TECHNOLOGY
### Division of Biology

Note: The symbol, *, is used for cross indexing and signifies that the mutant is carried in a stock whose number is shown at the right.

### Wild Stocks

1 Canton-S
* Florida . . . . . 830
2 Hikone A-S (strong amylase of Kikkawa)
3 Hikone A-W (weak anylase of Kikkawa)
4 Lausanne-S
5 Oregon-R-C
6 Swedish-c
7 Urbana-S

### Chromosome 1

* $ac_3$ . . . . . 174
8 ac w$^a$
9 amx/FM3, y$^{31d}$ sc$^8$ dm B 1
10 amx $lz^g$ v/y f:=
* amx . . . . . (see $lz^K$)
11 Ax
* $bb_{G3}$ . . . . . 26, 63, etc.
* $bb_1$ . . . . . 133
* $bb^N$ . . . . . 738, 780
* $bb^{poi}$ . . . . . 167
* $bb^{poi}$ . . . . . (see $bb^{G3}$)
12 B
13 $B_3Bx^r$ car/y f:=
* $B_i$ . . . . . 42
* $B^iB$ . . . . . 45
* $BB_{36b}$ . . . . . 43
* $BB^{36b}$ . . . . . 44
14 Bg B/In(1)AM
15 bi $ct^6$ $g^2$
16 bo v
17 br
18 $br_3w^e$ ec rb $t^4$/FM1, y$^{31d}$ sc$^8$ w$^a$ $lz^s$ B
* $br^3$ . . . . . 664
19 $Bx_2$
20 $Bx_3$
21 $Bx_J$
22 $Bx^r$
* $Bx_{r49k}$ . . . . . 13
* $Bx^{144}$ . . . . . 144
23 car
* $cho_2$ . . . . . 101
* $cho^2$ . . . . . 187
24 cm
25 cm $ct^6$
* $Co_{53}$ . . . . . 184
26 $cs_6$/y w bb
* $ct^6$ . . . . . 15, 25, etc.
* $ct^K$ . . . . . 175
27 $ct^n$ oc/FM1, y$^{31d}$ sc$^8$ w$^a$ $lz^s$ B
* cu-X . . . . . 787
* cv . . . . . 102, 103, etc.
28 $cx_{tg}$
29 $cx^{tg}$ oc/FM1, y$^{31d}$ sc$^8$ w$^a$ $lz^s$ B
30 dm/y f:=
31 dor/y f:=
32 $dor^1$/FM6, y$^{31d}$ sc$^8$ dm B (nub/+); see also 764
33 dow/FM6, y$^{31d}$ sc$^8$ dm B
* $dx_{st}$ . . . . . 38, 125
* $dx^{st}$ . . . . . 664, 665
34 dy
* e(bx)$_2$ . . . (= $en_2$-bx) . . 788
* e(bx)$^2$ . . (= $en^2$-bx) . . 678
* e(S)$^x$ . . . (= $en^x$-S) . . 666
35 Eag
36 ec
37 ec $ct^6$ s car/FM6, y$^{31d}$ sc$^8$ dm B
38 ec dx
* eq . . . . . 102, 737
39 Ext/FM6, y$^{31d}$ sc$^8$ dm B
40 f
41 f B/y f:=
42 f $B^3$/y f:=
43 f BB/y f:=
44 f $BB^{36b}$/y f:=
45 f $B^iB^i$/y f:=
46 f $_3$fu/y f:=
* $f_3$ . . . . . 167
* $f_{36a}$ . . . . . 157
47 $f_{B15}$
* $f_{B27}$ . . . . . 774
* f . . . . . 775
48 $fa_n$
* $fa^n$ . . . . . 782
* flp . . . . . (see flw)
49 flw
50 fo

51 fx/y f:=
* fu . . . . . 46
* $fw^{34e}$ . . . . . 146, 170
* $fw^{2}$ . . . . . 194
52 $g_2$
53 $g_2$ pl/FM3, $y^{31d}$ $sc^{8}$ dm B l
54 $g_4$ ty/y f:=
* g . . . . . 667,733
55 $gg_3$/FM6, $y^{31d}$ $sc^{8}$ dm B
56 gg
57 $gt_2$ $w^{a}$
58 Hk
* Hw . . . . . 114, 733, etc.
59 $Hw_3$/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B
60 if
61 kz
* l(1)7 . . . . . (see $dor^{1}$)
62 l(1)J1 $sc^{J1}$/l(1)J1 $sc^{J1}$/Dp(1;f)24
63 lh B car bb/y f:=
64 lz/FM3, $y^{31d}$ $sc^{8}$ dm B l
65 $lz_3$/y f:=
66 $lz_{34}$/y f:=
67 $lz_{36}$/y f:=
68 $lz_{37}$/y f:=
69 $lz^{48f}$
$lz^{50e}$/y f:=
* $lz^{BS}$ . . . . . 766
70 $lz^{D}$ $lz^{46}$ $lz^{g}$ $ras^{4}$ v/y f:=
* $lz^{g}$ . . . . . 667
* $lz^{K}$ . . . . . 10, 180, etc.
71 $lz^{s}$
* $lz_{y4}$ . . . . . 18, 27, etc.
* lz . . . . . 120, 766
72 $m_2$
* $m_D$ . . . . . 667,733
73 m/FM3, $y^{31d}$ $sc^{8}$ dm B l
74 M(l)n/FM6, $y^{31d}$ $sc^{8}$ dm B
75 M(l)o f/FM6, $y^{31d}$ $sc^{8}$ dm B
76 $M(l)o^{Sp}$/FM6, $y^{31d}$ $sc^{8}$ dm B
* M(l)Sp . . . . . (see $M(l)o^{Sp}$)
77 mal/y f:=
* $mal^{bz}$ . . . . . 183
78 na/y f:=
79 ny f/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B (ri)
80 oc ptg/$In(1)ClB^{3}$
* od . . . . . (see $os^{o}$)
81 $os^{o}$
82 $os^{s}$
83 pa $sn^{3}$/FM6, $y^{31d}$ $sc^{8}$ dm B
84 peb v
* pl . . . . . 53
* pn . . . . . 109, 176, etc.
85 $pn^{2}$
* $pn_3$ . . . . . 105
* $ptg_2$ . . . . . 679
86 $ptg_3$
* $ptg_4$ . . . . . 80
* ptg . . . . . 141
87 r/y f:=
88 $r^{39k}$ f B/In(1)AM
89 $ras_2$ dy
90 $ras_3$
91 $ras_4$ m
* $ras^{v}$ . . . . . 70
* ras . . . . . 845
92 rb
93 rb cx
94 $rg_2$
95 rst/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B
96 rux/FM6, $y^{31d}$ $sc^{8}$ dm B
97 $rux^{2}$
98 s
99 sbr
100 sc
101 sc cho t
102 sc cv v eq (sc reverted)
103 sc cv v f
104 sc $ec^{6}$ $cv_2$ $ct^{6}$ $v_2$ $g^{2}$ f/FM3, $y^{31d}$ $sc^{8}$ dm B l
105 sc $pn^{3}$ $g^{2rv}$ $Bx^{2}$ ... ($g^{2}$ rev., sc rev.)
105b sc $w^{b1}$ ec cv
106 sc z ec $ct^{6}$
107 sc z $swb^{62b}$ (Ives)
108 $sc_2$ z $w^{17G2}$ ec $ct^{6}$
109 $sc_{3B}$ pn/y f:=
110 $sc^{3-1}$
111 $sc_4$ w/y f:=
* $sc_5$ . . . . . 794
112 $sc_6$
113 $sc_7$ $w^{a}$
* $sc_8$ . . . . . 722, 796, etc.
* $sc_9$ . . . . . 784, 801, etc.
* $sc_{10}$ . . . . . 805
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114 $sc^{19}$/y Hw
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* $sc_{260-15}$ . . . . . 806
* $sc_{260-22}$ . . . . . 847
* sc . . . . . 807

* $sc^{z}$ . . . . . 846
115 $scp_{5}$ $t^{58d14}$/y f:=
116 $sd^{2}$
117 $Sh^{2}$
118 shf
* $sl_{2}$ . . . . . (in C1B, $C1B^{36d}$)
* $sl^{2}$ . . . . . 723
* $sn_{2}$ . . . . . 713
* $sn_{3}$ . . . . . 161
119 $sn^{3}$
120 $sn_{4}^{3}$ $lz^{y4}$ v/y f:=
121 $sn^{34e}$
122 $sn^{36a}$/y f:=
123 * sp-w . . . . . (see $w^{sp}$)
124 spl
* sta . . . . . (see T(1;3)sta)
* su( . . . . . 677
125 su(dx) dx
* $su(f)_{2}$ . . . (= $su^{W}$-f) . . 145
126 $su(s)^{2}_{2}$ $v_{a}$(bw)
127 $su(s)^{2}_{2}$ $w^{a}$ cv t
128 $su(s)^{3}_{S}$ cv v f/y f:=
129 $su(s)^{S}$ v/FMA3, $y^{a}$ (bw)
* $su(w^{a})$ . . (= su-$w^{a}$) . . 700, 708, etc.
* $su^{S2}$-v-pr . . . . . (see $su(s)^{S}$)
130 svr
131 svr $w^{a}$
132 $svr^{poi}$
133 $svr^{poi-dish}$ $bb^{G3}$
134 sw
135 sx $vb^{2}$ $os^{s}$/FM6, $y^{31d}$ $sc^{8}$ dm B
* sy . . . . . (see $os^{s}$)
136 $t_{2}$
137 $t^{3}$ v f
138 $t_{4}$
* $t_{5}$ . . . . . 18
139 $t^{12}$ v r
* tuh-l . . . $^{31d}$(= tu-h) . . 673
140 tw/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B
* ty . . . . . 54
* tyl . . . (= ty-l) . . . 779, 780
141 $un_{2}$ $Bx^{4}$/In(1)AM, $ptg^{4}$
142 $un^{4}$
143 v
144 v f $Bx^{r49k}$ car/y f:=
145 $v_{2}$ f su(f)
146 $v^{2}$ fw
147 $v^{36f}$
* $v^{0f}$ . . . . . 781
148 vb
* $vb^{2}$ . . . . . 135
149 vs
150 w
151 w m f
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* $w^{a}$ . . . . . 108
153 $w^{a}$
154 $w_{a2}$
155 $w_{a3}$
156 $w_{a4}$
157 $w_{bf}$ $f^{5}$
158 $w_{bf2}$ $f$
$w_{bf3}$
* $w_{Bwx}$ . . . . . 697
159 $w_{ch}$
160 $w_{co}$ $wy_{2}$
161 $w_{col}$ $sn^{2}$
162 $w^{e}$
163 $w_{e2}$
164 $w_{ec3}$
165 $w_{h}$
166 $w_{i}$ $f^{3}$ $bb^{N}$
167 $w_{sat}$
168 $w^{sp}$
169 $w^{t}$
170 $w_{u}$ fw
171 w
172 $wy_{2}$
* $wy^{2}$ . . . . . 192
173 y
174 y $ac_{K}$ v
175 y $ct^{K}$ (bw)
176 y pn
177 y pn w cm $ct^{6}$ $sn^{3}$ $oc^{3}$ $ras^{2}$ v $dy^{2}$ $g^{2}$ f $os^{o}$
car sw/FM7b, $y^{31d}$ $w^{a}$ $lz^{s}$ B
178 y pn w cm $ct^{6}$ $sn^{3}$ $oc^{SI}$ $ras^{2}$ v $dy^{2}$ $g^{2}$ f $os^{o}$
car sw/In(1)$sc^{8}$, In(1)dl-49, y v B
179 y sc
180 y $sc_{5}$ $lz^{g}$ v f/y f:=
181 y $sc_{D2}$
182 y $sc^{bz}$
183 y v f $mal^{bz}$
184 y w Co/y f:=
185 $y_{2}$ w spl
186 $y^{2}_{2}$
187 $y^{2}_{2}$ $cho^{2}$
188 $y^{2}_{2}$ cv v f
189 $y^{2}_{2}$ sc $w^{a}$ ec
190 $y^{2}_{2}$ $w^{a}$
191 $y^{2}_{2}$ $w^{a}$ $w^{2}$ $g^{2}$
192 $y^{2}$ $wy^{2}$ $g^{2}$ ($g^{2}$ partly reverted)

193 $y^{2S}$
194 $y_{2S}\ fw^{34e}$
195 $y_{3d}\ y_{3P}/y$ f:=
* $y_4$ . . . . . 812
* $y_{31d}$ . . . . . 814
* $y_{34c}$ . . . . . 9, 18, etc.
196 $y_{59b}$
* $y_{td}$ . . . . . 709
197 $y_{v2}$
198 $y$
199 z $w^{11E4}$

Chromosome 2

200 a px or
201 a px sp
202 $ab_2$
203 $ab_2$/T(Y;2)E
204 $ab_2$ $ix^2$ bw $sp^2$/In(2L+2R)Cy, Cy $dp^{1vI}$ Bl $L^4$ $sp^2$
* abb . . . . . 403
205 abr/In(2L+2R)Cy, Cy $hk_2$
206 abr/SM5, $al^2$ Cy $lt^v$ $sp^2$
207 ad
208 al
209 al b c $sp^2$
210 al dp b bw l(2)ax/SM5, $al^2$ Cy $lt^v$ $sp^2$
211 al dp b pr $ap^{b1t}$ bw/SM5, $al^2$ Cy $lt^v$ $sp^2$
212 al dp b pr Bl c px sp/SM1, $al^2$ Cy $sp_2$
213 al dp b pr Bl c px sp/In(2LR)O, $dp^{1vI}$ Cy pr $cn^2$
214 al dp b pr c px sp
215 al dp b pr Hx
216 $al_2$S ast ho/SM1, $al^2$ Cy $sp^2$
* $al^2$ . . . . . 210, 211, etc.
* alpha-l . . . . . (see tyr-l)
217 Alu
218 an/SM5, $al^2$ $_2$Cy $lt^v$ $_2sp^2$
219 $an^2$/SM1, $al^2$ Cy $sp^2$
220 ang
221 ant(ro)
222 $ap^4$/SM5, $al^2$ Cy $lt^v$ $sp^2$
223 $ap^{b1t}$
224 arch ch1/SM5, $al^2$ Cy $lt^v$ $sp^2$
225 $ast_3$ho cl
* $ast_4$ . . . . . 815
226 $ast^X$ dp cl
* ast . . . . . 300
227 Ata . . . . . 868
228 b
229 b tyr-l
230 b cn beta
231 b el $rd^s$ pr cn
232 b Go/In(2LR)Gla
233 b Go/SM5, $al^2$ Cy $lt^v$ $sp^2$
234 b gp
235 b j
236 b l(2)Bld pr c px sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
237 b lt $wx^{wxt}$ bw
238 b pr tk/T(Y;2)G
239 b sf
240 b vg
* ba . . . . . 251
241 Bl/In(2L+2R)Cy, Cy $bw^{45a}$ $sp^2$ or $^{45a}$
242 Bl/T(2;3)dp
243 Bl $L^2$/SM5, $al^2$ Cy $lt^v$ $sp^2$
244 Bl $stw^{48}$ $ap^{b1t}$ tuf sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
245 Bla/SM5, $al^2$ Cy $lt^v$ $sp^2$
246 blo
* blt . . . . . (see $ap^{b1t}$)
247 bri
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248 $bs^3$
* bs . . . . . 328
249 bur fs(2)El/SM5, $al^2$ Cy $lt^v$ $sp^2$
250 bw
251 bw ba
252 $bw_{2b}$ tu
253 $bw_4$
* $bw_{45a}$ . . . . . 686
* $bw_D$ . . . . . 241
254 $bw_{V1}$
* $bw_{V32g}$ . . . . . 328, 353, etc.
* $bw_{V34k}$ . . . . . 352, 739
* bw . . . . . 342
255 c
256 c wt px
257 cg c/SM5, $al^2$ Cy $lt^v$ $sp^2$
258 cg c/In(2LR)U
259 ch
260 ch1
261 ch1 en/SM5, $al^2$ $_{2b}$Cy $_2lt^v$ $sp^2$
262 ch1 l(2)bw $bw^{2b}$ $mr^2$/SM5, $al^2$ Cy $lt^v$ $sp^2$
263 chy
264 ck/SM5, $al^2$ Cy $lt^v$ $sp^2$
265 $cl_2$
266 $cl^2$/T(Y;2)E
267 $cn_2$
* $cn^2$ (in all stocks containing In(2R)Cy)
268 cn bw
269 cn en/SM5, $al^2$ Cy $lt^v$ $sp^2$

270 $cn_3$ l(@)crc/SM5, $al^2$ Cy $lt^v$ $sp^2$
271 $cn_3$/T(Y;2)C
272 $cn^{35k}$
* cq . . . . . (see $rk^4$)
273 cru/In(2L+2R)Cy, Cy ($w^e$)
274 Cy Bl $bw^D$/SM1, $al^2$ $sp^2$ (no Cy)
275 d/SM5, $al^2$ Cy $lt^v$ $sp^2$
276 d b/SM5, $al^2$ Cy $lt^v$ $sp^2$
277 da/SM1, $al^2$ Cy $sp^2$
277b da pr cn/SM5, $al^2$ Cy $lt^v$ $sp^2$
278 $dil^2$ hv bw sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
279 dke c
280 dp
281 $dp_2$ cn bw
* $dp^D$ . . . . . (see $dp^{lv2}$)
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* $dp^o$ . . . . . (see $dp^{ovN}$)
283 $dp_{o2}$
284 $dp^{olvR}$
285 $dp^{ovN}$/SM5, $al^2$ Cy $lt^v$ $sp^2$
286 $dp^{Rf}$
* $dp^{Th}$ . . . . . (see $dp^{olvR}$)
* $dp_{tx}$ . . . . . (see $dp^{lvI}$)
* $dp^v$ . . . . . (see $dp^{lv}$)
* $dp_{v2}$ . . . . . 690
287 $dp^{vl}$
* $dp^{vM}$ . . . . . (see $dp^{vM}$)
288 $dp^{vM}$/SM5, $al^2$ Cy $lt^v$ $sp^2$
289 $ds_{dp}$
290 $ds^{rv}$ ft $dp^{v2}$ l(2)M b pr/SM5, $al^2$ Cy $lt^v$ $sp^2$
291 ds $S_2$ G b pr/In(2L+2R)Cy, $al^2$ Cy $lt^3$ $L^4$ $sp^2$
292 $ds^{w^{sp}}_{33k}$/In(2L)Cy $t^{L^R}$, Su(S) $dp^{lv2}$ pr
* $ds^{33k}$ . . . . . 328, 353, etc.
293 $ds^{38k}$/In(2L)Cy, Cy $dp^{lv2}$ b pr
294 dsr
295 dw-24F cl/SM5, $al^2$ Cy $lt^v$ $sp^2$
296 dw-24F l(2)cg, cg/SM5, $al^2$ Cy $lt^v$ $sp^2$
* E(S) . . . (= EN-S) . . . 335, 395, etc.
297 ed Su(dx)$^2$
298 el
* en . . . . . 261, 269, 748
* esc . . . . . 816
299 ex
300 ex ds $S^X$ $ast^X$/SM1, $al^2$ Cy $sp^2$
* fes . . . . . (see fs(2)B)
301 fj l(2)Su(H)/SM5, $al^2$ Cy $lt^v$ $sp^2$
302 fj wt/SM5, $al^2$ Cy $lt^v$ $sp^2$
303 $fr^{lv2}$/In(2L+2R)Cy, Cy $dp^{lv2}_v$
304 $fr^2$ wt/SM5, $al^2$ Cy $lt^v$ $sp^2$
305 Frd/In(2L+2R)Cy, Cy $sp^2$
* fs 2.1 . . . . . (see fs(2)El)
306 fs(2)B Alu lt/SM5, $al^2$ Cy $lt^v$ $sp^2$
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* $hk^2$ . . . . . 205
311 ho
312 hv/SM5, $al^2$ Cy $lt^v$ $sp^2$
313 Hx/ see also 215
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315 hy a px sp/SM1, $al^2$ Cy $sp^2$
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318 $J^{34e}$
319 kn
320 $L_2$
321 $L^2$
322 $L^4$
323 $L^5$
324 $L^G$
325 $L^r$
326 $L^{si}$
* l(2)301 . . . . . 367
327 l(2)39 $a_3$ px slt sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
328 l(2)a $bs^3$, In(2L)t/$bw^{vl}$, $ds^{33k}$
* l(2)ax . . . . . 210
329 l(2)ay b c $sp^2$/SM5, $al^2$ Cy $lt^v$ $sp^2$
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* l(2)bw . . . . . 262
* l(2)C . . . . . 399
* l(2)cg . . . . . 296
* l(2)crc . . . . . 270
329b l(2)gd a px or/In(2LR)O, Cy $dp^{lvI}$ pr $cn^2$
330 l(2)gl a px or/SM5, $al^2$ Cy $lt^v$ $sp^2$
331 l(2)H $L^2$/SM5, $al^2$ Cy $lt^v$ $sp^2$
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336 lt/T(Y;2)A
337 lt std/SM2, $al^2$ Cy $lt^v$ $sp^2$
338 $lt_3$ stw
* $lt^v$ . . . . . 291, 864, 888
* lt . . . . . 206, 210, etc.
339 ltd
340 lw
* lys . . . . . 691
341 M(2)173/SM5, $al^2$ Cy $lt^v$ $sp^2$
* $M(2)B_S$ . . . . . (see $M(2)z^{B}_{V34}$)
342 $M(2)e_{S5}$/In(2L+2R)Cy, Cy, $In(2R)bw^v_2$
343 $M(2)H_2$/SM5, $al^2$ Cy $lt^v$ $sp^2$
344 $M(2)1_{S6}$/SM1, $al^2$ Cy $sp^2$
345 $M(2)m_3$/SM5, $al^2$ Cy $lt^v$ $sp^2$
346 $M(2)S2_9$/SM2, $al^2$ Cy $lt^v$ $sp^2$
347 $M(2)S2_9$/SM5, $al^2$ Cy $lt^v$ $sp^2$
* M(2)S3 . . . . . (see $M(2)S2_3$)
* M(2)S5 . . . . . (see $M(2)H_{S5}$)
* M(2)S6 . . . . . (see $M(2)m_{S6}$)
348 M(2)S7/SM5, $al^2$ Cy $lt^v$ $sp^2$
* M(2)S9 . . . . . (see $M(2)S2_9$)
* M(2)S11 . . . . . (see $M(2)e_S$)
349 M(2)z/SM5, $al^2$ Cy $lt^v$ $sp^2$
350 $M(2)z_{Sk}$ b/In(2L)Cy, Cy $dp^{lv2}$ b pr
351 $M(2)z^B$/SM5, $al^2$ Cy $lt^v$ $sp^2$
* Mal . . . . . 694
352 $mi/bw^{V32g}$
353 $mr_2$ $bs^2/bw^{V1}$, $ds^{33k}$
354 $mr^2$/In(2R)Cy, $cn^2$ Bld
355 msf/SM5, $al^2$ Cy $lt^v$ $sp^2$
* N-2G . . . (= N-2) . . . 413
356 net
357 net al ex ds S ast shv ho rub/SM1, $al^2$ Cy $sp^2$
358 net ed $Su(dx)^2$
359 $nub_2$ b pr
360 $nub^2$
361 $nw^2$/In(2L)Cy. In(2R)NS
* $or_{45a}$ . . . . . 200, 330
* or . . . . . 241
362 pd
363 pd $ll_2$
364 pd $ll^2$ sp
365 Pfd/SM5, $al^2$ Cy $lt^v$ $sp^2$
366 pi/SM5, $al^2$ Cy $lt^v$ $sp^2$
367 pi l(2)301/SM5, $al^2$ Cy $lt^v$ $sp^2$
* Pin . . . . . 415
368 pk cn
369 pk tuf ($sp^2$/+)
* $Pm_2$ . . . . . (see $bw^{V1}$)
* $Pm^2$ . . . . . (see $bw^{V32g}$)
370 $po_2$ vg
371 $po^2$
372 pr
373 pr cn/T(Y;2)C
374 $pr_{bw}$ cn ix/SM5, $al^2$ Cy $lt^v$ $sp^2$
375 pr
376 $pu_{Gr}$
* Pu . . . . . 881
377 puf
378 pw-c/SM5, $al^2$ Cy $lt^v$ $sp^2$
379 px
380 px bs (old Berlin stock of Goldschmidt)
381 px bw sp/T(Y;2)J
382 px bw mr $sp/bw^{V1}$, $ds^{33k}$
383 px slt sp
384 pym/In(2L+2R)Cy, Cy
385 pys
386 Q
* rc . . . . . 691
387 rd/SM5, $al^2$ Cy $lt^v$ $sp^2$
* $rd^s$ . . . . . 231
388 $rdo_2$
389 $rdo^2$ pr
* $Rev_B$ . . . . . 823
* $Rev^B$ . . . . . 753
390 $rh_4$
391 $rk^4$
392 rl
* rn . . . . . 882
* Roi . . . . . 441
393 rub
394 $Ruf/bw^{V1}$, $ds^{33k}$
* Rvd . . . . . (see $Rev^B$)
395 S/In(2L+2R)Cy, Cy E(S) (K-pn)
396 $S_2$ $Sp^2$ $ab^2$ ltd/SM5, $al^2$ Cy $lt^v$ $sp^2$
* $S^2_R$ . . . . . 335, 771
397 $S_X/bw^{V1}$, $ds^{33k}$
* $S^X$ . . . . . 300
398 sca
399 sca l(2)C/SM5, $al^2$ Cy $lt^v$ $sp^2$
400 SD-5/SM1, $al^2$ Cy $sp^2$
401 SD-72/SM5, $al^2$ Cy $lt^v$ $sp^2$
* $sf_2$ . . . . . 239
402 $sf^2$
403 shr $bw^{2b}$ abb sp/SM5, $al^2$ Cy $lt^v$ $sp^2$
404 shv
405 shv ho

* Sk . . . . . 350
* slt . . . . . 327, 383
406 sm px/SM5, al$^2$ Cy lt$^v$ sp$^2$
407 sm px pd/SM5, al$^2$ Cy lt$^v$ sp$^2$
408 so$_2$
409 so$^2$ b cn
* sp$_2$ . . . . . 201, 212, etc.
410 sp$^2$ bs$^2$
411 Sp/In(2L)t$_1$ l(2)R$^v$
412 Sp/SM5, al$^2$ Cy lt$^v$ sp$^2$
413 Sp Bl N-2G/SM5, al$^2$ Cy lt$^v$ sp$^2$
414 Sp J/SM5, al$^2$ Cy lt$^v$ sp$^2$
415 Sp J L$^2$ Pin/SM5, al$^2$ Cy lt$^v$ sp$^2$
416 spd gt-4/SM5, al$^2$ Cy lt$^v$ sp$^2$
417 sple
418 spt
419 std/SM5, al$^2$ Cy lt$^v$ sp$^2$
420 stw$_2$
421 stw$^2$
422 stw$_5$/T(Y;2)B
423 stw$^{48}$ ap$^{blt}$ tuf sp$^2$
* Su(dx)$_2$ . . (= Su$_2$-dx) . . 665
* Su(dx)$^2$ . . (= Su$^2$-dx) . . 358,664
* Su(er) . . (= Su-er) . . 693
425 Su(h)/In(2L+2R)Cy, Cy pr
426 Su(H) whd l(2)Su(H)/SM5, al$^2$ Cy lt$^v$ sp$^2$
* Su(S) . . . . . 292
* tet . . . . . 668
427 Tft/SM1, al$^2$ Cy sp$^2$
* Tg . . . . . 818
* tk . . . . . 238
428 tkd/SM5, al$^2$ Cy lt$^v$ sp$^2$
429 tkv
430 tri vg$^{No2}$/SM5, al$^2$ Cy lt$^v$ sp$^2$
* tu . . . . . 252
* tu-36a . . (= tu$^{36a}$) . . 604
431 tuf ltd
432 tyr-1 (p$^p$); see also 229
433 Uf
434 vg
435 vg$_D$ bw
436 vg$_{ni}$/SM5, al$^2$ Cy lt$^v$ sp$^2$
437 vg$_{No2}$
* vg$_{np}$ . . . . . 430
438 vg$_{nw}$
439 vg$_{nw}$ Hia/SM5, al$^2$ Cy lt$^v$ sp$^2$
440 vg$_{nw}$ Hia/T(2;3)S$^M$ In(2L+2R)Cy, Cy
441 vg$_U$/In(2L)t$_1$ Roi, In(2R)Cy, bw sp$^2$ or
442 vst/SM5, al$^2$ Cy lt$^v$ sp$^2$
443 whd
444 wt
* wx$^{wxt}$ . . (= wxt) . . . . 237

Chromosome 3

445 a(3)26
* a-3 . . . . . (see a(3)26)
446 aa h
447 aa tu-36e
448 abd
* Antp$^B$ . . . . . 826
449 app$_{hg}$
450 as$^{hg}$
451 as$^{hg}$ e$^s$
* Ata . . . . . 868
452 bar-3
* Bd . . . . . 566
453 Bd$^G$/In(3R)C, l(3)a
454 bf/TM6, ss$^-$ bx$^{34e}$ Ubx$^{P15}$ e
* bod . . . . . 563
* bp . . . . . (see bul$^{bp}$)
455 bul$^{bp}$
456 bul$^{bp}$/TM1, Me ri sbd$^1$
457 bv
* bx$_3$ . . . . . 594, 608
458 bx$^{34e}$ Cbx Ubx bxd pbx/T(2;3)ap$^{Xa}$
459 bx$^{34e}$
* bxd$_{107}$ . . . . . 458, 595, 873
* bxd$^{107}$ . . . . . 902
* by . . . . . 576, 577
* c(3)G . . (= c3G) . . . 600
460 ca
461 ca bv
462 ca$_2$ K-pn
463 ca$^{nd}$
* ca . . . . . 497
464 Cbx
465 cd
466 cmp ca/TM6, ss$^-$ bx$^{34e}$ Ubx$^{P15}$ e
467 cp
468 cp in ri p$^p$
469 cu
470 cu kar
471 cu kar ry$^8$
472 cur
473 cv-c
474 cv-c sbd$^2$
475 cv-d
* Cyd . . . . . 489
476 D/Gl
477 D$^3$ Sb ca$^2$/In(3L+3R)P

478 det
479 Dfd/In(3LR)Cx
480 $Dfd^{r}$
481 $Dl_{3}$ H $e^{s}$ cd/In(3R)P, spr
482 $Dl_{5}$/In(3R)C, e
483 $Dl_{7}$/In(3R)C, l(3)a
484 $Dl_{9}$/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^{s}$
485 $Dl_{11}$/In(3R)C, e
486 $Dl_{12}$/In(3L+3R)P, Dfd ca
487 $Dl_{13}$/In(3L+3R)P, Dfd ca
488 $Dl_{14}$/In(3R)C, Sb e l(3)e
489 $Dl_{B}$/In(3R)Cyd, Cyd
* $Dl_{x}$ . . . . . 827
490 $Dl_{x}$/In(3L+3R)P
* Dr . . . . . 556
491 $Dr^{Mio}$/TM6, ss $bx^{34e}$ $Ubx^{P15}$ e
492 drb
* dsx . . . . . 551
493 dwh/In(3L+3R)P, Dfd ca
* e . . . . . 681, 682, In(3R)C
* $e^{v}_{4}$(dp) . . . . . 690
494 $e_{11}$ wo ro
495 $e^{s}$
496 $e^{s}$
497 $e^{s}$ $ca^{nd}$/TM6, ss $bx^{34e}$ $Ubx^{P15}$ e
498 eg/In(3LR)Cx
499 $eg^{2}$/In(3LR)Cx
* er . . . . . 684, 693
500 eyg
* fl . . . . . 541
501 fz
502 $gl_{2}$
503 $gl^{2}_{3}$ $e^{4}$
504 $gl^{60j}$
505 $gl^{60j}$
506 Gl Sb/LVM
* gm . . . . . 559, 623
507 gro
508 gs
509 $h_{2}$
510 $h^{2}$
511 H/In(3R)P, hp
512 $H_{2}$Pr/In(3R)C, e
513 $H^{Xa}_{3}$/T(2;3)ap
514 $H_{57c}$/In(3R)C, Sb e l(3)e
* H . . . . . 620
* Hm . . . . . 878
* $Hn_{r}$ . . . . . 879
* $Hn^{r}_{3}$ . . . . . 518
515 $Hn^{r}_{3}$ sr
* Hu . . . . . 828
516 in
517 jv
518 jv $Hn^{r}$ h
519 jvl
* k . . . . . 588
* K-pn . . . . . 395, 462
* $kar_{2}$ . . . . . 470, 471, 614
520 $kar^{2}$
521 Ki
522 l(3)36d10/In(3LR)Cx, D
* l(3)a . . . . . 453, 483
523 l(3)ac $e^{s}$ M(3)w/LVM
* l(3)e . . . . . 488, 514, etc.
* l(3)PL . . . . . (In(3L+3R)P; In(3L+3R)P, Dfd ca)
* l(3)PR . . . . . same as above
524 l(3)tr Sb/In(3L)P, $In(3R)P^{4}_{1}$8, Me Ubx e
525 l(3)tr Ubx/TM1, Me ri sbd
* l(3)W . . . . . 605, 827
* l(3)XaR . . . . . 867
526 ld
527 $Ly/D^{3}$
528 Ly Sb/LVM
* M(3)36e . . . . . (see $M(3)be^{36e}$)
529 M(3)40130/In(3L+3R)P, Dfd ca
* M(3)124 . . . . . (see $M(3)w^{124}$)
* $M(3)B_{2}$ . . . . . (see $M(3)w^{B}$)
* $M(3)B^{2}$ . . . . . (see $M(3)w^{B2}$)
530 $M(3)be^{36e}$/In(3R)C, l(3)a
531 $M(3)h^{S37}$/In(3L)P, Me
532 $M(3)h^{y}$/In(3L)P, Me
533 M(3)S32/T(2;3)Me
534 M(3)S34/T(2;e)Me
535 M(3)S36/T(2;3)Me
* $M(3)h^{S37}$ . . . . . (see $M(3)h^{S37}$)
* M(3)w . . . . . 523
536 M(3)w/In(3R)C, e l(3)e
537 $M(3)w^{124}$/In(3R)C, e l(3)e
538 $M(3)w^{B}$/In(3R)C, e l(3)e
539 $M(3)w^{B2}$/In(3R)C, e l(3)e
* M(3)y . . . . . (see $M(3)h^{y}$)
540 ma
541 ma fl
542 mah
543 $Mc/T(2;3)ap^{Xa}$
544 mwh
545 $N\text{-}X/T(2;3)ap^{Xa}$
546 obt
547 p
548 $p^{p}$
549 $p^{p}$ bx sr $e^{s}$

550 $p^{p}$ cu
551 $p^{p}$ dsx/TM6, ss$^{-}$ bx$^{34e}$ Ubx$^{P15}$ e
552 pb/In(3LR)Cx$^{Xa}$
553 pbx/T(2;3)ap$^{1}$
554 Pc/TM1, Me ri sbd$^{1}$
* Pdr . . . . . 692
555 Pr/In(3R)C, e
556 Pr Dr/TM3, y$^{+}$ ac$^{+}$ ri $p^{p}$ sep bx$^{34e}$ $e^{s}$
557 Pt/T(2;3)ap$^{Xa}$, ca
558 pyd
559 R Ly/In(3L)P, gm
560 ra
561 red
562 ri
563 ri bod $e^{s}$/In(3L)P, Me, In(3R)C, Sb e 1(3)e
564 ri $p^{p}$/T(Y;2;3)F, st
565 ro
566 ro Bd ca/In(3R)C, l(3)a
567 ro ra ca/T(2;3)Me
567b roe $p^{p}$
568 $rs^{2}$
569 rsd
* $rt^{2}$ . . . . . 587
570 ru
571 ru h th st $p^{p}$ H $e^{s}$ ro/TM6, ss$^{-}$ bx$^{34e}$ Ubx$^{P15}$ e
572 ru h th st cu sr $e^{s}$ ca
573 ru h th st cu sr $e^{s}$ ca/TM3, ru Sb Ser
574 ru h th st cu sr $e^{s}$ Pr ca/TM6, ss$^{-}$ bx$^{34e}$ Ubx$^{P15}$ e
575 ru h th st $p^{p}$ cu sr $e^{s}$
576 ru lxd by
577 $ru^{g}$ jv se by
578 $ry_{8}$
* ry . . . . . 101 . . . 101 . 471
579 Sb/In(3LR)Ubx$^{101}$, Ubx$^{101}$
580 Sb H/In(3R)C, cd
581 Sb Ubx/T(2;3)ap$^{Xa}$
582 $Sb^{63b}$/In(3LR)Ubx$^{130}$, Ubx$^{130}$ $e^{s}$
583 $Sb_{Spi}$/In(3LR)Cx
* $Sb_{V}$ . . . . . 885, 886
* $sbd_{2}$ . . . . . 603
584 $sbd^{2}$
* $sbd_{105}$ . . . . . 757
* $sbd^{1}$ . . . . . 456, 525
585 se
586 se h
587 se $rt^{2}$ $th^{s}$/In(3L)P, Me
588 se ss k $e^{s}$ ro
* sed . . . . . (see $Hn^{r3}$)
* sep . . . . . 556, 825, 885
589 Ser/In(3R)C. e l(3)e
590 snb
591 sr
592 sr gl
593 ss
594 ss bx $Su(ss)^{2}$
595 ss bxd k $e^{s}$/T(2;3)ap$^{Xa}$
596 $ss^{a}$
597 $ss^{aB}$
598 $ss^{a40a}$
599 st
600 st c(3)G ca/TM1, me ri $sbd^{1}$ ($sp^{2}$)
601 st in ri $p^{p}$
602 st Ki $p^{p}$
603 st sbd $e^{s}$ ro ca
604 st sr $e^{s}_{2}$ ro ca (tu-36a)
605 st sr $H^{2}$ ca/In(3R)$P^{W}$, st 1(3)W ca
606 $st^{sp}$
* su(pd)$_{B}$ . . (= su-pd) . . 679
607 $su(pr)^{2}$/TM6, ss$^{-}$ bx$^{34e}$ Ubx$^{P15}$ e (pr)
608 $su(Hw)^{2}$ bx bxd/TM1, Me ri $sbd^{1}$ ($sp^{2}$)
* $Su(ss)^{2}$ . .(= $Su^{2}$-ss) . . 594
609 su(t) (t)
* su(tu) . . (= su-tu) . . 693
610 su(ve)ru ve h th
611 th
612 th st cp
613 th st pb $p^{p}$/TM6, ss$^{-}$ bx$^{34e}$ Ubx$^{P15}$ e
614 th st pb $p^{p}$ cu kar $su(Hw)^{2}$ jvl ss bx sr gl/TM6, ss$^{-}$ bx$^{34e}$ Ubx$^{P15}$ e
* $tra_{D}$ . . . . . 674
* $tra^{D}$ . . . . . 844
615 Tri/In(3LR)DcxF
616 tt wo
617 Tu (= Tubby)
* tu-36e . . (= tu$^{36e}$) . . 447
* tuh-3 . . . . . 673
618 tx
619 Ubx $e^{4}$/In(3L+3R)P, Dfd ca
620 $Ubx^{61d}$/$H^{57c}$
* $Ubx_{101}$ . . . . . 579
* $Ubx^{130}$ . . . . . 484, 582, 674, etc.
621 ve
622 ve h th
623 ve R/In(3L)P, gm
* vo-3 . . . . . (see $e(dp^{v})$)
624 W
625 W Sb/In(3LR)Cx
626 We/In(3L)P, Me, In(3R)C, e l(3)e
627 wk/In(3L+3R)P, Dfd ca
628 wo

* Xa . .(= T(2;3)ap$^{Xa}$) . . 458, 543, etc.

## Chromosome 4

629 ar/ey$^{D}$
630 bt
631 bt ey$^{R}$ sv$^{n}$
632 bt$^{D}$/ci$^{D}$
633 Ce$^{2}$/spa$^{Cat}$
634 ci ey$^{R}$
635 ci ey$^{R}$ sv$^{n}$
636 ci gvl bt
637 ci gvl ey$^{R}$ sv$^{n}$
638 ci sv$^{n}$
639 ci$^{361}$
640 ci$^{57g}$
641 ci$^{D}$/ey$^{D}$
642 ci$^{W}$
643 ey$^{2}$
644 ey$^{4}$
645 ey$^{D}$
* ey$^{R}$ . . . . . 629, 641, 662
* ey$^{R}$ . . . . . 634, 635, etc.
646 gvl
647 gvl ey$^{R}$
648 gvl ey$^{R}$ sv$^{n}$
649 l(4)2$^{f}$/ci$^{D}$ (Hochman)
650 l(4)4$^{c}$/ci$^{D}$ "
651 l(4)6$^{b}$/ci$^{D}$ "
652 l(4)14$^{b}$/ci$^{D}$ "
653 l(4)15$^{2}$/ci$^{D}$ "
654 l(4)21/ci$^{D}$ "
655 l(4)22/ci$^{D}$ "
656 l(4)25/ci$^{D}$ "
* l(4)AM-1 . . . . . (see l(4)22)
* l(4)PT-1 . . . . . (see l(4)6$^{b}$)
* l(4)PT-2 . . . . . (see l(4)2$^{f}$)
* l(4)PT-3 . . . . . (see l(4)4$^{c}$)
* l(4)SLC-1 . . . . . (see l(4)15$^{2}$)
* l(4)ST-1 . . . . . (see l(4)21)
* l(4)ST-2 . . . . . (see l(4)14$^{b}$)
* l(4)ST-3 . . . . . (see l(4)25)
* Mal . . . . . 694
657 spa$^{Cat}$
658 spa$^{pol}$/ci$^{D}$
659 spa$^{p65}$
660 spa$^{35a}$
661 sv$^{de}$
662 sv$^{de}$/ey$^{D}$
663 sv$^{n}$

## Multichromosomal Stocks

664 br$^{3}$ dx$^{st}$;ed Su(dx)$^{2}$(1;2)
665 dx$^{st}$;Su(dx)(1;2)
666 e(S)$^{x}$/FMA3, y$^{2}$;al S ast ho/SM1, al$^{2}$ Cy sp$^{2}$(1;2)
667 lz$^{D}$/In(1)dl-49, m$^{2}$ g$^{4}$;bw$^{Vl}$/In(2L+2R)Cy, Cy(1;2)
668 os$^{s}$;tet(1;2)
669 v;bw(1;2)
670 v;In(2R)bw$^{VDel}$/SM1, al$^{2}$ Cy sp$^{2}$(1;2)
671 w$^{ch}$/FMA3, y$^{2}$;Su(w$^{ch}$)/In(2L+2R)Cy, Cy(1;2)
672 sc z w$^{z}$ rst;halo(1;3)
673 tuh-1;tuh-3(1;3)
674 w$^{a}$ v/FMA3, y$^{2}$;tra/In(3LR)Ubx$^{130}$, Ubx$^{130}$ e$^{s}$(1;3)
675 w$^{e}$;Dp(2;3)P/TM6, ss$^{-}$bx$^{34e}$ Ubx$^{P15}$ e(1;3)
676 y;mwh(1;3)
677 y$^{2}$su(Cbx)$^{2}$ v/FMA3, y$^{2}$;Cbx/T(2;3)ap$^{Xa}_{34e}$(1;3)
678 y$^{2}$ e(bx)$^{2}$ w$^{bf}_{1}$/FMA3, y$^{2}$;sbd$^{2}$ ss bx$^{34e}$/TM1, Me ri sbd$^{1}$(1;3)
679 ptg;px pd;su(pd)(1;2;3)
680 FMA3, y$^{2}$;net;sbd$^{2}_{R}$;spa$^{pol}$(1;2;3;4)
681 y f:=;bw;e;ci ey$^{R}$(1;2;3;4)
682 y f:=;bw;e;spa$^{pol}$(1;2;3;4)
683 al dp b Bl c px sp/In(2L+2R)Cy, Cy; D/In(3L+3R)P(2;3)
684 b Su(er)$^{+}$ bw;st er(2;3)
685 bw;st(2;3)
686 bw$^{4}$;st(2;3)
687 bw$^{Vl}$, dp b/In(2L+2R)Cy, Cy sp$^{2}$;Sb/In(3LR)DcxF(ru h ca?)(2;3)
688 bw$^{Vl}$, ds$^{33k}$/In(2L+2R)Cy. Cy;H/In(3R)Mo, sr(2;3)
689 cn;ry$^{2}$(2;3)
690 dp$^{v}$;e(dp$^{v}$)(2;3)
691 lys rc;ss(2;3)
692 px pd;Pdr H, Dp(2;3)P/Pdr(2;3)
692b Ubx$^{P15}$ e(2;3)
693 Su(er)tu bw;st er su(tu)(2;3)
694 pr;Mal(2;4)

## Attached-X

695 br ec/y$^{3d}$
696 f B/su(s)$^{S}$ v
* FMA3, y$^{2}$ . .(= FMA3) . . 128, 129, etc.
697 w$^{bf3}$/sn$^{36a}$
698 y/g$^{2}$ ty

699 y pn/FM6, $y^{31d}$ $sc^{8}$ dm B
* y pn v . . . . . 709
* y v bb . . . . . 786
* y v f . . . . . 720
* y v f car . . . . . 780
* y w bb . . . . . 26
* y w f . . . . . 783
* $y_2$ sc $w^{a}$ $ec^{a}$ . . . . . 712
700 $y^{}$ $su(w^{a})$ $w^{a}$ bb/y $sc^{4L}$ $sc^{8R}$

Attached Autosomal Arms

701 C(2L)P3, +;C(2R)P3, +
702 C(2L)P3, $j^{63}$;C(2R)P4, px
703 C(2L)P4, dp;C(2R)P4, px
704 C(3L)P3, ri;C(3R)P3, sr
705 C(3L)P6, +;C(3R)P6, +
706 C(4)P1, ci $ey^{R}$/gvl $sv^{n}$
707 C(4)P2. ci $ey^{R}$/gvl $sv^{n}$

Attached-XY

708 v f B, $\overline{XY}/y^{2}$ $su(w^{a})$ $w^{a}$ $bb^{59b}$
709 y $su(w^{a})$ $w^{a}$, $\overline{XY}^{L}$.$Y^{S}$/y pn v (Extra Y present)
710 $Y^{S}/g^{2}$ B.$Y^{L}$ and y f:=$(dp^{olv})$(Stern)
711 $Y^{S}$ X.$Y^{L}$, In(1)EN, In(1)dl-49, $Y^{S}$ y.$Y^{L}$/ y X.Y;bw;e;ci $ey^{R}$

Triploid

712a C(1)RM, In(1)dl-49, $v^{Of}$ f/FM7
712b C(1)RM, y w $fa^{no}$/FM6 ♀ and FM6, $y^{31d}$ $sc^{8}$ dm B/$B^{S}$ Y $y^{+}$ ♂
712c C(1)RM, $y^{2}$ sc $w^{a}$ ec/FM6 ♀ and FM6, $y^{31d}$ $sc^{8}$ dm B/$B^{S}$ Y $y^{+}$ ♂

Extra-Y

713 In(1)$w^{m4L}$ $N^{264\text{-}84R}$, y sn/FM3, $y^{31d}$ $sc^{8}$ dm B 1/Y ♀;dm sn ♂ (DIS 28: 137)
714 y v f mal/mal Y ♀;In(1)dl-49, $B^{M1}$, Df $(1)mal^{6}$, $y^{+}$ v sn $^{X2}$/$mal^{+}$ Y ♂
715 y v f mal/$y^{+}$ $mal^{+}$ Y ♀;l(T2-4a)/$y^{+}$ $mal^{+}$ $Y^{-bb}$ ♂
* $Y^{c2}$ . . . . . 786
716 In($X^{}$)$w^{vc}$/In(1)dl-49, y w $lz^{s}$ ♀;In(1)d dl-49, y w $lz^{s}$/$sc^{8}$ .Y ♂
717 $X^{c1}$, y/y f:=/$y^{+}$ Y
718 $X^{c2}$, cv v f/ClB, v

Closed-Y

719 R(Y)$bw^{+}$/X;bw ("MYR")
* $Y^{c}$, $bw^{+}$ . . . . . (see R(Y)$bw^{+}$)
720 $Y^{Lc}$/y w $Y^{S}$ and y v f

## Deficiencies

Deficiencies-X

721 Df(1)$260\text{-}1^{263\text{-}20}$ Df(1)260-1/FM4, $y^{31d}$ $sc^{8}$ dm B
722 Df(1)$B^{263\text{-}20}$ Df(1)B/In(1)$sc^{7}$, In(1)AM.$sc^{8}$ car
723 Df(1)bb Df(1)bb, y $sl^{2}$ bb-/FM4. $y^{31d}$ $sc^{8}$ dm B
724 Df(1)$bb_{1}$ Df(1)bb, y v car bb-/In(1)AM
* Df(1)$bb^{268\text{-}42}$ . . . . . 738
725 Df(1)$ct^{}_{1}$ Df(1)ct, y/FM4, $y^{31d}$ $sc^{8}$ dm B
726 Df(1)$g_{1}$ f B/In(1)AM
727 Df(1)$m^{259\text{-}4c}$ Df(1)m/FM4, $y^{31d}$ $sc^{8}$ dm B
728 Df(1)mal Df(1)mal/In(1)dl-49, $lz^{s}$
729 Df(1)$N^{8}$ Df(1)N/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B
730 Df(1)$N^{264\text{-}39}$ Df(1)N$^{264\text{-}39}$ $w^{ch}$/FM4, $y^{31d}$ $sc^{8}$ dm B
731 Df(1)$N^{264\text{-}105}$ Df(1)N$^{264\text{-}105}$/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B
* Df(1)$rst^{2}$ $sc^{4L}$ $sc^{8R}$ . . . . . 95
* Df(1)$sc^{8}$ . . . . . 795
* Df(1)sc . . . . . 769
732 Df(1)$svr^{258\text{-}11}$ Df(1)svr, Dp(1;f)101, spl/y f:=
733 Df(1)$w^{258\text{-}11}$ Df(1)$w^{258\text{-}42}$, y/In(1)dl-49, y Hw $m^{2}$ $g^{4}$
734 Df(1)$w^{258\text{-}42}$ Df(1)$w^{258\text{-}45}$, y/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B
735 Df(1)$w^{258\text{-}45}$ Df(1)$w^{258\text{-}48}$, y/FM4, $y^{31d}$ $sc^{8}$ dm B
736 Df(1)$w^{258\text{-}48}$ Df(1)$w^{vco}$, y $sc^{5}$ spl;Dp(1;3)$w^{vco}$;y f:=

## Deficiencies-Y

737 Df(Y)$Y^{bb-}$ Df(Y)$Y^{bb-}$, $y^{2}$ $w^{e}$ $bb^{1}$ $eq^{e}$ $y^{1}$ $w^{e}$ $bb^{1}$/$w^{e}$ $bb^{1}$;$Y^{st}$ and $w^{e}$ $bb^{1}$;$Y^{+}$;In(2L+2R)NS, px sp/l(2)$mr^{2}$

738 Df(Y)$Y^{st}$

## Deficiencies-2

739 Df(2)M33a Df(2)M33a/$bw^{V32g}$

* Df(2)MB . . . . . (see Df(2L)M-$z^{B}$)

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

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

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

743 Df(2)$rl^{10a}$ Df(2)$rl^{10a}$ lt cn/$bw^{V1}$, $ds^{33k}$

744 Df(2L)al Df(2L)al/In(2L+2R)Cy. Cy E(S)

745 Df(2L)M-$z^{B}$ Df(2L)M-$z^{B}$/SM1, $al^{2}$ Cy $sp^{2}$

746 Df(2L)S2 Df(2L)S2/In(2L+2R)Cy, Cy E(S)

747 Df(2L)S3 Df(2L)S3/SM1, $al^{2}$ Cy $sp^{2}$

748 Df(2R)$42_{5}$ Df(2R)$42_{5}$ en/SM1, $Al^{2}$ Cy $sp^{2}$

749 Df(2R)$bw_{2}^{VDe2L}$ Df(2R)$bw_{2}^{VDe2L}$, $sp^{2}$/T(2;3)$ap_{R}^{Xa}$

750 Df(2R)$bw_{2}^{VDe2L}$ $Cy^{R}$ Df(2R)$bw_{2}^{VDe2L}$, In(2R)$Cy^{R}$/Gla

751 Df(2R)$Px_{2}^{B}$ Df(2R)$Px_{2}^{B}$, bw sp/SM1, $al^{2}$ $Cy^{v}$ $sp^{2}$

752 Df(2R)$vg_{C}$ Df(2R)$vg_{C}$/SM5, $al^{2}$ Cy $lt^{v}$ sp

753 Df(2R)$vg_{C}$ Df(2R)$vg_{C}$/In(2LR)$Rev^{B}$

754 Df(2R)$vg_{D}$ Df(2R)$vg_{D}$/SM5, $al^{2}$ Cy $lt^{v}$ $sp^{2}$

* Df(2R)$vg^{D}$ . . . . . (= $vg^{D}$) . . . . . 436

## Deficiencies-3

* Df(3L)Hn . . . . . 879

* Df(3L)Ly . . . . . (= Ly) . . . . . 527, 528

755 Df(3R)M-S31 Df(3R)M-S31/T(2;3)Me

756 Df(3R)ry Df(3R)ry/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^{s}$

757 Df(3R)$sbd^{105}$ Df(3R)$sbd^{105}$, $p^{sbd^{105}}$ bx sr $e^{s}$/LVM

## Deficiencies-4

758 Df(4)M Df(4)M/$ey^{D}$

## Duplications

* Dp(1;f)24 . . . . . (=$_{8}$Del(1)24) . . . . . 62, 808

759 Dp(1;f)101 Dp(1;f)101;In(1)$sc_{8}^{a}$, Df(0+ac)·$w^{a}$ $sc_{8}^{a}$·

760 Dp(1;f)107 Dp(1;f)107;In(1)$sc_{8}^{a}$, Df(0+ac)·$w^{a}$ $sc_{8}^{a}$·

761 Dp(1;f)118 Dp(1;f)118;In(1)$sc_{8}^{a}$, Df(0+ac)·$w^{a}$ $sc_{8}^{a}$·

762 Dp(1;f)135 Dp(1;f)135, $y^{2}$;In(1)$sc_{8}^{a}$, Df(0+ac)·$w^{a}$ $sc_{8}^{a}$·

763 Dp(1;f)R Dp(1;f)R/y dor/y dor

* Dp(1;f)$X_{9}^{c2}$ . . . . . (see Dp(1;f)R)

764 Dp(1;f)z Dp(1;f)$z^{9}$, Df(1)$sc^{J4}$/y f:=

765 Dp(1;1)112 Dp(1;1)112, $y_{50e}^{+}$ f (homozygous stock)

766 Dp(1;1)lz Dp(1;1)lz, $lz^{lz^{y4}}$/y f:=

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

768 Dp(1;3)126 Dp(1:3)126;v f/In(3L+3R)P, Dfd ca
769 Dp(1;3)sc$^{J4}$ Dp(1;3)sc$^{J4}$/Df(1)sc$^{8}$, w$^{a}$
* Dp(1;3)w$^{vco}$ . . . . . 736
770 Dp(2;2)S Dp(2;2)S, (S ast) (S ast$^{4}$) net dp cl/In(2L+2R)Cy, Cy E(S)
* Dp(2;3)P . . . . . 692
771 Qn(2;2)S Qn(2;2)S, (ast)$_{5}$, al ho/In(2L+2R)Cy, Cy S$^{2}$ E(S)

## Inversions

### Inversions-X

772 In(1)AB In(1)AB/y f:=
* In(1)AM . . . . . 14, 88, etc.
773 In(1)B$^{M1}$ In(1)B$^{M1}$, v B$^{M1}$ (tan-like); . . see also . . 784, 785, etc.
774 In(1)B$^{M2}$ In(1)B$^{M2}$(rv) f$^{B15}$ (reinv.; mosaic)
775 In(1)B$^{M2}$ In(1)B$^{M2}$, f$^{B27}$ B$^{M2}$/ClB
776 In(1)B$^{M2}$ In(1)B$^{M2}$ v$^{rv}$ B$^{M2}$
* In(1)bb . . . . . 723, 724
777 In(1)ClB$_{36d}$ In(1)Cl, sc t$^{2}$ v sl B$_{36d}$ (= ClB) . . . . . 80, 718
778 In(1)ClB$^{36d}$ In(1)Cl, sc t$^{2}$ v sl B$^{36d}$ (= ClB$^{36d}$) . . . . . 851
779 In(1)dl-49 In(1)dl-49, ty1
780 In(1)dl-49 In(1)dl-49, ty1 bb$^{1}$/y v f car
781 In(1)dl-49 In(1)dl-49, v$^{Of}$ f$^{n}$
782 In(1)dl-49 In(1)dl-49, y fa
* In(1)dl-49 In(1)dl-49, y Hw m$^{2}$ g$^{4}$ . . . . . 733, 798
783 In(1)dl-49 In(1)dl-49, y Su(Hw) Hw m$^{2}$ g$^{4}$/y$^{J1}$ w f;(nub/+)
784 Ins(1)dl-49, B$^{M1}$ In(1)dl-49, In(1)B$^{M1}$, l(1)J1 sc$^{S1}$ sc$^{8}$ pn$^{+}$ w$^{+}$ ec$^{+}$ rb$^{+}$ cm$^{+}$ ct$^{6}$ sn$^{3}$ ras$^{2}$ g$^{2}$ f$^{+}$ os$^{s}$ os$^{o}$ car 1/l(1)J1$^{+}$.Y (= "Maxy")
785 Ins(1)dl-49, B$^{M1}$ In(1)dl-49, In(1)B$^{M1}$, sc v B$^{M1}$ (homozygous)
786 Ins(1)dl-49, B$^{M1}$ In(1)dl-49, In(1)B$^{M1}$, y/Y$^{-bb}$ and y v bb/Y$^{-bb}$
787 Ins(1)dl-49, B$^{M1}$ In(1)dl-49, In(1)B$^{M1}$, y sc v cu-X B$^{M1}$
788 In(1)e(bx) In(1)e(bx), e(bx)/y f:=
* In(1)EN . . . . . 711
* Ins(1)FM1 In(1)FM1, In(1)dl-49, y$^{31d}$ sc$^{8}$ w$^{a}$ lz$^{s}$ B (= FM1) . 18, 27, etc.
* In(1)FM3 In(1)FM3, y$^{31d}$ sc$^{8}$ dm B 1 (= FM3) . . . . . 9, 53, etc.
* In(1)FM4 In(1)FM4, y$^{31d}$ sc$^{8}$ dm B (= FM4) . . . . . 712, 723
789 In(1)FM6 In(1)FM6 y$^{31d}$ sc$^{8}$ dm B/y f:=; . . . . see also 32, 33, etc.
790 In(1)FM7 In(1)FM7a, y$^{2}$ w$^{a}$ v$^{Of}$ B (homozygous) (see DIS 44: 101)
* In(1)FMA3$^{264-84}$ In(1)FMA3, y$^{2}$ (= FMA3)$^{31d}$ . . . . . 128, 129
791 In(1)N$^{264-84}$ In(1)N$^{264-84}$, y/FM6, y$^{31d}$ sc$^{8}$ dm B
792 In(1)rst$^{3}$ In(1)rst$^{3}$, rst$^{3}$ (homozygous)
793 In(1)rst$^{3}$ In(1)rst$^{3}$, y rst$^{3}$ car bb
* In(1)S . . . . . 809, 813
794 In(1)sc$^{4}$ In(1)sc$^{4}$, y sc$^{4}$
795 In(1)sc$_{7}^{4L}$ sc$^{8R}$ In(1)sc$_{7}^{4L}$ sc$^{8R}$, y; . . . . . see also 700
796 In(1)sc$^{7}$ In(1)sc$^{7}$, sc$^{7}$
797 In(1)sc$^{7}$ In(1)sc$^{7}$, sc$^{7}$ w$^{a}$
798 Ins(1)sc$^{7}$, AM In(1)sc$^{7}$, In(1)AM, sc$^{7}$/In(I)dl-49, y Hw m$^{2}$ g$^{4}$
799 Ins(1)sc$^{7}$, AM In(1)sc$^{7}$, In(1)AM, sc$^{7}$ car/FM4, y$^{31d}$ sc$^{8}$ dm (without B)
800 Ins(1)sc$^{7}$, B$^{M1}$ In(1)sc$^{7}$, In(1)B$^{M1}$, sc$^{7}$ w$^{43b}$ B$^{M1}$/y f:=

801 In(1)sc$^{8}_{8}$ In(1)sc$^{8}_{8}$, sc$^{8}_{8}$

802 In(1)sc$^{8}_{8}$ In(1)sc$^{8}_{8}$, sc$^{31d}_{8}$cv v f/y f:=

803 In(1)sc$^{8}_{8}$ In(1)sc$^{8}_{8}$, y sc$^{31d}_{8}$ w$^{a}$

804 In(1)sc$^{8}_{8R}$, dl-49 In(1)sc$^{8}_{8R}$, In(1)dl-49, y$^{31d}$ sc$^{8}$ (homozygous)

* In(1)sc$^{9}_{9}$ . . . . . 700, 784

805 In(1)sc$^{9}_{9}$, sc$^{9}_{9}$ Bx f t w$^{a}$ (homozygous)

806 In(1)sc$^{260-14}_{260-14}$ In(1)sc$^{260-14}_{260-14}$, sc$^{260-14}_{260-14}$

807 In(1)sc$^{260-22}_{260-22}$ In(1)sc$^{260-22}_{260-22}$, sc$^{260-22}_{260-22}$

808 In(1)sc$^{J1}_{J1}$ In(1)sc$^{J1}_{J1}$;Dp(1;f)24

* Ins(1)sc$^{S1}_{S1L}$, dl-49 In(1)sc$^{S1}_{S1L}$, In(1)dl-49, y v B$^{8}_{8R}$ . . . . . 178

* Ins(1)sc$^{S1L}_{s}$, sc$^{8R}_{os}$ In(1)sc$^{S1L}_{os}$. In(1)sc$^{8R}_{car}$, y sc$^{S1}_{1}$ sc$^{8}_{8}$ pn w ec rb cm ct$^{6}$ sn$^{3}$ ras$^{2}$ g$^{2}$ f . . . . . 784

809 Ins(1)sc$^{S1L}_{S1L}$, S, sc$^{8R}_{S1R}$ In(1)sc$^{S1L}_{S1L}$, In(1)S, In(1)sc$^{8R}_{S1R}$, sc$^{8}_{8}$ w$^{a}$ B (= Muller-5)

* In(1)sc$^{S1R}_{S1R}$ . . . . . 813

810 In(1)w$^{m4}_{m4}$ In(1)w$^{m4}_{m4}$ (bb?)

811 In(1)w$^{m4}_{3P}$ In(1)w$^{m4}_{3P}$, y cv m f/y f:=

812 In(1)y$^{3PL}_{3P}$ In(1)y$^{3PL}_{3P}$, y B (B reverted)

813 Ins(1)y$^{3PL}_{4}$, S, sc$^{S1R}_{4}$ In(1)y$^{3PL}_{4}$, In(1)S, In(1)sc$^{S1R}_{4}$/y f:=;sc$^{19i}_{4}$/In(2L+2R)Cy, Cy

814 In(1)y$^{4}_{4}$ In(1)y$^{4}_{4}$, y$^{4}_{4}$

## 2L Inversions

815 In(2L)Cy In(2L)Cy, al$^{2}$ ast$^{3}$ b pr (does not carry Cy mutant)

* In(2L)Cy$_{L}$ In(2L)Cy$_{L}$, Cy dp$^{lv2}$ b pr . . . . . 293, 350

* In(2L)Cy$_{L}$t$_{R}$ In(2L)Cy$_{L}$t$_{R}$, Su(S) dp$^{lv2}$ pr . . . . . 292

* In(2L)NS . . . . . 317

* In(2L)t . . . . . 328

816 In(2L)t In(2L)t, esc c sp/SM5. al$^{2}$ Cy lt$^{v}$ sp$^{2}$

817 In(2L)t In(2L)t, lt l L$^{4}$ sp$^{2}$/bw$^{Vl}$, ds$^{33k}$

* In(2L)t In(2L)t, l(2)R . . . . . 411

818 In(2L)Tg In(2L)Tg, Tg/SM5, al$^{2}$ Cy lt$^{v}$ sp$^{2}$

## 2L + 2R Inversions

819 In(2L+2R)Cy In(2L+2R)Cy, al$^{2}$ E(S) cn$^{2}$ sp$^{2}$ (does not carry Cy mutant)

* In(2L+2R)Cy In(2L+2R)Cy, al$^{2}$ Cy lt$^{3}$ L$^{4}$ sp$^{2}$ . . . . . 291, 864, 888

* In(2L+2R)Cy, Cy . . . . . 273, 384, etc.

* In(2L+2R)Cy, Cy bw$^{45a}_{lv2}$ sp$^{2}$ or$^{45a}$ . . . . . 241

* In(2L+2R)Cy, Cy dp$^{lvI}_{lv2}$ . . . . . 303

* In(2L+2R)Cy, Cy dp$^{lvI}_{lv2}$ Bl L$^{4}$ sp$^{2}$ . . . . . 204

* In(2L+2R)Cy, Cy dp$^{lvI}_{lv2}$ pr . . . . . 838

* In(2L+2R)Cy, Cy E(S)$^{2}$ . . . . . 395, 744, etc.

* In(2L+2R)Cy, Cy hk$^{2}$ . . . . . 205

* In(2L+2R)Cy, Cy L$^{4}$ sp$^{2}$ . . . . . 862, 869

* In(2L+2R)Cy, Cy pr$^{2}$ . . . . . 425, 887, etc.

* In(2L+2R)Cy, Cy sp$^{2}$ . . . . . 305, 687

* In(2L+2R)Cy, Cy S$^{2}$ E(S)$^{lv2}$ . . . . . 771

* In(2L+2R)Cy, Cy S$^{2}$ dp$^{lv2}$ E(S)$^{V34k}$ . . . . . 335

* Ins(2L+2R)Cy, bw$^{V34k}$ In(2L+2R)Cy, Cy$_{lv2}$In(2R)bw . . . . . 342

* Ins(2L)Cy, (2R)NS In(2L)Cy. Cy dp$^{lv2}$ pr, In(2R)NS. l px l(2)NS sp . 361

820 In(2L+2R)NS In(2L+2R)NS, b mr/In(2L+2R)Cy, Cy

| | | | |
|---|---|---|---|
| * | In(2L+2R)NS | In(2L+2R)NS, px sp | 738 |
| * | Ins(2L)t, (2R)Cy | In(2L)t, Roi In(2R)Cy, bw $sp^2$ or (= Roi) | 441 |

2LR Inversions

| | | | |
|---|---|---|---|
| 821 | In(2LR)102 | In(2LR)102, $ds^W$ $sp^2$/SM1, $al^2$ Cy $sp^2$ | |
| * | In(2LR)$bw^{V1}$ | In(2LR)$bw^{V1}$, $ds^{33k}$ | 328, 353, etc. |
| * | In(2LR)$bw^{V32g}$ | | 352, 739 |
| * | In(2LR)dp | | (see T(2;3)dp) |
| * | In(2LR)Gla | (= Gla) | 232 |
| * | In(2LR)$Pm_2$ | | (see In(2LR)$bw^{V1}$) |
| * | In(2LR)$Pm^2$ | | (see In(2LR)$bw^{V32g}$) |
| * | In(2LR)$Rev_B$ | (= Rev) | 823 |
| * | In(2LR)$Rev^B$ | | 753 |
| * | In(2LR)Rvd | | (see In(2LR)$Rev^B$) |
| * | In(2LR)SM1 | In(2LR)SM1, $al^2$ Cy $sp^2$ (= SM1) | 212, 216, etc. |
| * | In(2LR)SM5 | In(2LR)SM5, $al^2$ Cy $lt^v$ $sp^2$ (= SM5) | 206 210, etc. |
| * | In(2LR)U | (= U) | 258 |

2R Inversions

| | | | |
|---|---|---|---|
| * | In(2R)$bw^{V34k}$ | | |
| | In(2R)$bw^{VDe1}$ | In(2R)$bw^{VDe1}$ | 342 |
| 822 | In(2R)$bw^{VDe2}$ | In(2R)$bw^{VDe2}$, b/b lt l cn mi sp | |
| 823 | In(2R)bw | In(2R)bw/In(2LR)Rev l | |
| * | In(2R)$Cy_K$ | In(2R)Cy, $cn^2$ Bld | 354 |
| * | In(2R)Mo | | 868 |
| * | In(2R)NS | | 361 |

3L Inversions

| | | | |
|---|---|---|---|
| * | In(3L)$D_3$ | (= D) | 476, 522, etc. |
| * | In(3L)$D^3$ | (= $D^3$) | 477, 527 |
| * | In(3L)P | In(3L)P, gm | 559, 623 |
| * | In(3L)P | In(3L)P, Me | 531, 532, etc. |
| * | In(3L)P | In(3L)P, Me ca | 892, 894 |
| 824 | In(3L)P | In(3L)P, not-36e/R | |

3L + 3R Inversions

| | | | |
|---|---|---|---|
| * | In(3L+3R)LVM | (= LVM) | 506 523, 528 |
| * | In(3L+3R)P | In(3L+3R)P, l(3)PL l(3)PR (= Payne) | 477, 490, etc. |
| * | | In(3L+3R)P, l(3)PL l(3)PR, Dfd ca | 486, 487, etc. |
| * | Ins(3L)P, (3R)C | In(3L)P, Me, In(3R)C, e l(3)e | 626 |
| * | | In(3L)P, Me, In(3R)C, Sb e l(3)e | 563 |
| * | Ins(3L)P, (3R)P18 | In(3L)P, In(3R)P18, Me $Ubx^4$ e | 524 |

3LR Inversions

| | | | |
|---|---|---|---|
| * | In(3LR)Cx | (= Cx) | 479, 498, etc. |
| * | In(3LR)Cx | In(3LR)Cx, D | 522, 893 |
| * | In(3LR)DcxF | (= In(3LR)CxF) | 615, 687 |

* In(3LR)DcxF — In(3LR)DcxF, ru h ca . . . . . 840

825 In(3LR)sep — In(3LR)sep, sep ri pP

* In(3LR)P35 — . . . . . (= In(3LR)Pasadena-35) . . . . . 886

* In(3LR)TM1 — In(3LR)TM1, Me ri $sbd^{1}$ . . . . . (= TM1) . . . . 456, 525, etc.

* In(3LR)TM3 — In(3LR)TM3, $y^{+}$ $ac^{+}$ ri $p^{p}$ sep $bx^{34e}$ $e^{s}$ (= TM3) . . 556, 885

* In(3LR)$Ubx^{101}$ — . . . . . $^{130}$ . . $^{130}$ . . . . . $^{130}$ . . . . . 579

* In(3LR)$Ubx^{130}$ — In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^{s}$ . . (= $Ubx^{130}$) . . 484, 582, 674, etc.

## 3R Inversions

826 In(3R)$Antp^{B}$ — In(3R)$Antp^{B}$, $Antp^{B}$/TM1, Me ri $sbd^{1}$

* In(3R)C — In(3R)C, cd . . . . . 580

* In(3R)C, e . . . . . 482, 485, etc.

* In(3R)C, e l(3)e . . . . . 536, 537, etc.

* In(3R)C, l(3)a . . . . . 453, 483

* In(3R)C, Sb e l(3)e . . . . . 488, 514

* In(3R)Cyd — In(3R)Cyd, Cyd . . . . . (= Cyd) . . . . . 489

827 In(3R)$Dl^{B}$ — In(3R)$Dl^{B}$, st $Dl^{B}$/In(3R)$P^{W}$. st l(3)W ca

828 In(3R)Hu — In(3R)Hu. Hu $Sb^{Spi}$/In(3L+3R)P

829 In(3R)Mo — In(3R)Mo, sr/T(2;3)$ap^{Xa}$, ca; see also 688

* In(3R)P — . . . . . 481, 511

* In(3R)P18 — . . . . . (= In(3R)Pasadena 18) . . . . . 524

830 In(3R)$P^{Fla}$ — In(3R)$P^{Fla}$ (homozygous)

* In(3R)$P^{W}$ — In(3R)$P^{W}$, st l(3)W ca . . . . . 605, 827

## Translocations-1;Y

831 T(1;Y)1E — T(1;Y)1E, y/y f:=, cn bw

832 T(1;Y)2E — T(1;Y)2E/v car l(Stern #64)/y f:=; cn bw

## Translocations-1;2

833 T(1;2)$Bld^{257-15}$ — T(1;2)Bld, Bld/ClB (carries In(2R)Cy)

834 T(1;2)$f^{257-15}$ — T(1;2)$f^{257-15}$/In(1)AM

835 T(1;2)1t — T(1;2)1t/In(2L+2R)Cy, Cy (carries eq and possibly su(s)$^{3}$)

836 T(1;2)$N^{264-10}$ — T(1;2)$N^{264-10}$/FM6. y $^{31d}$ $sc^{8}$ dm B

837 T(1;2)$sc^{S2}_{19}$ — T(1;2)$sc^{S2}_{19}$/In(2L+2R)Cy. Cy

838 T(1;2)$sc^{19i}$ — T(1;2)$sc^{19i}$/y f:=;fs(2)B sc b pr/In(2L+2R)Cy, Cy $dp^{lvI}$ pr

## Translocations-1;3

839 T(1;3)263-4 — T(1;3)263-4, y $sc^{s}$ $B^{1}$/In(1)AM

840 T(1;3)143-3 — T(1;3)143-3, ru $e^{s}$ ca/In(3LR)DcxF, ru h ca

* T(1;3)Del-143" — . . . . . (see T(1;3)143-3)

841 T(1;3)$N^{264-6}$ — T(1;3)$N^{264-6}$, y/y w dm (= $N^{6}$)

842 T(1;3)04 — T(1;3)04/ClB

843 T(1;3)05 — T(1;3)05, D/y f:=

844 T(1;3)OR60 — T(1;3)OR60/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^{s}$ ♀;$tra^{D}$ Sb e/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^{s}$ ♂

845 T(1;3)$ras^{v}_{J4}$ — T(1;3)$ras^{v}$/y f:=

* T(1;3)sc — . . . . . 856

846 T(1;3)sc$^{z}$ T(1;3)sc$^{z}$/y f:=
847 T(1;3)sc$^{260-15}$ T(1;3)sc$^{260-15}$/FM6, y$^{31d}$ sc$^{8}$ dm B
848 T(1;3)sta T(1;3)sta/FM3, y$^{31d}$ sc$^{8}$ dm B I
849 T(1;3)sta T(1;3)sta/y f:=
850 T(1;3)v, v$^{vco}$ T(1;3)v, v/FM6, y$^{31d}$ sc$^{8}$ dm B
851 T(1;3)w$^{vco}$ T(1;3)w$^{vco}$, v f/ClB$^{36d}$

Translocations-1;4

852 T(1;4)B$^{S}_{8a}$ T(1;4)B$^{S}$/y f:=
* T(1;4)N$^{264-12}$ . . . . . (see T(1;4)N$^{264-12}$)
853 T(1;4)N$^{264-12}$ T(1;4)N$^{264-12}$/FM6, y$^{31d}$ sc$^{8}$ dm B
854 T(1;4)sc$^{8}_{m5}$ T(1;4)sc$^{8}_{m5}$, B$^{a}_{D}$w/y f:=
855 T(1;4)w$^{m5}$ T(1;4)w$^{m5}$/ey
856 T(1;4)w$^{m5}_{m258-18}$(1;3)sc$^{J4}$ T(1;4)w$^{m5}_{m258-18}$;T(1;3)sc$^{J4}_{D}$(ClB)
857 T(1;4)w$^{m258-18}$ T(1;4)w$^{m258-18}$, y/ci
858 T(1;4)w$^{m258-21}$ T(1;4)w$^{m258-21}$/FM1, y$^{31d}$ sc$^{8}$ w$^{a}$ lz$^{s}$ B
859 T(1;4)w$^{m258-21}_{VD3}$ T(1;4)w$^{m258-21}_{a}$, y w/FM4, y$^{31d}$ sc$^{8}$ dm B
* T(1;4)w$^{VD3}$ . . . . . (see T(1;4)w$^{m258-21}$)

Translocations-Y;2

* T(Y;2)A . . . . . 336
860 T(Y;2)B T(Y;2)B/b; see also 422
* T(Y;2)C . . . . . 271, 373
* T(Y;2)E . . . . . 203, 266
* T(Y;2)G . . . . . 238
* T(Y;2)J . . . . . 381
861 T(Y;2)rl T(Y;2)rl, lt cn/b lt bw

Translocations-Y;2;3

* T(Y;2;3)F . . . . . 564

Translocations-2;3

862 T(2;3)101 T(2;3)101, al$^{2}$ sp$^{2}$/In(2L+2R)Cy, Cy L$^{4}$ sp$^{2}$
863 T(2;3)101 T(2;3)101;ru h e$^{4}$ ro ca/In(3L+3R)P, Dfd ca
864 T(2;3)108 T(2;3)108, al c sp$^{2}$/In(2L+2R)Cy, al$^{2}$ Cy lt$^{3}$ L$^{4}$ sp$^{2}$
865 T(2;3)109 T(2;3)109, p$^{p}$/In(3L+3R)P, Dfd ca
866 T(2;3)A T(2;3)A, Bl;ru h D TA ss e$^{s}$/In(3L+3R)P
* T(2;3)ap$^{Xa}$ . . . . . (= Xa) . . . . . 458,543, etc.
* T(2;3)ap$^{Xa}$ T(2;3)ap$^{Xa}$, ca . . . . . 557, 829
867 T(2;3)ap$^{Xa}$ T(2;3)ap$^{Xa}$/l(3)XaR
868 T(2;3)Ata, T(2;3)Ata, Ata/T(2;3)Mot-K
869 T(2;3)B T(2;3)B, al sp$^{2}$/In(2L+2R)Cy, Cy L$^{4}$ sp$^{2}$
870 T(2;3)B$^{V4}$ T(2;3)B;ru h D TB ss e$^{s}$/In(3L+3R)P
871 T(2;3)bw$^{V5}$ T(2;3)bw$^{V4}$/SM1, al$^{2}$ Cy sp$^{2}$
872 T(2;3)bw$^{VDe3}$ T(2;3)bw$^{V5}$/SM5, al$^{2}$ Cy lt$^{v}$ sp$^{2}$
873 T(2;3)bw$^{VDe4}$ T(2;3)bw$^{VDe3}$;Ubx bxd/In(3LR)Cx
874 T(2;3)bw$^{V}$ T(2;3)bw$^{VDe4}$/SM5, al$^{2}$ Cy lt$^{v}$ sp$^{2}$

875 T(2;3)C T(2;3)C;ru h D TC ss $e^s$/In(3L+3R)P

* T(2;3)$dp_D$ . . . . . 242

876 T(2;3)$dp^D$ T(213)$dp^D$, $dp^D$/SM1, $al^2$ $Cy_{sp^2}$

877 T(2;3)E T(2;3)E/SM5, $al^2$ Cy $lt^v$ $sp^2$

878 T(2;3)Hm T(2;3)Hm, Hm/In(2L+2R)Cy. Cy

879 T(2;3)Hn T(2;3)Hn, Df(3L)Hn, Hn/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^s$

* T(2;3)Me . . . . . 533, 534, etc.

* T(2;3)$P_{Gr}$ T(2;3)P, P . . . . . 675, 692

* T(2;3)$p^{Gr}$ . . . . . (see T(2;3)$Pu^{Gr}$)

880 T(2;3)$Pu^4_{Gr}$ T(2;3)$Pu^4_{Gr}$, $Pu^4_{Gr}$/C(3)x

881 T(2;3)$Pu^{Gr}$ T(2;3)$Pu^{Gr}$, $Pu^{Gr}$/SM1, $al^2$ Cy $sp^2$

882 T(2;3)rn T(2;3)rn/In(2R)Cy

883 T(2;3)Dp-S T(2;3)Dp-S, ho/In(2L+2R)Cy, Cy E(S) (hom. viable)

884 T(2;3)$S^L$ T(2;3)$S^L$/In(2L+2R)Cy, Cy E(S)

* T(2;3)$S^M$ . . . . . 440

885 T(2;3)$Sb^V$ T(2;3)$Sb^V$, $Sb^V$, In(3R)Mo/TM3, $y^+$ $ac^+$ ri $p^p$ sep $bx^{34e}$ $e^s$

886 T(2;3)$Sb^V$ T(2;3)$Sb^V$, $Sb^V$, In(3R)Mo, In(3LR)P35/Sm1, $al^2$ Cy $sp^2$;In(3LR)$Ubx^{130}$ $Ubx^{130}$ $e^s$

* T(2;3)Xa . . . . . (see T(2;3)$ap^{Xa}$)

Translocations-2;4

887 T(2;4)a T(2;4)a/In(2L+2R)Cy, Cy pr; $ey^2$

888 T(2;4)$ast^v$ T(2;4)$ast^v$/In(2L+2R)Cy, $al^2$ Cy $lt^3$ $L^4$ $sp^2$

889 T(2;4)b T(2;4)b/In(2L+2R)Cy, Cy pr;$ey^2$

890 T(2;4)d T(2;4)d, al dp px sp/In(2L+2R)Cy, Cy pr;$ey^2$

891 T(2;4)d T(2;4)d/In(2L+2R)Cy, Cy pr

Translocations-3;4

892 T(3;4)A2 T(3;4)A2/In(3L)P, Me ca

893 T(3;4)A12 T(3;4)A12/In(3LR)Cx, D

894 T(3;4)A13 T(3;4)A13, ve ca/In(3L)P, Me ca

895 T(3;4)A28 T(3;4)A28, ve ca (homozygous)

896 T(3;4)c T(3;4)c/In(3L+3R)P, Dfd ca

897 T(3;4)e T(3;4)e/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^s$

898 T(3;4)e T(3;4)e, h th st cu sr $e^s$ ca/In(3L+3R)P, Dfd ca

899 T(3;4)f T(3;4)f/In(3L)P, Me

900 T(3;4)f T(3;4)f, h th st cu sr $e^s$ ca/In(3L+3R)P, Dfd ca

Transpositions

901 Tp(3)$bxd^{100}_{107}$ Tp(3)$bxd^{100}_{107}$, ri/T(2;3)Me

902 Tp(3)$bxd^{107}$ Tp(3)$bxd^{107}$, bx $bxd^{107}$ sr $e^s$/$bx^{34e}$ Mc

903 Tp(3)Vno Tp(3)Vno/$H^2$

## ROMA, ITALY: CITTA' UNIVERSITARIA
Istituto di Genetica

### Wild Stocks

A 1 Canton-S
A 2 Marzi
A 3 Oregon

### Chromosome 1

B 1 car bb
B 2 e($w^e$)$w^e$/C(1)DX, y f
B 3 gt v
B 4 mal
B 5 pn
B 6 $r^9$/C(1)DX, y f
B 7 sc cv v f B/C(1)DX, y f
B 8 sc z ec
B 9 sw
B10 w
B11 $w^a$
B12 $w^{Bwx}$
B13 $w^{cf}$
B14 $w^{cf}$/C(1)DX, y f
B15 $w^{ch}$
B16 $w^{bl}$
B17 y
B18 y ac sc pn/C(1)DX, y f
B19 y cv v g/C(1)DX, y f
B20 y $fa^n$ $sn^3$
B21 y w
B22 y $w^a$ spl rb
B23 $y^2$ v mal
B24 $y^2$ $w^{cf}$
B25 $y^{31d}$ $v^{Of}$ $w^a$ f sn/C(1) DX, y f
B26 y l(1)J $l^{259}$ w m f/S-5/ $sc^8$.Y

### Chromosome 2

C 1 b cn c bw
C 2 b cn vg bw
C 3 b pr cn
C 4 Bl L/Cy Su(Cy)
C 5 Bl $L^2$/SM5, $al^2$ Cy $lt^v$ $cn^2$ $sp^2$
C 6 Bl $stw^{48}$ $ap^{blt}$ tuf sp/ SM5, $al^2$ Cy $lt^v$ $cn^2$ $sp^2$
C 7 bw
C 8 $bw^D$
C 9 cn bw
C10 cn en/SM5, $al^2$ Cy $lt^v$ $cn^2$ $sp^2$
C11 cv-2
C12 ds S G b br/Cy, $al^2$ $lt^3$ $L^4$ $sp^2$
C13 Frd/SM5, $al^2$ Cy $lt^v$ $cn^2$ $sp^2$
C14 $L^2$/SM5, $al^2$ Cy $lt^v$ $cn^2$ $sp^2$
C15 Pin/Cy
C16 Pin L/Cy
C17 pr cn
C18 px bw sp
C19 S Sp $ab^2$ ltd/SM5, $al^2$ Cy $lt^v$ $cn^2$ $sp^2$
C20 Sp/Cy
C21 Sp Bl L/Cy
C22 Sp Bl L Pin/Cy
C23 Sp J Pin/Cy
C24 Sp b pr cn $L^2$ Pin/SM5, $al^2$ Cy $lt^v$ $cn^2$ $sp^2$
C25 Tft/Cy

### Chromosome 3

D 1 bul
D 2 ca
D 3 ca K-pn
D 4 $D^3$ H/In(3L)P, Me
D 5 eg/In(3LR)Cx
D 6 $eg^2$/In(3LR)Cx
D 7 Gl/In(3LR)$bx^{130}$
D 8 Gl Sb/LVM
D 9 Gl Sb/In(3LR)$Ubx^{130}$
D10 $Hn^{r-3}$ sr
D11 H/Sb sr In(3R)Me
D12 $H^2$/Tp(3)Vno
D13 Ly Sb/LVM
D14 Ly Sb/In(3LR)$Ubx^{130}$
D15 Ly st/In(3LR)$Ubx^{130}$
D16 R Ly/In(3L)P, gm
D17 $ry^2$
D18 ru h th st cu sr $e^s$ ca ("rucuca")
D19 ru h th st $p^p$ cu sr $e^s$ ("rupes")
D20 Sb/In(3LR)$Ubx^{130}$
D21 st
D22 st c-3-G ca/In(3LR)$Ubx^{130}$
D23 st Sb/In(3LR)$Ubx^{130}$
D24 st ve
D25 th
D26 th st cp
D27 th st cp in ri $p^p$

### Chromosome 4

E 1 ci $ey^R$
E 2 ey
E 3 $spa^{pol}$

### Multichromosomal

F 1 $y^2$;bw(1;2)
F 2 $y^2$ cho;lys rc(1;2)
F 3 y;Gl Sb/TM2(1;3)
F 4 y;$spa^{pol}$(1;4)
F 5 y pn;C(4)RM, ci $ey^R$/O(1;4)
F 6 al $L^4$ Cy Sp/$bw^{V1}$;H/Sb sr In(3R)Me(2;3)
F 7 bw;e(2;3)
F 8 bw;st(2;3)
F 9 lys rc;ss(2;3)
F10 ("sifter O")S Sp $P^-$ T(2;3), Ins CXF/ $dp^{txI}$ Cy, Ins 05 pr $cn^2$;Dl H e $p^i$(2;3)
F11 Sp/Cy;Ly Sb/TM2(2;3)
F12 Sp/Cy;Gl Sb/TM2(2;3)
F13 Sp/Cy;$spa^{pol}$(2;4)
F14 "Basc" In(1)$sc^{S1L}$ $sc^{8R}$+S, $sc^{S1}$ $sc^8$ $w^a$ B;Sp/Cy;e(1;2;3)
F15 y;bw;st(1;2;3)
F16 y B;Sp/Cy;$spa^{pol}$(1;2;4)
F17 "Basc" In(1)$sq^{S1L}$ $sc^{8R}$+S, $sc^{S1}$ $sc^8$ $w^a$ B; SM1/T(2;3)S9 bw e/TM2;$spa^{pol}$(1;2;3;4)
F18 y;bw;st;$spa^{pol}$(1;2;3;4)
F19 y;SM1/T(2;3)S9 bw e/TM2;$spa^{pol}$(1;2;3;4)
F20 y/Y·$y^+$;SM1/T(2;3)S9 bw e/TM2;$spa^{pol}$ (1;2;3;4)
F21 y B;Sp/Cy;st ve;$spa^{pol}$(1;2;3;4)
F22 y B;Sp/Cy;st ca;$spa^{pol}$(1;2;3;4)
F23 y B/Y·$y^+$;SM1/T(2;3)S9 bw e/TM2;$spa^{pol}$ (1;2;3;4)
F24 y w;SM1/T(2;3)S9 bw e/TM2;$spa^{pol}$(1;2;3;4)

### Translocations

G 1 T(1;4)$B^S$(16 A1), $y^2$ cv v $B^S$ car/C(1)DX, f
G 2 T(1;4)$w^{m5}$
G 3 T(Y;2)A/lt

G 4 T(Y;2)B/b
G 5 T(Y;2)E/ab$^2$
G 6 T(Y;2)G/b pr tk
G 7 T(Y;2)J/px bw sp
G 8 T(Y;2)rl, lt cn/b lt bw
G 9 T(2;3)63-3
G10 T(2;3)63-18
G11 T(2;3)63-23
G12 T(2;3)64-33
G13 T(2;3)bw$^{VDe4}$/SM5, al$^2$ Cy lt$^v$ cn$^2$ sp$^2$
G14 T(2;3)A;Bl;ru h D TA ss e$^s$/In(3L+3R)P
G15 T(2;3)B;ru h D TB ss e$^s$/In(3L+3R)P
G16 T(2;3)dp
G17 T(2;4)a/In(2L+2R)Cy, Cy pr;ey$^2$
G18 T(2;4)ast$^v$/In(2L+2R)Cy, al$^2$ Cy lt$^3$ L$^4$ sp$^2$
G19 T(2;4)b/In(2L+2R)Cy, Cy pr;ey$^2$

## Inversions

H 1 In(1)65, y f/B$^S$ Y
H 2 In(1)dl-49 Hw m$^2$/y w$^a$ N$^{614-10}$
H 3 In(1)dl-49, In(1)B$^{Ml}$, y/Y$^{-bb}$ & y v bb/Y$^{-bb}$
H 4 In(1)dl-49, y fa$^n$
H 5 In(1)dl-49, y Hw m$^2$ g$^4$/Df(1)N$^8$
H 6 In(1)dl-49, v$^{Of}$ f
H 7 In(1)EN, y bb/sc$^8$.Y
H 8 In(1)l-v 231, y l(1)v 231/C(1)RM, y w/sc$^8$.Y
H 9 In(1)N$^{264-84}$, y/FM6, y$^{31d}$ sc$^8$ dm B
H10 In(1)rst$^3$, y rst$^3$ car bb
H11 In(1)sc$^4$,sc$^{8R}$, y$^{sc4+8}$ cv v f/C(1)DX, y f
H12 In(1)sc$^4$, y sc$^4$
H13 In(1)sc$^7$, In(1)AM, sc$^7$/In(1)dl-49, y Hw m$^2$ g$^4$
H14 In(1)sc$^8$, sc$^8$
H15 In(1)sc$^8$, y$^{31d}$ sc$^8$ w$^a$
H16 ("Basc")In(1)sc$^{S1L}$ sc$^{8R+S}$, sc$^{S1}$ sc$^8$ w$^a$ B
H17 ("Binscy")In(1)sc$^{S1L}$ sc$^{8R}$+dl-49, y sc$^{S1}$ sc$^8$ v B
H18 ("new Binsc")y sc$^{S1}$ B In 49 sc$^8$/C(1)DX, y f
H19 In(1)w$^{m4}$(bb?)
H20 In(2R)bw$^{VDe1}$, b/b lt l cn mi sp
H21 In(2R)bw$^{VDe2}$/In(2LR)Rev l
H22 In(2L)Cy, Cy dp$^{lv2}$ b pr/ds$^{38k}$
H23 In(2L)Cy$^L$ t$^R$, Su(S)dp$^{lv2}$ pr/ds$^W$
H24 In(2L)NS/J
H25 In(2L)t, lt l L$^4$ sp$^2$/bw$^{Vl}$ ds$^{33k}$
H26 l(2)a bs$^3$, In(2L)t/bw$^{Vl}$ ds$^{33k}$
H27 In(2L+2R)Cy, al$^2$ E(S)cn$^2$ sp$^2$ (does not carry Cy mutant)
H28 In(2L+2R)NS, px sp/s$^2$ Cy pr Bl cn$^2$ L$^4$ bw sp
H29 In(2LR)102, ds$^W$ sp$^2$/SM1, al$^2$ Cy cn$^2$ sp$^2$
H30 In(2LR)dp/Bl (see T(2;3)dp....G 16)
H31 In(2LR)Gla/b Go
H32 Bld, In(2R)Cy/mr$^2$
H33 In(2R)Mo$^K$/T(2;3)Ata, Ata
H34 In(3R)C, l(3)a/Bd$^G$

## Deficiencies and Duplications

I 1 Df(2R)bw$^5$, sp$^2$/T(2;3)ap$^{Xa}$
I 2 Df(2R)bw$^{VDe2L}$ Cy$^R$/In(2LR)Gla
I 3 Df(2R)MS2$^8$/SM1, al$^2$ Cy cn$^2$ sp$^2$
I 4 Df(2R)MS2$^{10}$/SM1, al$^2$ Cy cn$^2$ sp$^2$
I 5 Df(2R)MS4/SM1, al$^2$ Cy cn$^2$ sp$^2$
I 6 Df(2)rl$^{10a}$ lt cn/bw$^{Vl}$, ds$^{33k}$
I 7 Df(2R)vg$^c$/Rev$^B$
I 8 M(2)c$^{33a}$/al$^2$ InMis Cy cn$^2$ sp$^2$
I 9 M(2)H$^{S5}$/SM5, al$^2$ Cy lt$^v$ cn$^2$ sp$^2$
I10 M(2)e$^S$/Cy, bw$^{V34}$
I11 Dp(1;f)z$^9$, Df(1)sc$^{J4}$/C(1)DX, y f
I12 Dp(1;1)B$^S$ RMG, y w$^a$ B$^S$/In(1)sc$^{S1}$, In(1)dl-49, v
I13 px pd;Pdr H, Dp(2;3)P/Pdr

## X Chromosomes with a Y arm attached

L 1 Y$^S$ X.Y$^L$, In(1)EN, v f B/O;C(4)RM, ci ey$^R$/O
L 2 Y$^S$ X.Y$^L$ Inv(1)EN, Y$^S$ B f v y.Y$^L$ y$^+$/y v bb/O
L 3 Y$^S$ X.Y$^L$ Inv(1)EN, Y$^S$ v cv y.Y$^L$ y$^+$/y$^2$ su(w$^a$)w$^a$ bb/O
L 4 Y$^S$ X.(P-7), In(1)EN, Y$^S$ y f/C(1)RM, y v f/Y
L 5 Y$^S$ X.Y$^L$ Inv(1)EN, y$^+$ Y$^S$ y.Y$^L$ y$^+$/y$^2$ su(w$^a$)w$^a$ bb/O
L 6 X.Y$^L$(c-2), y cv v f car bb$^-$.Y$^L$/C(1)RA (ND-27)sc v f--In(1)sc$^8$, f v sc$^8$./Y"
L 7 Y$^S$ X.(FR1), Y$^S$ y cv v f./C(1)DX, y f/Y

## Attached XY Chromosomes

M 1 X Y$^S$.Y$^L$(110-8 Parker), y$^2$ su(w$^a$)w$^a$ Y$^S$.Y$^L$ y$^+$/C(1)RM, y v bb/O
M 2 XY$^S$.Y$^L$(129-16 Parker), y$^2$ su(w$^a$)w$^a$ Y$^S$.Y$^L$ y$^+$/C(1)RM, y v bb/O
M 3 Y$^S$ X.Y$^L$, In(1)EN+dl 49, Y$^S$ car f v y.Y$^L$/C(1)RM, y$^2$ su(w$^a$)w$^a$ bb/O

## Compound Chromosomes

N 1 C(1)DX, y f (see B2,B6,B',B14,B18,B19,B25, G1,H11,H18,I11 and L7)
N 2 C(1)RA(ND-27)sc v f--In(1)sc$^8$, f v sc$^8$. (see L6)
N 3 C(1)RM, y v f (see L4)
N 4 C(1)RM, y w (see H8)
N 5 C(2L)Pr, dp;C(2R)P4, dp
N 6 C(3L)RM #4, ri;C(3R)RM #4, sr
N 7 C(4)RM, ci ey$^R$ (see F5 and L1)

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

#### Wild Stocks

1 Canton-S
2 Canton-S-C (highly inbred)
3 Cockaponsett Forest, Conn.
4 IF-38, Idaho Falls, Idaho
5 NB-1, New Britain, 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)

#### Chromosome 1

14 B
15 bi
16 bi $ct^6$ $g^2$
17 car
18 $ct^6$
19 dor/ClB
20 dor/$FM_4$, $y^{31d}$ $sc^8$ dm B
21 fa
22 fu/ClB
23 fs(1)N/M-5
24 g/ClB
25 $g^{50e}$
26 $g^{50e}$/y f:=
27 $Hw^{49c}$/M-5
28 l(1)mys/M-5
29 $lz^{50e}$
30 na/FM3, $y^{31d}$ $sc^8$ dm B l
31 $pn^2$
32 sc ec v g f/ClB
33 sc ec cv $ct^6$ v g f/FM3, $y^{31d}$ $sc^8$ dm B l
34 sc ec v g f
35 $sc^{S1}$ B, In-S, $w^a$ $sc^8$(M-5)
36 $sn^3$/y f:=
37 $sn^3$ v
38 $sn^3$ v B
39 $sn^4$ oc $ptg^3$/+:=
40 v
41 w
42 w m f
43 w spl
44 $w^a$
45 $w^a$ v B
46 $w^{bf}$
47 $w^{bf}$/FM4, $y^{31d}$ $sc^8$ dm B
48 $w^{bl}$
49 $w^e$
50 $y^2$ sc $w^a$ ec/y f:=
51 $y^2$ v f
52 $y^2$ $w^a$ cv $sn^{55a}$ v/M-5
53 $y^2$ $w^a$ w/y f:=
54 $w^a$ $fw^{49c}$/y f:=

#### Chromosome 2

55 al
56 al b c $sp^2$
57 al dp b pr c px sp
58 b
59 b cn vg
60 b vg
61 $bs^2$
62 bw
63 bw $bs^{cy}$
64 cn
65 cn bw
66 cn bw Kr/Pm
67 dp
68 dp $bw^a$
69 $L^2$/Cy $sp^2$
70 $ltd^{37b}$
71 $ltd^{37b}$ vg
72 M(2)$l^2$/Sml, $al^2$ Cy $sp^2$
73 net al ex ds S ast/Sml, $al^2$ Cy $sp^2$
74 pr
75 rc
76 sca
77 vg
78 vg c
79 vg tu-bw

#### Chromosome 3

80 $Dfd^{r-l}$
81 e
82 $e^4$ wo ro
83 $e^{11}$
84 $e^s$
85 Gl Sb/LVM
86 Ly/$D^3$
87 Ly Sb/LVM
88 ru h st cu sr $e^s$ ca
89 ry
90 se
91 se e
92 ss
93 $ss^a$
94 st
95 $Ubx^{130}$ $e^s$/Xa

#### Chromosome 4

96 ci $ey^R$
97 $ey^2$
98 Scn/$ey^D$
99 $sv^{de}$/$ey^D$
100 $sv^n$
101 $spa^{Cat}$/$ci^D$

#### Multichromosomal

102 $ct^{45e}$ v;bw;e(1;2;3)
103 g;cn(1;2)
104 v;bw(1;2)
105 v;bw;e(1;2;3)
106 v;bw;e;$ey^2$(1;2;3;4)
107 $sc^{S1}$ B, In-S, $w^a$ $sc^8$;In SMl, $al^2$ Cy $sp^2$/ dp b Pm $ds^{33k}$;C Sb/$Ubx^{130}$ $e^s$ (1;2;3)
108 v;e(1;3)
109 w;e(1;3)
110 $w^a$ v;e(1;3)
111 $w^e$;cn(1;2)
112 y w;ant(1;2)
113 $y^2$ v f;bw(1;2)
114 bw;e(2;3)
115 bw;st(2;3)
116 cn bw;e(2;3)
117 cn;se(2;3)
118 dp;e(2;3)
119 Pm, dp b/Cy $sp^2$;Sb/CxF (ru h ca?)(2;3)
120 pr;$ey^2$(2;4)
121 e;$ey^2$(3;4)

#### Closed-X

122 $X^c$, y/y f:=
123 In($X^{c2}$)$w^{vc}$ f/dl-49, y w $lz^s$

#### Deficiencies

124 Df(1)$g^1$, f B/In(1)AM
125 Df(1)$N^8$/dl-49, y Hw $m^2$ $g^4$
126 Df(1)$N^{45e}$/dl-49, y Hw $m^2$ $g^4$
127 Df(1)$w^{258-11}$/dl-49, y Hw $m^2$ $g^4$
128 Df(1)$w^{258-42}$/dl-49, y Hw $m^2$ $g^4$
129 Df(1)$w^{258-45}$/dl-49, y Hw $m^2$ $g^4$
130 Df(1)y sc/M-5(Vogt)

#### Translocation

131 T(Y;2)C/pr cn

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

### Wild Stocks

1 Berlin wild B
2 Berlin wild K
3 Canton-S
4 Oregon-R

### Chromosome 1

101 B
102 car
103 cv
104 f
105 m
106 sc ec ct v g f
107 $su(s)^2$ $w^a$ cv t
108 v
109 w
110 $w^a$
111 $w^{bf}$
112 $w^e$
113 w $sn^3$
114 $w^{co}$ $sn^2$
115 $w^{ch}$ wy
116 wy
117 y ac sc pn
118 y cv v f
119 y w
120 z $w^{l1E4}$

### Chromosome 2

201 al dp b pr c px sp
202 b cn vg
203 bw
204 vg

### Chromosome 3

301 $bx^{34e}$
302 $Dfd^{r-L}$
303 $e^{l1}$
304 jv se
305 ri
306 ru h th st cu sr $e^s$ ca
307 st
308 Tu

### Chromosome 4

401 $ar/ey^D$
402 bt $ey^R$ $sv^n$
403 ci $ey^R$
404 ci gvl bt
405 $ey^2$

### Multichromosomal

501 $su(s)^2$ v;bw
502 bw;st
503 cn;ss
504 vg;e

### Inversions

601 In(1)Cl, sc $l(1)t^2$ v sl B/+ (=ClB)
602 In(1)dl-49, tyl $bb^1/C(1)RM$, y v f car
603 $In(1)sc^8$ + dl-49, $y^{Sl}$ v f B
604 $In(1)sc^8$ + dl-49, $y^{Sl}$ v f B;bw; e;$spa^{pol}$
605 $In(1)sc^{SlL}$ $sc^{8R}$ + S, $sc^{Sl}$ $sc^8$ $w^a$ B (=M-5)
606 $In(2L)Cy/L^2$
607 In(2LR)SM5, $al^2$ Cy $lt^v$ $cn^2$ $sp^2$/Bl $L^2$
608 $In(3L)D^3/Ly$
609 In(3LR)TM3, $y^+$ ri $p^p$ sep Sb $bx^{34e}$ $e^s$ Ser/$Me^{65d}$ h th
610 $In(1)sc^{SlL}$ $sc^{8R}$ + S, $sc^{Sl}$ $sc^8$ $w^a$ B; In(2LR)SMl, $al^2$ Cy $cn^2$ $sp^2/In(2LR)bw^{Vl}$, dp b $bw^{Vl}$ $ds^{33k}$;$In(3LR)Ubx^{130}$, $Ubx^{130}$ $e^s$/C Sb;$spa^{pol}$

### Attached-X and -XY

701 C(1)RM, y/+
702 C(1)RM, y f/+;bw;e;$spa^{pol}$
703 $Y^SX.Y^L$, In(1)EN + dl-49, $Y^S$ car f v $y.Y^L$/ C(1)RM, $y^2$ $su(w^a)w^a$ bb/O
704 $B^SY$ $y^+/y$ v;bw

### Selected Stocks

801 Berlin wild, DDT-resistant 1

## IOWA CITY, IOWA: UNIVERSITY OF IOWA
### Department of Zoology (Milkman)

cv-2
A variety of polygenic crossveinless stocks.

## OXFORD, ENGLAND: UNIVERSITY OF OXFORD
### Department of Biochemistry, Genetics Laboratory

### Wild Stocks

Oregon-R
Various Swedish and Jugoslav strains

### Chromosome 1

gr
w
$w^a$
w m f
w m f/ClB
M-5

### Chromosome 2

ab
bw
cn
cn bw
dp cn bw
Cy $L^4$/Pm
vg
1(2)Me

### Chromosome 3

e
$Ns/Antp^B$

### Chromosome 4

$ey^2$
$ey^{opt}$

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

**Wild Stocks**

1 Canton-S
2 Daekwanryung (Korea)
3 Damyang (Korea)
4 Keuksando-1 (Korea)
5 Keuksando-2 (Korea)
6 Kwangju-1 (Korea)
7 Oregon-B
8 Oregon-R-C
9 Oregon-S
10 Samarkand
11 Seoul-1 (Korea)
12 Seoul-2 (Korea)
13 Seoul-3 (Korea)
14 Seoul-4 (Korea)
15 Suwon (Korea)
16 Swedish-c
17 Yangdong (Korea)
18 Dangjin (Korea)
19 Wonju (Korea)
20 Ansung (Korea)

**Chromosome 1**

101 B
102 bo
103 br
104 $Bx^3$
105 cm
106 cc
107 fa
108 rg
109 sc cv v eq
110 sc cv v f
111 t
112 $t^2$ v f
113 v
114 w
5 $w^a$
116 $w^{bf2}$
117 $w^{ch}$
118 $w^{col}$
119 $w^e$ $bb^1$/ClB
120 y
121 y ac v
122 y sc $mf^2$
123 $y^2$ cv v f
124 Basc/y $sc^8$ y

**Chromosome 2**

201 a px sp
202 ab
203 al
204 al bc $sp^2$
207 bw
208 bw ba
209 Bl/Cy, $bw^{45a}$ $sp^2$ $or^{45a}$
210 c
211 c l
212 cn bw
213 Cy/Pm
214 ex
215 ho
216 L
217 $L^4$
219 pr
220 vg
221 wt

**Chromosome 3**

301 aah
302 bul
304 cu
305 gl
306 h
308 ra
309 ro
310 ru
311 se
312 ss
313 st

**Chromosome 4**

401 bt
403 ci gvl bt
404 ey

**Multichromosomal**

504 Cy/Pm;Sb/Ubx(2;3)
505 Cy/Pm;D/Bd(2;3)

**Inversions**

801 $Vg^{nw}$ Hia/SM5, $al^2$ Cy $lt^L$ Sp
802 $Vg^u$/Roi, bw sp or

**Translocations**

901 T(2;3)Xa/Sb $bx^D$

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

**Wild Stocks**

Canton-S
Umgazi River
Roma
Pavia
Bisignano
Sciolze

**Chromosome 1**

B
$Hw^{49c}$/FM1
$Hw^{49c}$ $f^5$/ClB
$Hw^{49c}$ $sn^3$/ClB
w
y f:= & $Hw^{49c}$ $sn^3$
$v^{36f}$
v
$su^{s2}$-v-pr v/FM3(bw)
vf $su^w$-f
"Basc"($sc^{s1}$ B Ins $w^a$ $sc^8$)
y Hw & RM, $sc^8$ B $w^a$ $sc^{s1}$:=
y Hw In 1 $B^{M1}$ & y f:=
y

**Chromosome 2**

bw

**Chromosome 3**

h
ry
$su^b$ pr/In(3R)C, e;(pr)
$su^2$-Hw bx bxd/TMi, Mc, ri(TM1)
$h^1$ gs th

**Multichromosomal**

bw;st
$bw^{75}$;st
vg;$e^{11}$
Cy 05/PM;Ubx/Sb

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

#### Wild Stocks

1 Canton-S
2 Chieti-v
3 Crkwenica
4 Gaino
5 Jaslo o.c.
6 Moltrasio
7 Oregon-R
8 Pavia
9 S. Maria
10 Sevelen
11 Suna
12 Urbana
13 Valdagno
14 Varese
15 Aspra
16 Ponza
17 Giannutri
18 S. Antioco

#### Chromosome 1

19 B
20 $N^{B-S}$
21 $ptg^2$
22 sc ec cut v g f
23 v
24 sd
25 $w^a$
26 $w^e$
27 y w
28 abw
29 wmf

#### Chromosome 2

30 b cn
31 sp a px
32 ab
33 b cn vg
34 blt
35 $blt^S$
36 bsp
37 bw ba
38 c wt px
39 cn
40 cn c wt px
41 dp cl b
42 ft
43 $ll^2$
44 net
45 S.o.
46 $so^2$ b cn
47 $So^C$
48 $sp^2$ $bs^2$
49 al bc sp

#### Chromosome 3

50 cp
51 $gl^3$
52 mwh
53 mwh se
54 mwh ri ss k $e^s$ ro
55 ru
56 ve
57 obt
58 th
59 tx
60 th tx
61 h
62 c
63 se cp e

#### Multichromosomal

64 $px^{43j}$ co;ru jv se st ca
65 w;vg
66 y;al bw sp

#### Inversions on X Chromosome

67 ClB/+
68 ClB $y/yg^4$
69 $l(1)7/dl$-49 y hw $m^2$ $g^4$
70 Muller-5
71 Muller-5/lozenge

#### Inversions on Chromosome 2

72 Cy sp/Pm
73 Cy E-S/S
74 Cy $cn^2$ bw sp/Gla In LR
75 Gla/spd gt-4

#### Inversions on Chromosome 3

76 H/Sb sr In(3R)Me
77 ltr/Sb sr In(3R)Me
78 Me ca/ru cu ca
79 ve h th C3 G Sb Ubx/ st C3 G ca
80 Florida In(3R)Payne
81 Cy $L^4$ sp/Pm;H/Sb sr In(3R)Me
82 y w;Cy$L^4$ sp/Pm;H/Sb sr In(3R)Me
83 y $sc^{S1}$ In-49 $sc^8$;bw st $p^p$
84 bsp/bsp;Sb Me/H

#### Deficiencies

85 Df(2)$Px^2$ Df(2)Px, bw sp/SM1, al Cy $sp^2$
86 Df(2)$bw^5$ Df(2)$bw^5$ $sp^2$/Xa
87 Minute(2)Bridges
88 M(2)33a/$al^2$ In Mis Cy $cn^2$ $sp^2$

#### Translocations

89 T(1;4)$B^S(16A_1)$, $y^2$ cv v $B^S$ car/ y f:=

#### Special Stocks

90 "sz e" $Y^{Lc}/X.Y^S$ & y v f.:;e
91 "szw" $Y^{Lc}/X^w.Y^S$
92 $y^2$ $su^{wa}$ $w^a$ b b/v f B.X.Y

#### Stocks selected for Tumor manifestation

93 tu Al
94 tu Bl
95 tu B3
96 tu C1
97 tu C2
98 tu C3
99 tu C4
100 tu C5
101 tu D
102 tu $So^C$
103 tu Aspra
104 Freckled/Curly
105 Frd/Cy L.
106 yw;Cy L/Frd;Sb Me/H
107 yw;Cy L/Pm(Frd); Sb Me/H
108 q 156 melanotic
109 e 144 melanotic
110 lm
111 lnd

## CLAYTON, QLD., AUSTRALIA: MONASH UNIVERSITY
### Department of Genetics and Psychology

#### Wild Stocks

Oregon K
Oregon R
Riverside
East African
Bermuda
Hikone
Canton S

#### Chromosome 1

B
w
y
w, y
t
mal
v

#### Chromosome 2

bw
cn
vg
cn bw
pd
b pr vg
Cy Bl L
pr
pym/cy
dy stw bw

#### Chromosome 3

$e^{11}$
e
se
ve
ry

## LIMBE, MALAWI: UNIVERSITY OF MALAWI
### Department of Biology, Genetics Section

**Wild Stocks**

São Paulo
Limbe

**Chromosome 1**

B
cv
$lz^{cl}$/Muller-5
m

sn
v
w
w cv sn
w:= and +

**Chromosome 2**

$bw^{60g}$
cn bw

dp
sca
vg

**Chromosome 3**

e
$Ly/D^3$
$ss^a$
ve

**Chromosome 4**

$ci^{57g}$
$ci^W$
$spa^{pol}$

**Multichromosomal**

Cy/Pm;D/Sb
y;bw;st
Sm5/Bla;Tm3/Sb

## SOUTH ORANGE, NEW JERSEY: SETON HALL UNIVERSITY
### Department of Biology

**Wild Stocks**

Oregon-R
Canton-S
Urbana-S
Swedish-c
3 South Orange Strains

**Chromosome 1**

B
Basc
f
$g^2$
m
$m^DB_2$ (homozygous)
$ras^2$
w

$w^a$
$w^e$
w B
w f
w m f
y
y cv v f
y B & y f:=

**Chromosome 2**

b
b vg
bw

**Chromosome 3**

e
ru h th st cu sr $e^s$ ca
st

## CLEVELAND, OHIO: CLEVELAND STATE UNIVERSITY
### Department of Biology

**Wild Stocks**

Oregon-R
Lausaunne-S

**Chromosome 1**

y
br
$pn^2$
w
$w^{sat}$
$w^{co}$
$w^{Bwx}$
Ax
fa
$fa^{no}$
ec
bo
cx
cm
$sn^3$
v
dy

s
f
$Bx^3$
car
$y^2$ $w^a$ $ct^6$
y cv v f
w m f
fa $fa^{no}$ sn
ec dx
cv f
f B
dor/ClB
$N^8$/y Hw In49 $m^2$ $g^4$
y w $sn^3$ B & y f:=
$sc ct^6$ car/y f:=
$sc^{51}$ B InS $w^a$ $sc^8$ ("Basc")

**Chromosome 2**

net
al
ho
dp

$J^{34e}$
$rk^4$
b
Coi
hk
Bl/esc
Alu
stw
ad
$L_2$
$L^2$
c
$bw^2b$
Pin
Hx
al b cv sp
al dp b pr
b cn bw
b vg
Cy/Pm
Cy/Pm; st
$dp^{02}$ $dp^{lvl}$ b/Cy Bl L
$ds^{38k}$/Dy(2L)$dp^2$ b pr

U/cg C
$Px^2$ bw sp/SM1 $al^2$ Cy $sp^2$
Bl $L^2$/Cy $dp^2$
b Go/Gla
$Pu^2$/SM1, $al^2$ Cy $sp^2$
Ruf/$ds^{38k}$ Pm

**Chromosome 3**

ve
se
eyg
app
th
st
cp
W
drb
$bx^{34e}$
Cbx
$e^{ll}$
cd
bar-3

rsd
ca
bv
ca K-pn
ve h th
se ss k $e^s$ ro
D/Gl
Gl Sb H/Payne
$Bd^g$/In3R C, 1(3)a
Mc/$ap^{Xa}$

**Chromosome 4**

$spa^{pol}$
bt
$ey^2$
ar/$ey^D$

**Multichromosomal**

cn;st
b;$e^{ll}$
bw;e

## HONOLULU, HAWAII: UNIVERSITY OF HAWAII
### Department of Genetics

Wild Stocks

1 Oregon-R
2 Canton-S

Chromosome 1

3 B
4 ec
5 m
6 sc cv v f
7 v
8 w
9 $w^a$
10 y
11 y w spl

Chromosome 2

12 dp cn bw
13 b
14 b vg
15 Bl/In(2L+2R)Cy, Cy $bw^{45a}$ $sp^2$ $or^{45a}$
16 $L^2$
17 vg

Chromosome 3

18 cu
19 $e^s$
20 gl
21 $p^p$ cu

Chromosome 4

22 ey

Others

23 bw;st
24 f BB/y f:-
25 70 2nd & 3rd chromosomal lethals from Korea

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

Wild Stocks

1 Canton-S
2 Oregon-R (iso)
3 Hikone

Chromosome 1

101 B
102 car
103 cm
104 Eag
105 f
106 $Hk^1$
107 $Iz^{50e}$
108 Muller-5
109 phenylfultyroless-1
110 $rb_3$
111 $sn^3$
112 $Sk^5$
113 v
114 w
115 $w^a$
116 $y_2$
117 $y^2$
118 $y^{34c}$
119 $y^{2s}$
120 $y^{v2}$
121 y w f
122 y w m f

Chromosome 2

201 b
202 b $gp_2$
203 b vg
204 bw
205 cl
206 cn
207 cn bw
208 cn Cy/Pm
209 cn fes(K)bw/Cy
210 cn vg bw
211 l(2)gl cn bw/Cy
212 $px_1$
213 $ry^1$
214 $ry^2$
215 vg

Chromosome 3

301 bar-3
302 $ca^2$
303 $e^{11}$
304 jv
305 ld
306 se
307 $ss^a$
318 st
309 $tx^{52j}$

Chromosome 4

401 $pol_1$
402 $sv^2$

Multichromosomal

1:2
501 v;bw
502 v;cn

1;3
503 y;$e^{11}$

2;3
504 albasp rucuca
505 b;se
506 bw;st ss
507 $cl^{57j}$;$ss^a$
508 cn;st
509 vg;$e^{11}$

2;3;4
510 bw;st;$sv^n$
511 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 TG-57 (multiple)(Korea)
705 WMB (multiple)(Japan)

(B) Amylase

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

## UPTON, NEW YORK: BROOKHAVEN NATIONAL LABORATORY

### Wild Stocks

W-1 Canton-S
W-2 Oregon-R

### X Chromosome

X-1 $pn^2$
X-2 w
X-3 y cv v f car
X-4 y v

### Chromosome 2

2-1 b vg
2-2 bw
2-3 dp

### Chromosome 3

3-1 e

### Chromosome 4

4-1 $spa^{Cat}/ci^D$

### Multichromosomal Stocks

X,3-1 C(1)RM, y f/Y;ca K-pn
2,3-1 bw;e
2,3-2 In(2LR)SM1, $al^2$ Cy $cn^2$ $sp^2$/In(2LR)$bw^{V1}$, dp b $bw^{V1}$ $ds^{33k}$;In(3R)C, Sb/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^s$

### Inverted Chromosomes

INX-1 In(1)FM6, $y^{31d}$ $sc^8$ $w^{FM6}$ $dm^+$ B
INX-2 In(1)$sc^{4L}$ $sc^{8R}$ + S, y $sc^4$ $sc^8$ $w^a$ B/C(1)RM, $y^2$ $su(w^a)w^a$ bb/$y^+Y$
INX-3 In(1)$sc^{8L}$ $sc^{S1R}$ + dl-49, $y^{31d}$ $sc^-$ v f B/y 1(1)$J1^{259}$ w m f/$y^+Y$
INX-4 In(1)$sc^{S1L}$ $sc^{8R}$ + S, $sc^{S1}$ $sc^8$ $w^a$ B

### Attached XY

XY-1 $Y^SX.Y^L$, In(1)EN, ptg oc $sn^5$/C(1)RM, sc $ct^n$ oc ptg.In(1)dl-49, car $sn^{X2}$ y

### Y Derivatives

Y-1 $y^+Y$/In(1)dl-49, y $sc^{S1}$ B v f/C(1)RM, y f

## NORWICH, ENGLAND: JOHN INNES INSTITUTE

### Wild Stocks

1 Bayfordbury
2 Hampton Hill
3 Oregon-K
4 Samarkand
5 Teddington

### Inbred Lines

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

### Chromosome 1

10 v
11 w
12 y w

### Chromosome 2

13 b pr

### Chromosome 3

14 b pr vg
15 bw
16 cn
17 dp b cn bw
18 vg
19 st

### Multichromosomal

20 Cy $L^4$/Pm;H/Sb
21 bw;e

### Inversions

22 Muller-5

## PÔRTO ALEGRE, BRAZIL: UNIVERSIDADE FEDERAL DO RIO GRANDE DO SUL
## Instituto de Biociências, Departamento de Genética

### Wild Stocks

Buenos Aires
Oregon
Leningrado
Brisbane
Eldorado (Rio Grande do Sul, Brazil)
Some inbred strains

### Chromosome 1

$pn^2$
w
$w^e$
$w^{bl}$
$w^h$
$ras^2$
f
sc cv v f
y v
y

### Chromosome 2

pr
ltd
cn
bw
pd
vg
L
st bw

### Chromosome 3

p
e
se
e se
p v

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

**Wild Stocks**

4 strains from N.S.W. and Victoria

**Chromosome 1**

In $rst^3$
w
$w^{bl}$
y

y w
sc ec cv $ct^6$ $vg^2$ f/ FM3, $y^{31d}$ $sc^8$ dm B 1

**Chromosome 2**

b j
net
vg
Cy/Pm

**Chromosome 3**

$e^{11}$

**Multichromosomal**

In(2L+2R)Cy, Cy $bw^{45a}$ $sp^2$ $or^{45a}$;In(3LR)$Ubx^{130}$, $Ubx^{130}$/T(2;3)$ap^{Xa}$
In(1)$sc^{S1L}$ $sc^{8R}$+S, $sc^{S1}$ $sc^8$ $w^a$ B;In(2L+2R)Cy, Cy $bw^{45a}$ $sp^2$ $or^{45a}$;In(3LR)$Ubx^{130}$, $Ubx^{130}$/ T(2:3)$ap^{Xa}$

## BRNO, CZECHOSLOVAKIA: J.E. PURKYNĚ UNIVERSITY
### Department of Genetics

**Wild Stocks**

1 Oregon K (inbred)
2 Hikone R (inbred)
3 Suchumi (inbred)
4 Moravec (inbred)
5 Krnov 65 (inbred)
6 Krnov 66 (inbred)
7 Moskva (inbred)

**Chromosome 1**

8 y
9 w
10 v
11 B
12 y v
13 Muller-5

**Chromosome 2**

14 dp
15 cn
16 bw
17 cn vg/Oregon K
18 cn vg/Suchumi
19 b cn vg
20 dp b cn bw
21 al b c sp
22 al dp b pr c px sp
23 Cy/Bl L
24 Cy/al b pr lt ltd cn a px pd bw

**Chromosome 3**

25 se
26 e
27 se e
28 ru cu ca
29 Gl Sb/LVM
30 Me Sb e/He
31 Gl/$Ubx^{130}$
32 Sb/$Ubx^{130}$

**Chromosome 4**

33 pol
34 $ey^2$

**Multichromosomal**

35 w;e
36 $w^a$;e
37 b;se
38 Cy L/Pm;H/Sb
39 Cy/Pm;Ly/$D^3$
40 Cy/Pm;D/Sb
41 Pm dp b/Cy $sp^2$;Sb/D Cx F
42 Sb Ubx/T(2:3)Xa
43 Cy/Pm;H/C Sb
44 y w;Cy $L^4$ sp/Pm;H/Sb sr, In(3R)Me

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

**Wild Stocks**

3 Samarkand
5 $+^3$

**Chromosome 1**

101 "Basc" (In(1)$sc^{S1L}$ $sc^{8R}$ + S, $sc^{S1}$ $sc^8$ $w^a$ B)
105 cm $ct^6$ $sn^3$
108 $f^{36a}$ car/y f
109 $Hw^{49c}$/FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
116 sc ec cv $ct^6$ v $g^2$ f/ FM3, $y^{31d}$ $sc^8$ dm B 1
142 y ac $sn^3$ v
144 y ac $sn^3$ sx $yb^2$ sv/ v $sc^{S1}$ B In-49 v $w^a$ $sc^8$
156 y sc m $f^5$
159 y $sn^3$
160 y w
170 FM7, In(1)$sc^8$, dl-49, $y^{31d}$ $sc^8$ $w^a$ $v^{0f}$ B
172 FM7-lz/$sc^{10-1}$

**Chromosome 2**

212 bw
215 cg c/U
232 vg

**Chromosome 3**

310 H/In(3)hp
314 mwh e
320 se h
324 $ss^a$
353 h H/h hp
354 st

**Chromosome 4**

408 ci $ey^R$
431 ci ey x ci ey/+

**Multichromosomal**

504 y ac;$esc^D$
508 y;mwh
523 b;Msc/+

**Triploid**

559 ec rb cv/FM6, $y^{31d}$ $sc^8$ dm B

## BOGOTÁ, COLOMBIA: UNIVERSIDAD DE LOS ANDES
### Instituto de Genética

| | | |
|---|---|---|
| Fusa XXI | Vermellón | Vestigial |
| Caracolicito X | Ebony | Dumpy |
| Orito (Putumayo) | Sepia | Cy L/Pm |
| Fusa XXV B | White | |

## BHAGALPUR, INDIA: BHAGALPUR UNIVERSITY
### Department of Zoology, Drosophila Laboratory

| Chromosome 1 | Chromosome 3 |
|---|---|
| White eye | Se Cu |
| | Se Vg |

## NAPLES, ITALY: UNIVERSITY OF NAPLES
### Institute of General Biology and Genetics & Institute of Zoology

| Wild Stocks | Muller-5 | b | cn vg $bw^2$ | se |
|---|---|---|---|---|
| | v | b cn | Cy/Bl $L^2$ | se h |
| Oregon-R | w | b cn bw | Cy/Pin | se Sb |
| | $w^a$ | b cn vg | | st |
| Chromosome 1 | w cv mal | b cn vg bw | Chromosome 3 | |
| | y | bw | | Multichromosomal |
| cv | | $bw^D$ | cd | |
| cv mal | Chromosome 2 | cl | Sb | cv mal;se |
| ct | | cn | Sb/In(3R)Na | w;vg |
| mal | $awu^2$ | | XR | |

## SAPPORO, JAPAN: HOKKAIDO UNIVERSITY
### Faculty of Science, Zoological Institute

| Wild Stocks | Chromosome 1 | Chromosome 2 |
|---|---|---|
| Canton S | y w m f | Cy |
| Sapporo, Hokkaido, Japan | w | v vg |
| Otoinepp, Hokkaido, Japan | | vg;se(2;3) |

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

See DIS 44 (1969): 19-20 and revision in DIS 46 (1971): 33.

## KENSINGTON, N.S.W., AUSTRALIA: THE UNIVERSITY OF NEW SOUTH WALES
### School of Wool and Pastoral Sciences

| Wild Stocks | Chromosome 1 | Chromosome 2 | Multichromosomal |
|---|---|---|---|
| Oregon R C | B | dp b j fr | w;vg;e |
| 7 Eastern Australian strains | y cv v f | | |

## SAN BERNARDINO, CALIFORNIA: CALIFORNIA STATE COLLEGE
### Natural Sciences Division

**Wild Stocks**

1 Oregon-R-C

**Chromosome 1**

2 br $w^e$ ec rb $t^4$/FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ B
3 f BB/y f:=
4 $g^2$
5 w

**Chromosome 2**

6 al b c $sp^2$
7 cn bw
8 $vg^D$/SM5, $al^2$ Cy $lt^V$ $sp^2$

**Chromosome 3**

9 Ly/$D^3$
10 Ly Sb/LVM
11 red
12 se h
13 st

**Chromosome 4**

14 $ci^D$/$ey^D$
15 $bw^{V1}$, $ds^{33k}$/In(2L+2R)Cy, Cy;H/In(3R)Mo, sr (2;3)
16 T(2;3)Hn T(2;3)Hn, Df(3L) Hn, Hn/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^s$

## ASSUIT, U.A.R.: UNIVERSITY OF ASSUIT
### Department of Genetics

| Wild Stocks | Chromosome 1 | Chromosome 2 | Chromosome 3 | Multichromosomal |
|---|---|---|---|---|
| ORK | w | L | st | dp;e |
| Nobareya | $w^a$ | bw | e | bw;st |
| Ghorayeb | B | vg | PP | M-5;Cy/L |
| Mattmar | y | Cy | | |
| | $Sc^{S1}$ B Fn S $w^a$ $sc^8$ | dp | | |
| | | Cy/L | | |

## LONDON, ENGLAND: St. BARTHOLOMEW'S HOSPITAL MEDICAL COLLEGE
### Zoology Department, Genetics Laboratory

**Wild Stocks**

(a) mass mated
Kaduna
Oregon-K

(b) inbred by brother-sister mating
Kaduna
Oregon-K

**Chromosome 1**

(balanced with Inscy)

| | | |
|---|---|---|
| dm | mgt | sld |
| fin | pun | slm |
| gt $w^a$ | $rst^2$ | ty $g^2$ |
| lf | sla | |

M-5

## UTICA, NEW YORK: MASONIC MEDICAL RESEARCH LABORATORY
### Aging Program

**Wild Stocks**

Oregon-R
Swedish-c

**Chromosome 1**

w

## MEXICO CITY, MEXICO: NATIONAL COMMISSION OF NUCLEAR ENERGY
### Genetics and Radiobiology Program

Same as DIS #46: 36 except: Delete a3, f3, f4, j2, j8, j17 and m3
Correct h8 to read: st c3G en In(3LR)$Ubx^{130}$ $Ubx^{130}$,$e^s$

PADOVA, ITALY: UNIVERSITÀ DEGLI STUDI DI PADOVA
Istituto di Biologia Animale

| Wild Stock | Chromosome 1 | Chromosome 2 | Chromosome 3 |
|---|---|---|---|
| 1 Varese | 2 sc ec ct v gt f | 8 b cn vg | 12 ru b ss $p^p$ st $e^s$ |
| | 3 v | 9 cn | 13 se |
| | 4 $w^a$ | 10 dp cl b | |
| | 5 $w^{bl}$ | 11 net | Inversion on 2 |
| | 6 $w^e$ | | |
| | 7 y w | | 14 Cy sp/Pm |

TÜBINGEN, GERMANY: UNIVERSITY OF TÜBINGEN
Department of Genetics

| Wild Stocks | Chromosome 1 | Chromosome 2 | Chromosome 3 |
|---|---|---|---|
| Oregon | Muller 5 | vg bw | L Cy/Pm |
| Wien 1965 | ClB/v ptg oc sn | | L/Cy |
| Wien 1966 | y v f | | |
| Ponza III | | | |

## MELANOGASTER - NEW MUTANTS

Report of D.E. Jeffery

The following chromosomal rearrangements were all X-ray induced. All involve rearrangements of the $hairy^+$ allele, and all exhibit position effect when in heterozygous condition with hairy.

In(3L)$h^{+38}$: Inversion (3L) $hairy^+$ Jeffery 67a9. Breakpoints 66D,80F.

T(2;3)$h^{+40}$: Translocation (2;3) $hairy^+$ Jeffery 67a12. In addition to the reciprocal exchange of the distal ends of 2L and 3L, 3L has section 66D-73C inverted. Breakpoints: 30B, 66D, 73C. New Order: 40-30C/66D-61; 80-73C/66E-73B/30B-21.

T(3;4)$h^{+44}$: Insertional translocation of 63A-66F of 3 into 101F of 4 Jeffery 67b8. Breakpoints 63A, 66F, 101F.

T(2;3)$h^{+47}$: double translocation between 2L and 3L, and 2L and 3R Jeffery 67b10. Breakpoints 23A,34C,66D,98F. New Order: 40-34C/98A-100; 80-66D/23A-21; 81-98F/34C-23A/66D-61.

Report of I.R. Franklin and W. Rumball

Mdh-$NADP^{0.90}$: NADP-dependent malic dehydrogenase, slow form

Mdh-$NADP^{1.0}$: NADP-dependent malic dehydrogenase, intermediate form

Mdh-$NADP^{1.1}$: NADP-dependent malic dehydrogenase, fast form 3-53.1 Three variants of a NADP-dependent malic dehydrogenase have been found in natural populations from New South Wales. Mdh-$NADP^{1.0}$ is the most common allele, with a frequency usually exceeding 95%. Variants were identified by polyacrylamide gel electrophoresis in a continuous 0.1M tris-borate buffer, pH 8.9. Each gel was stained in 50 ml of 0.2M tris-HCl buffer, pH 8.5 with 3.5 mg NADP, 12.5 mg nitro blue tetrazolium, 1.0 mg phenazine methosulphate and 20 mg sodium hydrogen malate. The locus was located, in test crosses to 'rucuca', between curled (3-50.0) and stripe (e.62.0). Eighty-six recombinants between these two loci were tested, and in sixty-four cases the Mdh-NADP alleles were associated with curled. The Mdh-NADP locus is therefore located at 3-53.1 ± 0.6.

Report of I.R. Franklin and G.K. Chew

To-1$^{F}$: Tetrazolium oxidase, fast form

To-1$^{S}$: Tetrazolium oxidase, slow form 3-33.3 Several unstained regions are apparent on gels stained for dehydrogenases with tetrazolium salts. One of these oxidases has been found to be polymorphic in several natural populations of D. melanogaster. The frequency of the rarer slow allele has been observed at frequencies between 5-10%. The multiply marked third chromosome stock 'rucuca' is homozygous for the slow allele. Heterozygotes are characterised by three bands. To-1 maps between hairy and Esterase-6, 2.6 centiMorgans from Est-6.

Report of M. Carfagna and I. Melon

awu$^{2}$: augenwulst Spontaneous in a wild stock. The eye's phenotype corresponds exactly to that of lost awu described by Volkart, 1959, DIS 33: 100. Expression variable, often asymmetrical. Complete penetrance in homozygote; incomplete, about 22%, in heterozygote, which always shows minor expression. Viability is good to excellent. The location is not exactly corresponding to 2-56.8 ± 1.4 as indicated by Volkart for awu. Extensive recombination studies gave the location 2-53.7 ± 0.7. It has not be studied cytologically. RK3.

Report of D.J. Fox and K. Madhavan

Hexokinase-3 map position reported as 2-79± in DIS 46: 42 should read 2-73±.

## DROSOPHILA SPECIES - STOCKS

### POUGHKEEPSIE, NEW YORK: MARIST COLLEGE
Department of Biology

D. pseudoobscura

Payson, Arizona (3 strains)
Pine Creek, Ariz. (3 strains)
Baker Butte, Ariz. (3 strains)
Flagstaff, Ariz. (1 strain)
Lake Mary, Ariz. (3 strains)
Grand Canyon, N.Rim, Ariz. (3 strains)
Prescott, Ariz. (4 strains)
Sierra Ancha Mtns., Ariz. (1 strain)
Portal, Ariz. (1 strain)
Crystal Lake, Calif. (3 strains)
Sequoia Nat. Pk., Calif. (3 strains)
Yosemite Nat. Pk., Calif. (3 strains)
Nederland, Colo. (1 strain)
Black Canyon, Colo. (1 strain)
Custer, S. Dakota (3 strains)
Logan, Utah (1 strain)

D. persimilis

Crystal Lake, Calif. (1 strain)
Sequoia Nat. Pk., Calif. (2 strains)
Yosemite Nat. Pk., Calif. (3 strains)

D. busckii

Princeton, N.J. (1 strain)

D. hydei

Poughkeepsie, N.Y. (1 strain)

D. robusta

Princeton, N.J. (1 strain)
Poughkeepsie, N.Y. (1 strain)

D. immigrans

Poughkeepsie, N.Y. (2 strains)

D. affinis

Poughkeepsie, N.Y. (2 strains)

D. melanogaster

Princeton, N.J. (1 strain)
Poughkeepsie, N.Y. (4 strains)

## GENEVA, SWITZERLAND: UNIVERSITY OF GENEVA
### Department of Genetics

D. hydei Chromosomes are numbered according to Berendes' map. The correlation with Spencer's linkage groups, and with chromosome elements, is as follows:

| Element | A | B | C | D | E | F |
|---|---|---|---|---|---|---|
| D. melanogaster chromosome | 1=X | 2 L | 2 R | 3 L | 3 R | 4 |
| D. hydei chromosome | | | | | | |
| Spencer 1949 | X | IV | III | V | II | VI |
| Berendes 1963 | 1=X | 3 | 5 | 4 | 2 | 6 |

Mutant names and symbols are the same as in D. melanogaster only where actual homology is well established. Different names were chosen deliberately in most other instances. In the case of eye color in particular, ommochrome-deficient mutants are listed as "red A, B, etc." according to their localization on chromosome elements A-F, different loci on the same element as "red A-1, -2, etc.", and similarly drosopterin-deficient mutants as "bn A, etc." Homology of the cinnabar mutant has been established, but it is not certain which of the present loci is cn.

Abbreviations: loc = localization; rec = received; ref = reference; syn = synonymous. (For descriptions of these mutants see Drosophila Species - New Mutants, this DIS).

### Wild Stocks

W-1 São Paulo 56
W-2 Leiden 65
W-3 Alicante 67
W-4 Madeira 68
W-5 Zürich 70

### Element A

A-1 C(1)RM, $w^{iv}y^{Lt}$/Y x $w^{sw}$/Y
A-2 $w^{ak}$
A-3 $w^{rz}$
A-4 $w^{hg}$
A-5 redA-$3_{Gr}$
A-6 bnA-1Ko bnA-$2_{Gr}$
A-7 $v^{55}$bnA-$2_{Gr}$
A-8 v Ax sc y m redA-$2_{Sp}$ bb
A-9 C(1)RM $w^{iv}y^{Lt}.y^{Lt}N^{691}w^{iv}$/Y x v sc sn/Y
A-10 v sc y m $f^{64}$redA-$2_{Sp}$ bb
A-11 sdx
A-12 $y^{tl}m^{tl}$redA-$2_{H}$
A-13 y m bnA-$1_{Sp}$

### Element B

B-1 blu ng nt
B-2 Smr/+
B-3 pci
B-4 kb/blu ng nt
B-5 lpl/blu ng nt

### Element C

C-1 $Ap^{68}$/+
C-2 Gk/+
C-3 Stw/Stw
C-4 Sf pb redC-$1_{Sp}$ sca/pb redC-$1_{Sp}$ sca
C-5 mt
C-6 tk redC-$1_{Sp}$ sca
C-7 bed pb redC-$1_{Sp}$ sca
C-8 vg pb redC-$1_{Sp}$ sca
C-9 C(1)RM, $w^{iv}y^{Lt}$;redC-$1_{Sp}$ bnC-1;Ex

### Element D

D-1 c-V se jv ic blr
D-2 c-V se jv ht redD-$1_{G1}$ ruw

### Element E

E-1 Bls/+
E-2 $Dl^{64}$/+;ruw
E-3 H/+
E-4 $H^{67}$/+
E-5 $e^{d}$bnE-$1_{G1}$
E-6 aa5
E-7 aa3 redE-$1_{Hy}$;nt;h;sca
E-8 $Sp^{2}Dl^{59}$Kf/Tp_2)Anp

### Element F

F-1 Ex/$Ci^{67}$
F-2 Ex/Ex
F-3 Ci/Ci;T(Y;2;3;5)Do

### Chromosome aberrations

T-1 $In(1)f^{3}$
T-2 $Tp(1)w^{m1}$
T-3 $In(1)w^{m2}/w^{iv}$ x $w^{iv}$
T-4 $In(1)w^{m2}$Hess
T-5 $In(1LR)w^{m3}$
T-6 In(2)Lew
T-7 $T(1;2)v^{t3}$/+ x $T(1;2)v^{t3}$
T-8 $T(1;2;4)H^{Jag}$/+
E-8 $Tp(2)Anp/Sp^{2}Dl^{59}$Kf
F-3 T(Y;2;3;5)Do;Ci/Ci
A-1 C(1)RM, $w^{iv}y^{Lt}$/Y x $w^{sw}$/Y
A-9 C(1)RM $w^{iv}y^{Lt}.y^{Lt}N^{691}w^{iv}$/Y x v sc sn/Y

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

**D. virilis**

Wild Stocks

1 Hikone (Japan)
2 Kaidema (Japan)
3 Kochi (Japan)
4 New York (USA)
5 Pasadena (USA)

Chromosome 1

7 $v^4$
8 $w^a$
9 y

Chromosome 2

10 eb

Chromosome 3

11 cn

Chromosome 4

12 cd

Chromosome 5

13 st $B^3$ pe
14 st es

Multichromosomal

15 ru;mt $w^e$ sb
16 v;es(1;5)

**D. simulans**

Wild Stocks

15 strains

Chromosome 1

16 v
17 y w

Chromosome 2

18 net

Chromosome 3

19 jv se
20 st se

Other species

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

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

simulans

wild

Chromosome 1

$f^2$
v
y w

Chromosome 2

b
bw
dh b pm(py sd?)
net
net b py sd pm
$py^2$
$py^2$ up
stw (from su-bb)
up

Chromosome 3

st $Dl^2$ pe/st pe
$H^h$ pe
jv st pe
rd
rd jv se
st pe
st Ubx pe/st pe

## HONOLULU, HAWAII: UNIVERSITY OF HAWAII
### Department of Genetics

**D. immigrans**

Hawaii, Hawaii
Oahu, Hawaii
Kwangju, Korea

**D. mercatorum mercatorum**

Bisexual

El Salvador
Manizales, Colombia

Oahu, Hawaii
Rochester, New York
sl w e
m
v pm vl
v d

Parthenogenetic

SO-1-Im
RSB-6-Im
OB-2-Im

RSB-7-Im
w 40A'-Im
RSS-18-Im
O-3-Im
29cId-Im
RS-3-Im
SFRSB-7-Im
S-1-Im
S-4-Im
S-6-Im
S-7-Im
S-11-Im

**D. paranaensis** Please write for current list of Hawaiian species.

## SAPPORA, JAPAN: HOKKAIDO UNIVERSITY
### Faculty of Science, Zoological Institute

D. virilis (2 strains) D. sordidula (1) D. albomicans (1) D. ezoana (1)

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

D. melanogaster
D. takahashii
D. nepalensis
D. suzukii
D. malerkotliana
D. jambulina
D. panjabiensis
D. immigrans

## CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGO
### Department of Biology

**D. americana**

1 Independence
2 Anderson

**D. texana**

3 New Orleans

**D. virilis**
**Wild Stocks**

4 Pasadena lethal-free
5 Texmelucan

**Chromosome 1**

6 $w^{50112}$

**Chromosome 2**

7 b bk dt
8 va

**Chromosome 3**

9 $gp^2$
10 S/+
11 sv t tb $gp^2$

**Chromosome 5**

13 $B^3$ pe
14 pe
15 ru
16 ru st mh
17 ru st mh pe
18 st es $pe^{Jap}$

**Multichromosomal**

19 b;cn;$B^3$ pe
20 b;tb $gp^2$;cd;pe
21 b;sv t tb $gp^2$;cd;pe
22 b;sv t tb $gp^2$;pe
23 cd;pe
24 cn;pe
25 $gp^2$;pe
26 $gp^2$ S/$gp^2$ +;ru st mh
27 pe;gl
28 "scute"(II);$pe^{m3}$
29 t;cd
30 $v^{48a}$;pe
31 v48a w;pe
32 $v^{48b}$;pe
33 $y^{40a}$;pe

## MISIMA, JAPAN: NATIONAL INSTITUTE OF GENETICS
### D. ananassae

**Wild Stocks**

Barro Collorado, Panama 69 (low elevation)
Turrialba, Costa Rica 101 (high elevation)
Hawaii
D-pp (Pago Pago, dark)
L-pp (Pago Pago, light)
IM-4 (Madras, India
L-Upolu (light)
F2 (Peng-Hu Is.)
F3 ( " )
F5 ( " )
F8 ( " )
Ph-5 (Malaybalay, Philippines)
Ph-15 ( " " )
D-Tonga
V-Truk

**Chromosome 1**

y (Hinton)
$w^{65}$
$w^{65}$ $y^{51}$
$w^{65}$ $f^{49}$ $y^{51}$
$w^{65}$ $sn^{65}$ $y^{51}$
kk
$w^{65}$ kk $y^{51}$
$w^{65}$ ty $y^{51}$
ty
$kk^c$
$w^{65}$ $kk^c$
od

**Chromosome 2**

$b^{65}$
$ba^{65}$
$bw^R$
ma (Hinton)
$se^T$
Arc $bw^R$
Arc $se^T$
b ma
b $se^T$
$bw^R$/$ba^{65}$
cd $ba^{65}$ - 12
cd $bw^R$
cd $se^T$ $ba^{65}$
j b
ma $ba^{65}$
$se^T$ $ba^{65}$
b $bn^{67}$
cd $bw^R$ $bn^{67}$/
pe
Dl
Dl pe
Dl ext
ext
ext se
cd Dl $bw^R$

**Chromosome 3**

px
$px^2$
ru
$ru^2$
$px^2$ ru
$sm^{66}$
$sm^{66}$ $px^2$
bri Rf
bri Rf px
bri ru
$M^{65}$ px
$M^{65}$ ru
bri $M^{65}$ ru
M-b
M-b pc
pc
M-c
M-c px
Snp M-c
Snp bri ru
Snp
M-d
M-d $ru^2$
ri M-d
ri
mot

**Chromosome 4**

$bb^{67}$
$bb^2$

**Multichromosomal**

f;cd (Hinton)
f;cd $se^T$
f $y^{66}$;cd $ba^{65}$
$w^{65}$ $f^{49}$;cd
$kk^c$;cd $bw^R$
$w^{65}$;px
$w^{65}$ $y^{51}$;M-d
$w^{65}$;$bb^{67}$
f;cd;px
b $se^T$;$px^2$
$bw^R$;bri
$bw^R$;ru
j b $se^T$;ru
ma;bri
$se^T$;ru
Dl;px
Dl $bw^R$;ru
cd $bw^R$;M-d

## 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
D. immigrans: Lexington, Kentucky

Some of these stocks are not continuously available since they are difficult to maintain under laboratory conditions; however, most can be field collected from March through October.

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

D. simulans

| Wild Stocks | Chromosome 2 | Chromosome 3 | Stocks selected for tumor manifestation |
|---|---|---|---|
| 1 Aspra | 4 net | 5 st | |
| 2 Serpentara | | | 6 tu Bl |
| 3 Giannutri | | | 7 tu Aspra |

## MEXICO CITY, MEXICO: NATIONAL COMMISSION OF NUCLEAR ENERGY
### Genetics and Radiobiology Program

| | | | | |
|---|---|---|---|---|
| 3 | D. virilis | 9 | D. pseudoobscura | (Mexico City) |
| 4 | D. hydei | 10 | D. immigrans | " " |
| 5 | D. ananassae | 13 | D. virilis | " " |
| 7 | D. neohydei | 12 | D. busckii | " " |
| 8 | D. simulans | 14 | D. funebris | " " |

## NORWICH, ENGLAND: JOHN INNES INSTITUTE

1 D. simulans

## UTICA, NEW YORK: MASONIC MEDICAL RESEARCH LABORATORY
### Aging Program

D. funebris D. pseudoobscura D. saltans D. virilis

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

All of our D. pseudoobscura lines, listed in DIS 43: 75 (1968) have been discarded.

## PADOVA, ITALY: UNIVERSITÀ DEGLI STUDI DI PADOVA
### Istituto di Biologia Animale

1) D. hydei 2) D. pseudoobscura 3) D. simulans

## CLEVELAND, OHIO: CLEVELAND STATE UNIVERSITY
### Department of Biology

D. virilis

| Wild Stocks | | Multichromosomal |
|---|---|---|
| | Brazil | |
| | Chile | |
| Argentina | Texmelucan | b;tb gp;cd;pe |

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

D. auraria

Race A (10 wild strains)
Race B (4)
Race C (10)

D. immigrans (2 wild strains)
D. lutea (2 wild strains)
D. pseuddobscura
D. suzukii (2 wild strains)
D. virilis (6 wild strains)

## LIMBE, MALAWI: UNIVERSITY OF MALAWI
### Department of Biology, Genetics Section

Twenty species of Drosophila are kept in stock.

## TÜBINGEN, GERMANY: UNIVERSITY OF TÜBINGEN
### Department of Genetics

D. subobscura

Küsnacht
Belgrad
Lipari 1
Ponza 2/3

Norwegen
Griechenland
$Ba/1^{8}$
Va/+
$Va/Ba^{210}$
ch cu

D. pseudoobscura

wild

D. ambigua

wild

## SANTIAGO, CHILE: UNIVERSIDAD DE CHILE
### Facultad de Medicina, Departamento de Genética

D. brncici: Colombia
D. buskii: Chile (La Serena)
D. camaronensis: Chile (Azapa)
D. funebris: Chile (La Serena, Valdivia, Tierra del Fuego y Punta Arena)
D. gasici: Chile (Arica), Colombia, Bolivia (Cochabamba)
D. gaucha: Brasil (M. Capoes, C. de Jordan and Taimbas), Argentina (Cordoba, San Luis), Bolivia
D. hydei: Chile (Camarones, Azapa, Copiapó, Antofagasta), Bolivia (Cochabamba).
D. immigrans: Chile (Valdivia, La Granja, Las Viscachas)
D. mercatorum: Chile (Arica, Azapa, Antofagasta), Bolivia (Cochabamba)
D. mesophragmatica: Bolivia (La Paz), Perú (Cuzco, Machu-Picchu)
D. pavani: Chile (Copiapó, Vallenar, La Serena, Curicó, Viña del Mar, Olmúe, Bellavista, Colbún, Los Queñes, Chillań, La Granja, Las Vizcachas, Algarrobo)
D. repleta: Chile (Arica, La Cisterna, Azapa)
D. simulans: Chile (Azapa)
D. viracochi: Perú (Machu-Picchu)
D. virilis: Chile (Santiago)

## PÔRTO ALEGRE, BRAZIL: UNIVERSIDADE FEDERAL DO RIO GRANDE DO SUL
### Instituto de Biociências, Departamento de Genética

D. willistoni

Wild strains from: Florida, Perú, Cuba, Guatamala, Equador, Brasil: Tracua, Serra do navio (Amapá), Manaus and Tabatinga (Amazonas), Pôrto Velho (Guaporé) Bélem (Pará), Maranguape (Ceará), Salvador (Bahia) Xingú (Mato Grosso) Brasilia, Chapadinha (Distrito Federal), Tijuca (Guanabara), Itatiaia and Angra dos Reis (Rio de Janeiro), Ilha das Cobras (Paraná), Iperoba, Tubarão and Florianópolis (Santa Catarina), São Pedro and Eldorado (Rio Grande do Sul)

| Chromosome 1 | Chromosome 2 | Chromosome 3 |
|---|---|---|
| $w^e$ y sn ru(Inv)/lethal | 207/Broad<br>Em ph | pink(Inv)/lethal |

D. paulistorum

Wild strains from: Brazil: Xingú (Mato Grosso), Maranguape (Ceará), Florianópolis (S.C.). F68 - H82

D. pavloskiana (12) Apoteri
D. equinoxialis Cuernavaca - Tefé
D. tropicalis Palmas (Goiás) 1975.1 Belém - Brasil
D. cubana Trinidad - S. Domingos fr. J.I. Townsend

D. nebulosa

Radiosensitive and radioresistant strains
Wild strains from Lima (Perú), Tingo Maria Perú), Posto Duque (Amazonas)

D. polymorpha several wild strains and select morphs
D. pseudoobscura PP10, AR/ST
D. stoni T-10
D. victoria GH-3
D. lebanonnensis Casteeli, Lebanon
D. Pattersoni Texas

## BOGOTA, COLOMBIA: UNIVERSIDAD DE LOS ANDES
### Instituto de Genética

D. paulistorum

Yaguaracaca A2
Yaguaracaca A1
Yaguaracaca B1
Yaguaracaca B2
Mitú 1A
Mitú 1B
Mitú 2B
Valparaiso 1
Valparaiso 2
Caripe 8 (Ven)
British Guiana
Gigante (Huíla)
Raposo 95 - a,b,c,d

Fusa
Angra 24 (Brasil)
Chocó
Belem 11 (Brasil)

D. Willistoni - Banano

Marco 2
Yaguaracaca A (Bajo)
Yaguaracaca B (Alto)
San Pablo (Brasil) Bosque
Recuerdo
Condoto
Valparaiso (Caquetá)

Mitú 2
Macarena
Sasaima
Manizalez
Mitú 1
Piojó

Equinoxialis

Mitú 2A
San Pablo (Bosque)
Mitú 2A
Mitú 1
Marco 1 (Brasil)
Mesas

Valparaiso
Turbo B2
Yaguaracaca A (Bajo)
Yaguaracaca B1
Macarena
Umaripunta bosque (Vaupes)

Tropicalis

Mitú 2
Valparaiso (Caquetá)
San Pablo (Bosque)
Macarena
Umaripunta Est. 2 (Vaupes)

## LEEDS, ENGLAND: UNIVERSITY OF LEEDS
### Department of Zoology

D. busckii
D. funebris
D. hydei
D. immigrans
D. littoralis
D. obscura
D. phalerata (several strains)
D. subobscura
D. transversa

## KALYANI, INDIA: KALYANI UNIVERSITY
### Department of Zoology, Genetics Laboratory

D. ananassae

1. wild
2. se px po fa

## BARCELONA, SPAIN: UNIVERSITY OF BARCELONA
### Department of Genetics, Faculty of Sciences

D. affinis - Nebraska (USA)
D. ambigua - Spanish stocks
D. bifasciata - Pavia (Italy)
D busckii - Spanish stocks
D. buzzati - Spanish stocks
D. cameraria - Spanish stocks
D. emarginata
D. funebris - Spanish stocks
D. hydei - Spanish stocks
D. immigrans - Spanish stocks
D. littoralis - Spanish stocks
D. mercatorum - Spanish stocks
D. obscura - Spanish stocks
D. phalerata - Spanish stocks
D. persimilis
D. pseudoobscura - Texas (USA)
D. repleta - Spanish stocks
D. simulans - Spanish stocks
D. subobscura - Spanish stocks mutant stocks
D. testacea - Spanish stocks
D. tolteca - Medellin (Colombia)
D. transversa - Spanish stocks
D. tristis - Snery (Switzerland)
D. victoria - Prescott (USA)
Megaselia scalaris - Spanish

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

Wild Stocks

(a) D. ananassae (7 strains)
(b) D. bipectinata (Calcutta)
(c) D. malerkotliana
(d) D. nasuta
(e) D. kikkawai
(f) D. raychaudhurii
(g) D. latifshahi
(h) D. seguyi

Mutants of D. ananassae

Chromosome 1

y
$w^a$
vs

Chromosome 2

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

Chromosome 3

px pc
stw pc
stw px
stw
px
pc

Unlocated mutants

dct
sp
ci
arch

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

D. simulans (2 strains)
D. tristis Zürich
D. obscura (23 strains)
D. bifasciata (9 strains)
D. subobscura (8 strains)
D. ambigua (3 strains)
D. hydei (5 strains)
D. phalerata (11 strains)
D. testacea (6 strains)
D. kunzei Interlaken, Switzerland
D. transversa (15 strains)
D. limbata (2 strains)
D. busckii (7 strains)
D. immigrans (1 strain from Finland & 1 from Zürich)
D. funebris (7 strains)
D. littoralis (11 strains)
D. subarctica (8 strains)
D. willistoni (1 strain from Galápagos)
Chymomyza costata (14 strains)
Scaptomyza pallida (1 strain)

Note: If not mentioned especially, stocks are collected from natural populations in Finland, Sweden and Norway.

BHAGALPUR, INDIA: BHAGALPUR UNIVERSITY
Department of Zoology, Drosophila Laboratory

D. ananassae

Bhagalpur population

$ST^2/ST^2$, $ST^3/ST^3$
AL/AL, $ST^3/ST^3$
$ST^2/ST^2$, DE/DE
AL/AL, DE/DE
New gene arrangement in IIL

Darjeeling population

$ST^2/ST^2$, $ST^3/ST^3$
AL/AL, $ST^3/ST^3$
$ST^2/ST^2$, DE/DE
AL/AL, DE/DE
New gene arrangement in IIL
Pericentric inversion between IIL & IIIL

## DROSOPHILA SPECIES - NEW MUTANTS

Report of F.M.A. van Breugel

$w^mCoY$: white-mottled Confluens Y Cytology: $Dp(1;Y)16B_2\text{-}17B_1$ NOR?;$Y^L$. Insertional duplication (probably including part of the X chromosomal nucleolus organizer region) by transfer of a small fragment of X into the Y chromosome. Derived from $w^{ml}$ and found as a single exceptional male from a cross: $Dp(1)Co^{Nt}$, $w^{hg}/w^{iv}$♀ x $w^{ml}$, y, m♂. The male was $y^+m^+$ and showed large-spotted type of mottling. Males carrying the duplication Y chromosome in addition to being mottled in the presence of a white-marked X chromosome, show a Confluens phenotype, a wing vein character caused by duplication of the $Notch^+$ locus. The $w^mCo$ duplication not infrequently may become transferred by some exchange mechanism, in the male, to the X chromosome. $w^mCo$ serves as a suitable marker of the Y chromosome. RK1.

$Xw^mCo$: white-mottled Confluens X Cytology: $Dp(1;1)16B_2\text{-}17B_1$NOR?;$X^R$. Product of X-Y exchange in $X^w/w^mCoY$ males. Insertional duplication of euchromatin of X, including $w^+$ and $N^+$, into the heterochromatic (=Right) arm of X. Insertion quite terminally in heterochromatin most likely neighboring the X chromosomal nucleolus organizer region. RK1-2.

C(1)RM $w^{hg}.w^{hg}$ (white-honing) Spontaneous as a single homozygous $w^{hg}$ female in a $w^{iv}$ marked attached-X stock of Gregg (1957). Rather dark white allele, amber to prunelike in color. $w^{hg}$ is also available in detached condition (X-rayed). RK1.

C(1)RM $In(1)N^{68}$, $w^{hg}.w^{hg}$ Inversion $Notch^{68}$ in one arm of the C(1)RM chromosome. Cytology: C(1)RM $In(1)\ 17A_{1,2}\text{-}18A_{1,2}$. X-ray induced. Females have $w^{hg}$ eye color and notched wings and often malformed eyes. The w locus is included in the inversion. $In(1)N^{68}$, $w^{hg}$ is also available in detached condition (X-rayed). RK1.

C(1)RM $Dp(1)Co^{Nt}$, $w^{hg}.w^{hg}$ Insertional duplication Confluens-Net in one arm of C(1)RM $w^{hg}.w^{hg}$, including the $N^+$ and $w^{hg}$ loci. Cytology: C(1)RM Dp(1)16A-18D; 20D. X-ray-induced. Females have a darker eye color than ordinary attached $w^{hg}$, indicating triplication of the $w^{hg}$ locus. The abnormal wing venation is caused by a triplication of the $N^+$ locus. $Dp(1)Co^{Nt}$, $w^{hg}$ is also available in detached condition (X-rayed). RK2(attached). RK3(detached).

$w^{rz}$ (white-roze) Arose spontaneously; found as two pink-eyed males in a $w^{m2}/w^{iv}$ stock containing supernumerary Y chromosomes. RK1.

(Supplementary information will be given in Genetica 41, 1970 and 42, 1971, in press).

## hydei

Report of H. Gloor

(Abbreviations: loc = localization; rec = received; ref = reference; syn = synonymous. Some of these have been reported previously. See also Geneva's hydei stock list, this issue.)

white alleles:

$w^{ak}$: white-abrikoos Hess 65e. syn: $w^{a}$. loc: A. X-ray induced as $w^{ak}/w^{iv}$. Eye color light orange, the same in both sexes. rec: 68b from Hess, Tübingen, Germany as stock $w^{a}$. ref: van Breugel 1970.

$w^{hg}$: white-honing van Breugel 68k. loc: A. Spontaneous in stock C(1)RM $w^{iv}$ $y^{Lt}$ as ♀ C(1)RM $w^{hg}$ $y^{Lt}$. Eye color dark brown-red. ref: van Breugel 1970.

$w^{iv}$: white-ivoor Clausen 23. syn: w. loc: A. Spontaneous. Eye color off-white, ivory. rec: 62b from Gregg, Oxford, Ohio as stock 56. ref: Clausen 1923, Spencer 1949, van Breugel 1970.

$w^{rz}$: white-roze van Breugel 68b. loc: A. Spontaneous as 2♂♂ in cross In(1LR)$w^{m2}/w^{iv}$/Y x $w^{iv}$/Y(Y). Eye color pink. ref: van Breugel 1970.

$w^{sw}$: white-sneeuw Holleman 67f. loc: A. Spontaneous as 1 ♂ in wild stock São Paulo. Eye color pure white. ref: van Breugel 1970.

red eye mutants

v: vermilion Clausen (?). loc: A. Eye color a brilliant scarlet. Homology with v of D. melanogaster tested through interspecific eye disk transplantation by G.W. Beadle. rec: 61k from Gregg, Oxford, Ohio as stock 59. ref: Clausen 1923, Spencer 1949, Gregg & Smucker 1965, van Breugel et al. 1968.

$v^{55}$: vermilion-55 Gloor 55c. loc: A. X-ray induced allele of v.

red $A\text{-}2_{Sp}$: red eye-2 of Spencer on element A Spencer. syn: $ch^{I}$: cherry-I. loc: A. Spontaneous. Eye color orange brown, darkens with age. rec: 61g from Gregg, Oxford, Ohio as stock 59. ref: Spencer 1949, Gregg & Smucker 1965.

red $A\text{-}2_{H}$: red eye-2 of Hess on element A Hess 63b. syn: $ch^{t}$: cherry-tomato. loc: A. X-ray induced as 1♂. Allelic and similar in phenotype with red A-2Sp. rec: 67m from Green, Davis, California as stock $y^{tl}$ $m^{tl}$ $ch^{to\text{-}1}$. ref: Hess & Green 1965.

red $A\text{-}3_{Gr}$: red eye-3 of Green on element A Green 63k. syn: $to^{3}$: tomato-3. loc: A. X-ray induced as 1 ♂. Eye color red orange, darkens with age to dull brown. rec: 64c from Hess, Tübingen, Germany as stock $to^{3}$. ref: Hess & Green 1965.

red $C\text{-}1_{Sp}$: red eye-1 of Spencer on element C Spencer 37. syn: $cn^{A62\ 37}$: cinnabar (?). Eye color a brilliant scarlet. rec: 62k from Gregg, Oxford, Ohio as stock 97a. ref: Spencer 1949, Gregg & Smucker 1965.

red $D\text{-}1_{Gl}$: red eye-1 of Gloor on element D Gloor. loc: D. Spontaneous. Eye color scarlet.

red $E\text{-}1_{Hy}$: red eye-1 of Hyde on element E Hyde 15. syn: st: scarlet. loc: E. Eye color bright scarlet, darkens with age. rec: 61i from Wheeler, Austin, Texas as stock st jv pb sca. ref: Hyde 1915, Spencer 1949, Gregg & Smucker 1965.

red E-$G1$: red eye-1 of Gloor on element E Gloor 68. loc: E. Allele of red E-1Hy, but much darker. From wild population on Madeira.

brown eye mutants

bn A-$1Sp$: brown eye-1 of Spencer on element A Spencer 37. syn: $gn^{w93\ 37}$: garnet. loc: A. Heterozygous wild ♀ collected in Wooster, Ohio. Eye color translucent purple brown, darkens with age. rec: 62g from Gregg, Oxford, Ohio as stock 62d. ref: Spencer 1949, Gregg & Smucker 1965.

bn A-$1Ko$: brown eye-1 of Kobel on element A Kobel 67c. loc: A. Spontaneous as several ♂♂. Allele of bn A-1Sp.

bn A-$2Gr$: brown eye-2 of Green on element A Green 63j. syn: pn: prune. loc: A. X-ray induced as 1 ♂. Eye color purplish dark brown. rec: 64a from Green, Davis, California as stock $\overline{XX}$ w Lt x pn. ref: Hess & Green 1965.

bn C-1: brown eye-1 on element C syn: $br^{A\ 135\ 38}$: brown (?) Spencer 38. loc: C. Eye color slightly darker than wild type, in combination with red C-1Sp bright orange. rec: 65g from Gregg, Oxford, Ohio as stock $\overline{XX}$ w Lt;or;Ex.

bn E-$1G1$: brown eye-1 of Gloor on element E Gloor 55. syn: peach. loc: E. X-ray induced. Eye color waxy orange brown. Allelic with eye color 52 of Gregg & Smucker 1965.

se: sepia Spencer 38. loc: D. From wild population at Gatlinburg, Tennessee. Eye color dark brown, changing to black with age; accumulation of sepiapterin. rec: from Gregg, Oxford, Ohio as stock 121a. ref: Spencer 1949, Gregg & Smucker 1965.

morphological mutants

aa: abnormal abdomen Gloor. loc: E. A frequent phenodeviant in wild populations and especially after X-irradiation, the abnormal abdomen character often has a hereditary component. Sternites and tergites incomplete, hairs correspondingly eliminated. Penetrance and expressivity highly variable, depending on environment and genetic background. Larval and pupal segmentation normal. aa3 and aa5 are stocks which differ in several properties (dominance, expressivity, penetrance), both containing an allelic major factor on element E. Ref: Kobel & van den Bosch 1970.

$Ap1^{68}$: Aeroplan-68 van Breugel 68. loc: C. Spontaneous as 1 ♀. Wings spread. Homozygous lethal.

Ax: Abruptex Kobel 66h. loc: A. 1 ♀ in $F_2$ of X-irradiated first instar larvae. Veins of $Ax/+$ shortened (at 18°C nearly wild type), acrostichals fewer with corresponding pigment pattern changed. Enhanced by H. Lethal in ♂; Durchbrenner are small, weak, sterile, and show extreme vein erosion.

bb: bobbed Spencer. loc: A. Frequently found in wild strains. Females show reduction in bristle size, abnormal abdominal sclerites, lowered fertility, late emergence, late sexual maturation after emergence. rec: 61g from Gregg, Oxford, Ohio as stock 59. ref: Spencer 1949.

bed: breed Gloor 60. loc: C. Spontaneous. Wings broad, long, curved downwards.

Bls: Blaas Gloor 63m. loc: E. X-ray induced. Homozygous lethal. Wings spread at right angle to body, often blistered; alula without marginal hairs; viability and fertility low.

blr: blister Spencer 38. loc: D. syn: bl. From wild population at Azusa. Blister in 2nd posterior cell, penetrance lower at 18°C. rec: from Gregg, Oxford, Ohio as stock 121a. ref: Spencer 1949.

ble: blue Gloor. loc: B. Spontaneous. Color blueish gray, especially of thorax.

br: broad Kobel 66g. loc: A. Spontaneous. Wings shortened, broad. Distance between crossveins equals length of posterior crossvein. Legs (femur, tibia) shortened.

c-V: crossveinless-V Clausen (?). loc: D. Posterior crossvein missing. rec: 611 from Gregg, Oxford, Ohio as stock 121a. ref: Spencer 1949.

Ci: Cubitus-interruptus Spencer 37. syn: $Gp^{W\ 90\ 37}$: Gap. loc: F. Found as 1 ♂ in Wooster, Ohio. Heterozygotes with gap in L-5 proximal to posterior crossvein, homozygotes without L-5 and with gap in L-4. rec: 611 from Gregg, Oxford, Ohio as stock 122b. ref: Spencer 1949.

$Ci^{67}$: Cubitus-interruptus-67 Kobel 67m. loc: F. Spontaneous as 1 ♂ Do; $Ci^{67}$. L-5 with gap proximal to posterior crossvein; enlarged lateral pigment spots on thorax. Homozygous lethal.

$Dl^{59}$: Delta-59 Gloor 59. syn: $Dl^{1}$. loc: E. Veins broadened into deltas at junction with margin. Weak allele. Suppressed by H. Homozygous lethal. ref: Gloor & Kobel 1966.

$Dl^{63}$: Delta-63 Gloor 63m. loc: E. Spontaneous. Veins thickened, especially at junction with margin and with crossveins, L-1,2 extremely broad. Not entirely suppressed by H. Allelic to $Dl^{59}$. Homozygous lethal.

$Dl^{64}$: Delta-64 Gloor 64d. loc: E. Spontaneous. Phenotype intermediate between $Dl^{59}$ and $Dl^{63}$. Enhancer of ruw. Homozygous lethal.

$e^{d}$: ebony-dunkel Gloor 55. loc: E. X-ray induced. Larval spiracle sheaths, puparium, body, veins and wing blade darker than wildtype; tarsal bristles black instead of golden brown. Heterozygote indistinguishable from wild type.

Ex: Extension Spencer 38. loc: F. From wild population at Azusa, California. Heterozygotes with enlarged lateral pigment spots on thorax. In homozygotes spotted thorax pattern obliterated by evenly distributed brown pigment; thorax humpy. rec: 65g from Gregg, Oxford, Ohio as stock $\overline{XX}$ w Lt;or;Ex. ref: Spencer 1949.

$f^{64}$: forked-64 Gloor 64c. loc: A. X-ray induced as single ♂. Bristles gnarled with ends forked; thorax color darker than wild. ref: Hess & Green 1965.

Gk: Gekneused Gloor. loc: C. X-ray induced. Anterior crossvein missing, L-1 in marginal cell squashed, marginal bristle pattern disarranged. Homozygous lethal.

H: Hairless Gloor. loc: E. Anterior dorsocentrals missing, sockets present. Frequently some of the acrostichal and abdominal hairs lost. Veins not shortened. Interaction with Ax, sca, Dl and other mutants. Homozygous lethal.

$H^{67}$: Hairless-67 Kobel 67. loc: E. Spontaneous as 1 ♀. Similar to H.

ht: hart Davids 61d. loc: D. Spontaneous as 1 ♂. Thorax forshortened, creased anteriorly. Campaniform sensillum of anterior crossvein transformed into bristle.

ic: incomplete Spencer 37. loc: D. From wild population at Azusa, California. L-2 and L-5 incomplete distally or gap in L-2; wings slightly warped. rec: 611 from Gregg, Oxford, Ohio as stock 121a. ref: Spencer 1949.

jv: javelin Spencer 37. loc: D. From wild population at Azusa, California. Bristles long, erect, not tapered, often hooked or broken. Thorax dark. rec: 61i from Wheeler, Austin Texas as stock 139 Spencer. ref: Spencer 1949.

kb: kabeuter Gloor. loc: B. Spontaneous. Wings spread, short, rounded, curved downwards. Posterior crossvein absent or interrupted; thorax short, groove between thorax and

scutellum incomplete; abdominal hairs bristly. All leg segments shortened. Development slow; sterile.

Kf: Kerbflugel Gloor. loc: B. X-ray induced. Wing margin notched, especially costal, 2nd and 3rd posterior cell. Expression variable. Homozygous lethal. ref: Gloor & Kobel 1966.

lpl: lethal-polyploid Gloor. loc: B. Spontaneous in wild stock Rabat, Morocco. High degree of polyploidy in the imaginal anlagen. Lethal at late larval stage. ref: Gloor 1951, Staiger & Gloor 1952, Gloor & Staiger 1954.

m: miniature Spencer. loc: A. Spontaneous as 1 ♂. Small, shortened dark wings, flies small. rec: 61l from Gregg, Oxford, Ohio as stock 59. ref: Spencer 1949.

$m^{tl}$: miniature-Tübingen-1 Green 63i. loc: A. X-ray induced as 1 ♂. Allele of m. rec: 67m from Green, Davis, California as stock $y^{tl}$ $m^{tl}$ $ch^{to-1}$. ref: Hess & Green 1965.

mt: mat van Breugel 66. loc: C. Bromouracil-induced. Eyes dim, bristles broken, wings somewhat smaller and slightly warped.

$N^{691}$: Notch-691 Frei 691. loc: A. X-ray induced. Wing notched, veins thickened, eyes often reduced. Homo- and hemizygous lethal.

ng: no-groove Kobel 67. loc: B. Spontaneous. Groove between thorax and scutellum imperfect, scutellum with nick, bristle pattern on thorax disarranged.

nt: netvleugel Baumgartner 49. syn: confluent. loc: B. Extra veins off posterior crossvein and in marginal cell, wing frequently blistered. Suppressed by Smr. rec: 55 from Mainx, Vienna, Austria as stock confluent. ref: Mainx 1949.

pb: pearly-body Spencer, syn: pearly (?), brown-thorax. loc: C. Thorax color brown-gray, spotted pigment pattern obscured. rec: 62k from Gregg, Oxford, Ohio as stock 97a.

pci: posterior-crossvein-interrupted Gloor 67. loc: B. Spontaneous. Posterior crossvein missing or interrupted, legs (femur, tibia) often short, thorax large with pigment pattern slightly abnormal.

ruw: ruw-oog Spencer (?). syn: r: rough. loc: D. Eyes rough, facets irregular. Enhanced by Dl. rec: 61i from Wheeler, Austin, Texas as stock 1878.3.

$sc^{1}$: scute-1 Spencer. loc: A. Spontaneous as 1 ♂ in a cherry stock. Removes anterior orbitals and posterior sternopleurals; effect on other bristle groups variable. rec: 61k from Gregg, Oxford, Ohio as stock 59. ref: Spencer 1949.

sca: scabrous Spencer. syn: roughest. loc: C. From Gatlinburg wild population. Eyes large, bulging, some facets very large, rounded and irregularly distributed. Extra acrostichal rows; most bristles subject to twinning. rec: 62k from Gregg, Oxford, Ohio as stock 97a.

sdx: spreadex Gloor 67d. syn: spread. loc: A. Spontaneous as 1 ♂. Wings spread at an angle of 90°; flies small. Allelic with spread Green 63 (DIS 40).

Sf: Spitzflügel Gloor 55h. loc: C. X-ray induced. Wings long, pointed. Homozygous lethal.

Smr: Small-rough-eye Green 63. loc: B. X-ray induced. Eyes slightly smaller and texture rough by irregularity of facets and corresponding hairs. Suppressor of nt. Homozygous lethal. rec: 64a from Green, Davis, California as stock R/+.

sn: singed Spencer. loc: A. Spontaneous as single ♀ sn/+. Short gnarled bristles and hairs. Reported as female sterile, but now fertile. rec: 61k from Gregg, Oxford, Ohio as stock 59. ref: Spencer 1949.

Sp$^2$: Spaltthorax-2 Gloor 55h. loc: E. X-ray induced. Thorax and scutellum cleft by longitudinal groove; in weaker expression only nicked scutellum and disturbed hair pattern on thorax. Homozygous lethal. ref: Gloor & Kobel 1966.

Stw: Straw Kobel 66. loc: C. Spontaneous as 1 ♀. Body color and wings pale yellow; spotted pigment pattern on thorax obscured; bristles black; larval spiracle sheaths pale. Homozygous viable.

tk: thick Kobel 66. loc: C. Spontaneous as several flies. Tarsal segments swollen, wings blistered.

vg: vestigial Spencer 38. loc: C. From Gatlinburg wild population. Wings variably scalloped and nicked. rec: 55 from Spurway, London, as stock vg. ref: Spencer 1949.

y: yellow Clausen. loc: A. Body, wings and bristles yellow; wildtype thoracic pigment pattern indistinct; larval mouth parts lighter. rec: 61k from Gregg, Oxford, Ohio as stock 59. ref: Spencer 1949.

y$^{Lt}$: yellow-Light Spencer 37. syn: Light. loc: A. From wild population at Azusa, California as 1 ♂. Dark thoracic spots reduced, in homozygotes reduction more pronounced; body color lighter, bristles black. Phenotype of males intermediate between homo- and heterozygotes. rec: 62d from Gregg, Oxford, Ohio as stock 56. ref: Spencer 1949, Hess & Green 1965.

y$^{t1}$: yellow-Tübingen-1 Hess 621. loc: A. X-ray induced as 1 ♂. Phenotypically like y. rec: 76m from Green, Davis, California as stock y$^{t1}$ m$^{t1}$ ch$^{to-1}$. ref: Hess & Green 1965.

chromosome aberrations

In(1)f$^3$: Inversion-forked-3 Green 631. In(1)8C;18B van Breugel. X-ray induced as 1 ♂. Allelic with f$^{64}$. Homozygous viable. rec: 64m from Hess, Tübingen, Germany as stock In(X)f$^2$. ref: Hess & Green 1965, van Breugel et al. 1968.

In(1LR)w$^{m3}$: Inversion-white-mottled-3 Green 64b. In(1LR)16D3-4-17A1-2;NOR$^D$. New order: h$^L$NOR? / 16D3-4 - 1Ah$^S$·h$^L$NOR / 17A1-20 van Breugel. X-ray induced as 1 ♀. Heterozygotes over white have small and large patches on reddish background. Homozygotes have facet-sized spots on yellow background. Males nearly wildtype, but mottling distinct. rec: 67m from Green, Davis, California as stock w$^{m3}$/w Lt. ref: Hess & Green 1965, van Breugel 1970.

Tp(1)w$^{m1}$: Transposition-white-mottled-1 Green 631. Tp(1h;1e)1$^M$;17B1-2. New order: 20-17B2 / h$^L$NOR / 17B1 - 1h$^S$·h$^L$ / Nor / h$^L$ van Breugel. X-ray induced as 1 ♀. w$^{m1}$/w$^{iv}$ has large wild sectors on orange brown background; homozygotes and males nearly wild type. rec: 67m from Green, Davis, California as stock w$^{m1}$/w Lt. ref: Hess & Green 1965, Mukherjee 1965, van Breugel 1970.

In(1)w$^{m2}$: Inversion-white-mottled-2 Green 631. In(1L)h$^S$ - 1A;16C-D1 + In(1LR)9C3;NOR$^P$. New order: h$^L$NORh$^L$(?) / 9C4 - 16CD1 / h$^S$·h$^L$ / 9C3 - A1h$^S$ / 16D1 - 20 van Breugel. X-ray induced as 1 ♀. w$^{m2}$/w$^{iv}$ has single dark facets on a yellowish background. Modified by extra Y chromosomes. Hemizygous lethal but viable with Y from stock w$^{m2H}$ (w$^{m2}$-Hess, derived from w$^{m2}$) in this condition darker mottling of the eyes. Homozygotes light yellowish. rec: 67m from Green, Davis, California as stock w$^{m2}$/w and 66 from Hess, Tübingen, Germany as w$^{m2}$/w$^{m2}$. ref: Hess & Green 1965, Mukherjee 1965, Gloor et al. 1967, van Breugel 1970.

In(2)Lew: Inversion-Lewontin Lewontin 55. In(2)23D;35B van Breugel. From wild population at Raleigh, North Carolina. Present in several wild populations. Phenotype wild. rec: 60a from Lewontin, Rochester, New York as stock In(2)Lew/+. ref: Warters 1944.

Tp(2)Anp: Transposition-Antennapedia Gloor 55h. Tp(2)22D1;26D4;34A1. New order: 21-D1 / 26D4 - 34A1 / 26D4 - 22D1 / 34A1 - 48 Berendes. X-ray induced as 1 ♂. Antenna frequently transformed into mesothoracic leg. Penetrance and expressivity variable, environment-dependent. Homozygous lethal. ref: Berendes 1962, Gloor & Kobel 1966, van Breugel et al. 1968.

T(1;2)$v^{t3}$: Translocation-vermilion-Tübingen-3 Green 63k. T(1;2)18C1;44C van Breugel. X-ray induced as 1 ♂. Allelic to v. Homozygous lethal. rec: 68a from Hess, Tübingen, Germany as stock T(X,2)$v^{t3}$. ref: Hess & Green 1965, van Breugel et al. 1968.

T(1;2;4)$H^{Jag}$: Translocation-Hairless-Jaguar Gloor 63m. T(1;2;4)1h;39D;76C. New order: 20-1· / 76C - 94; 21 - 39D / 1h; 77-76C / 39D - 48 van Breugel. X-ray induced as 1 ♀. Allelic to H. Homozygous lethal. ref: van Breugel et al. 1968.

T(Y;2;3;5)Do: Translocation-Doorn Gloor 61c. T(Y;2;3;5)Y;24A;26D5;70C2;97B1. New order: 21 - 24A / 97B1 - 122; 49 - 70C2 / 26D5 - 48; 95 - 97B1 / 24A - 26D5 / Y; Y·/ 70C van Breugel & Kobel. X-ray induced. Tarsus of first leg somewhat shortened; sensilla campaniformia on L-3 transformed into bristles. Penetrance variable, enhanced by ht. Heterozygous females obtained by crossing to C(1) are sterile. ref: Berendes 1962.

References: Berendes, H.D. 1962, Chromosoma (Berl.) 14: 195-206; Breugel, F.M.A.v. 1970, Genetica 41 (Nr 4); Breugel, F.M.A.v. et al. 1968, Genetica 39: 165-192; Clausen, R.E. 1923, Amer. Nat. 57: 52-58; Gloor, H. 1951, Rev. Suisse Zool. 54: 520-521; Gloor, H. & H. R. Kobel 1966, Rev. Suisse Zool. 73: 229-252; Gloor, H. & H. Staiger 1954, J. Hered. 45: 289-293; Gregg, T.G. & L.A. Smucker 1965, Genetics 52: 1023-1034; Hess, O. & M.M. Green, 1965, DIS 40: 37-39; Hyde, R.R. 1915, Amer. Nat. 49: 183-185; Kobel, H.R. & Bosch, J.J.v.d. 1970, Genetica 41: 119-140; Mainx, F. 1949, DIS 23; Spencer, W.P. 1949, Genetics, Paleontology, and Evolution, 23-44. Princeton Univ. Press; Staiger, H. & H. Gloor 1952, Chromosoma 5: 221-245; Warters, M. 1944, Univ. Texas Publ. 4445: 129-174.

## pseudoobscura

Report of S.E. Moyer and V.J. Merluzzi

$w^{ec}$: white-ecru. Spontaneous during synthesis of Ba/Δ Bl/L from Bl Sc pr(ST) and upt bx Ba gl/Δ; or L/or kindly provided by Mr. Boris Spassky from Rockefeller University. Allelic with w (Spassky) on X. Lighter than $w^{ec}$ and $w^{i}$ of melanogaster kindly provided by Dr. Irwin I. Oster, Bowling Green State University. Eyes light buff, light yellow or "green gold", depending on age. Testes colorless. Eye color comparisons for sex, $w^{ec}$ and the $F_1$ ♀ $w^{ec}$ x w♂ are as follows:

$$F_1♂ = w^{ec}♂ > w^{ec}♀ > F_1♀$$

Lancefield (1922 Genetics 7: 335) has described another multiple allele of this locus, white-eosin. Tests for pseudoallelism in more than 80,000 progeny from $w^{ec}$ w females has not resulted in crossing over; including 40,000 from $w^{ec}$ w; Ba/+;Bl/+ females designed to increase crossing-over on X (see Welshons and Nicoletti, 1963, DIS 38: 80).

## QUOTABILITY OF NOTES

For previous listings see DIS 38, 42, 43, 44, and 45

Angus, D. 35:71; 42:96-97; 42:112; 45:109; 45:153-155
Bryant, P.J. 46:94
David, J. 46:84
David, J. and J. Bouletreau-Merle 46:83
Doane, W.W. 45:189
Langjahr, S. 46:126
Mather, W.B. 27:101; 33:146-147; 33:147; 34:91-92; 37:104; 38:55; 40:66; 41:125-126; 41:126-128; 42:85; 43:96; 43:100-101; 43:101; 44:72; 44:89; 44:98; 45:74; 45:111; 45:156-157 46:80
Roberts, R. 46:158

Nickla, H.* Arizona State University, Tempe, Arizona. Riboflavin content in Malpighian tubes of D. melanogaster.

Larvae of many eye color mutants of D. melanogaster have less yellow pigment (YP) in their Malpighian tubes (MPT) than wild type larvae[1]. No mutants other than those influencing eye color have been found to alter the color of MPT[2]. Kikkawa[3] has suggested that the YP in MPT of D. melanogaster is 3-hydroxykynurenine, an intermediate in the synthesis of brown eye pigments[4], while Forrest and Mitchell[5] have implicated sepiapterin, an intermediate in the synthesis of red eye pigments[6]. However, YP in MPT of the American roach (Periplaneta americana), Tribolium confusum, and Ephestia kuhniella is mainly riboflavin[7,8,9]. In this communication, results are presented which suggest that the major component of YP in MPT of D. melanogaster is riboflavin.

The technique used for chromatographic separations followed that of Hadorn and Mitchell[10]. Ascending paper chromatography was carried out in the dark with n-propanol and 5% ammonia (2:1) as the solvent. Flies were reared on standard agar-cornmeal-brewer's yeast-molasses-sucrose-propionic acid medium.

For comparison with YP, sepiapterin was obtained from heads of sepia mutant flies by paper chromatography and was identified by its fluorescent color (yellow) and Rf value[10,11]. Following elution in 50% aqueous acetone[5], the visible portion of the absorption spectrum was determined (in 50% acetone) at pH values 1.8, 7.4, and 13.4. In alkaline solution, there is a maximum at 441nm; in neutral and acid solution, there is a maximum at 418nm. Riboflavin was also chromatographed and eluted in 50% aqueous acetone. At pH 6.1 the absorption maxima are 445nm and 375nm; 447nm and 375 nm at pH 8.9; and 450nm and 353nm at pH 12.0. Yellow pigment from MPT was separated chromatographically from abdomens of approximately 1,300 wild type (Urbana) female flies. These chromatograms, when viewed under ultraviolet light reveal a bright yellow spot with approximately the same Rf value as riboflavin and sepiapterin. Several mutants (light and clot), which have pale MPT upon visual examination, were chromatographed. In both cases there was a reduction in concentration of this yellow fluorescent spot indicating that it is responsible for the yellow color of MPT in wild type flies. Following elution, the visible portion of the absorption spectrum of YP was determined at pH values 7.6 and 13.3. The absorption spectra obtained at these pH values are similar, which suggests the absence of large amounts of sepiapterin. In addition, the portion of the spectrum from 430nm to 540 nm resembles that of riboflavin.

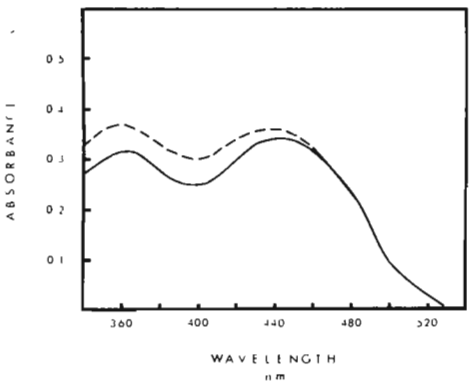



Figure 1. Absorption spectrum of riboflavin before (solid line) and after (broken line) treatment with bromine.

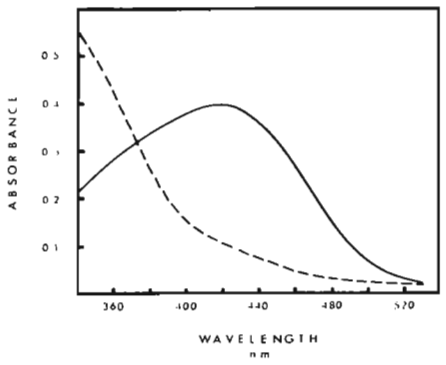



Figure 2. Absorption spectrum of sepiapterin before (solid line) and after (broken line) treatment with bromine.

Bromine causes immediate decolorization of sepiapterin producing two blue fluorescent compounds[5], without altering the characteristic yellow color of riboflavin[12]. Figures 1 and 2 present the absorption spectra of riboflavin and sepiapterin respectively before and after treatment with bromine (0.1ml of bromine/3ml of solution). Inspection of the sepiapterin

solution revealed a complete absence of yellow color after bromine treatment. Similar treatment of YP from MPT did not alter its absorption spectrum (Figure 3) and did not cause loss of color. The absence of the typical riboflavin spectrum (Figure 3) probably results from incomplete purification of YP as only single dimension chromatography was used.

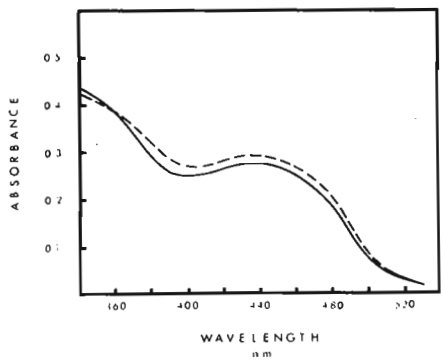



Figure 3. Absorption spectrum of the yellow pigment from the Malpighian tubes of wild type flies before (solid line) and after (broken line) treatment with bromine.

After bromine treatment, riboflavin, sepiapterin, and YP were chromatographed using two solvent systems and their Rf values were determined (Table 1). The fluorescent color was determined with an ultraviolet light. The absence of a blue fluorescent spot in bromine-treated YP strongly suggests that little, if any, sepiapterin is present in MPT. Since 3-hydroxykynurenine is an alpha amino acid, its presence can be determined by the ninhydrin test. Chromatograms (developed in n-propanol and 5% ammonia) containing kynurenine, sepiapterin, and YP from MPT were sprayed with a 0.2% (in acetone) solution of ninhydrin. Only kynurenine produced a positive ninhydrin reaction. Hadorn and Mitchell[10] also found that ninhydrin-positive materials are found only in trace amounts in chromatographed MPT.

Weber and Roberts[14] demonstrated that the primary site of riboflavin storage in Tribolium confusum is in the MPT. They concluded that the ability to store riboflavin in MPT is under control of a single gene whereas the amount stored is controlled by a small number of different genes. Sang[15] demonstrated that riboflavin is an essential dietary factor for normal development in D. melanogaster. The results presented in this communication support the hypothesis that riboflavin accumulates in MPT[13], and that the ability to absorb riboflavin from the diet and store it in MPT is intricately related to eye pigment metabolism in D. melanogaster. The nature of this relationship is under investigation.

Table 1. Mean Rf values of riboflavin, sepiapterin, and the yellow pigment from Malpighian tubes following treatment with bromine. Kynurenine not previously treated with bromine is also given.

| Compound | Visible Color | Fluorescent Color | Rf* A | Rf* B |
|---|---|---|---|---|
| Riboflavin | Yellow | Yellow | .34 | **.22** |
| Sepiapterin | Colorless | Blue | .16 | .17 |
| Yellow Pigment | Yellow | Yellow | .35 | .23 |
| Kynurenine | Yellow | Blue | .55 | .36 |

*Solvents: (A) n-propanol and 1% ammonia (2:1), (B) n-butanol, acetic acid, and water (4:1:1).

References: [1]Brehme, K.S., and Demerec, M., Growth 6:351 (1942); [2]Brehme, K.S., Proc Natl. Acad. Sci. U.S. 27:254 (1941); [3]Kikkawa, H., Adv. in Genetics 5:107 (1953); [4]Butenandt, A., Weidel, W., and Schlossberg, H., Z. Naturforsch., 4b:242 (1949); [5]Forrest, H.S., and Mitchell, H.K., J. Am. Chem. Soc. 76:5658 (1954); [6]Kaufman, S., Ann. Rev. Biochem. 1:171 (1967); [7]Metcalf, R.L., and Patton, R.L., J. Cell. Compar. Physiol. 19:373 (1942); [8]Weber, J. and Roberts, C.W., Canad. J. Genet. Cytol. 8:796 (1966); [9]Caspari, E., and Blomstrand, I. Genetics 43:679 (1958); [10]Hadorn, E., and Mitchell, H.K., Proc. Natl. Acad. Sci. U.S. 37:650 (1951); [11]Gregg, T.G., and Smucker, L.A., Genetics 52:1023 (1965); [12]Wagner-Jauregg, T., in The Vitamins (edit. by Sebrell, W.H., and Harris, R.S.), 299 (Academic Press, New York, 1954); [13]Ziegler, I., Adv. in Genetics, 10:349 (1961); [14]Weber, J., and Roberts, C.W., Canad. J. Genet. Cytol. 9:302 (1967); [15]Sang, J.H., Exp. Biol. 33:45 (1955).

I thank Drs. J.R. Cronin and J.T. Justus for their advice and interest in this work. This investigation was supported by National Institutes of Health.

*Present address: Department of Biology, Creighton University, Omaha, Nebraska 68131.

Kuroda, Y. National Institute of Genetics, Misima, Japan. Fibroblastic cells derived from pupal ovary of D. melanogaster in culture.

Ovaries obtained from 48-hour pupae of the Oregon-R strain of D. melanogaster, which were reared under sterile conditions, were cut into several fragments and cultured at 28°C in T-5 flasks with 0.8 ml of Kuroda's Drosophila medium K-6' (1,2), which was slightly modified and supplemented with 0.1 mg/ml fetuin, 5 mg/ml peptone and 15% fetal bovine serum.

After several hours of cultivation, many fibroblastic cells came out from the cut end of ovarian fragments explanted, and stretched their cytoplasm on the surface of culture flasks. They increased gradually in number in further cultivation and formed a network around the original explants after 24 hours of cultivation (Fig. 1). Mitotic figures were frequently observed. Under a phase microscope some small granules were observed in the cytoplasm and one or two nucleoli were found in the nucleus. From their morphology and behavior, these fibroblastic cells seem to be derived from the lumen cells in the ovarian cavity.

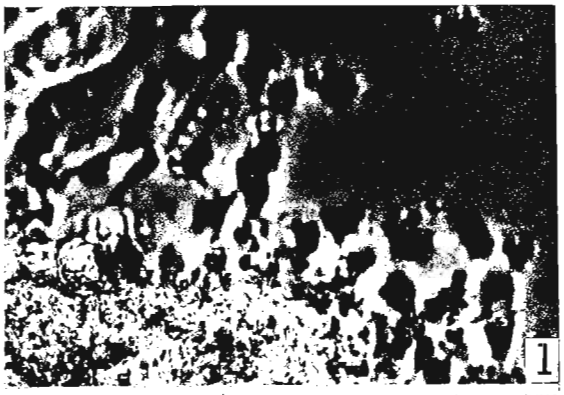



Fig. 1. Fibroblastic cells derived from a pupal ovarian fragment. After 24 hours of cultivation. Phase. x 540.

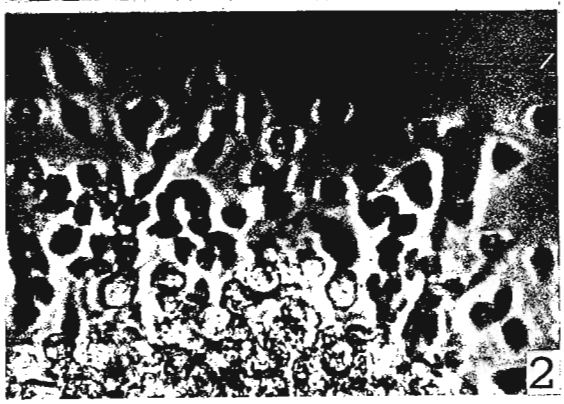



Fig. 2. Fibroblastic cells derived from an embryonic fragment. After 24 hours of cultivation. Phase. x 540

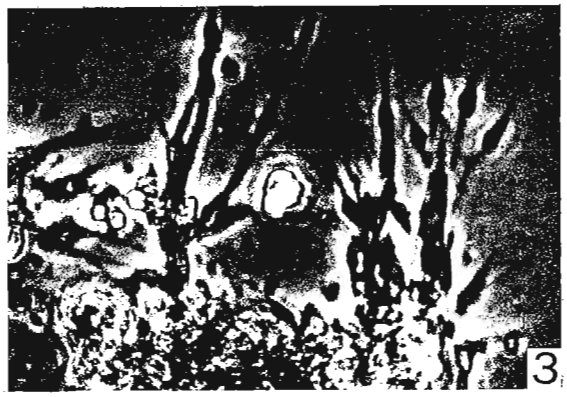



Fig. 3. Slender spindle-shaped cells derived from another embryonic fragment. After 24 hours of cultivation. Phase. x 540.

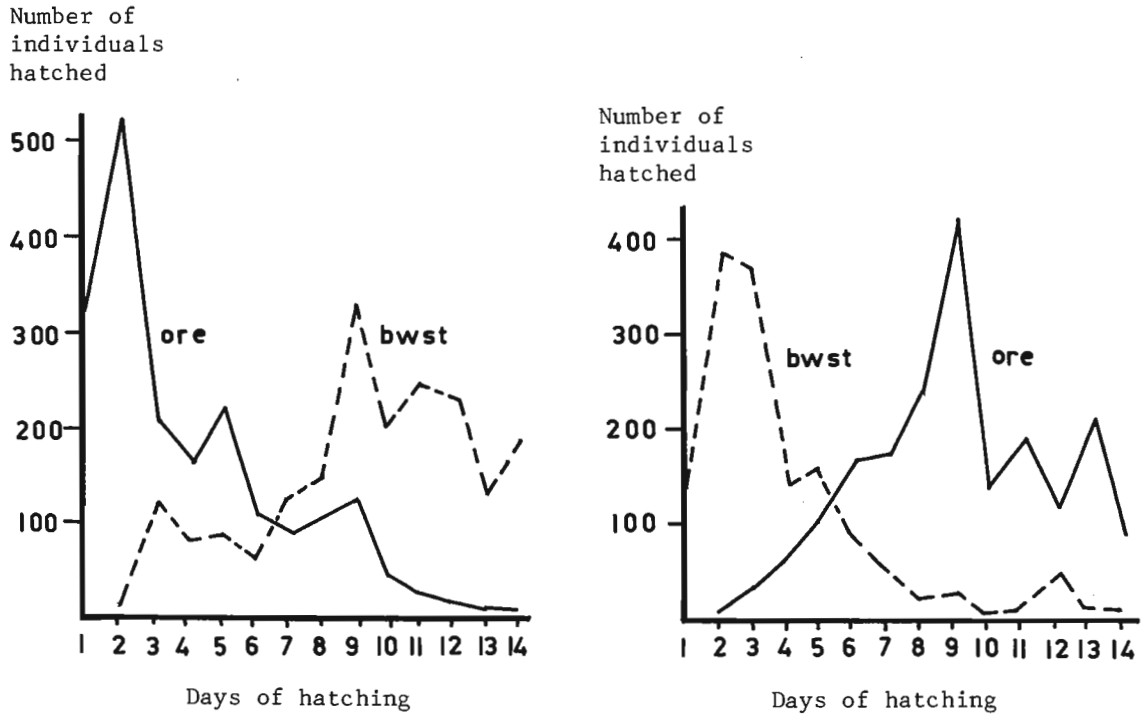
Of interest is the finding that the fibroblastic cells derived from ovarian fragments were very similar in morphology to one type of cells derived from embryonic fragments (Fig. 2). Among several types of cells derived from fragments of 12-hour embryos two types of cells were predominantly observed under the culture conditions employed: the fibroblastic cells and more slender spindle-shaped cells (Fig. 3).

Some insect cell lines established by Grace (3,4) were derived from ovarian tissues of Antheraea eucalipti and Bombyx mori. Although it is uncertain that Grace's cell lines originated from the same type of cells in the ovary as the fibroblastic cells observed in the present study, these fibroblastic cells may have some advantage in growing under in vitro culture conditions.

References: 1. Kuroda, Y., 1969 Japan. J. Genetics 44 Suppl. 1: 42; 2. Kuroda, Y., 1970 Exp. Cell Res. 59: 429; 3. Grace, T.D.C., 1962 Nature 195: 788; 4. Grace, T.D.C., 1966 Nature 216: 613.

Chita, O. Institut für allgemeine Biologie, Vienna, Austria and University of Craiova, Rumania. Larval-larval or larval-female interaction in D. melanogaster.

In order to find out the effect of earlier egg laying in a culture bottle with abundance of food a small experiment was carried out as described underneath. For this purpose 100 fertilized normal females (Oregon-strain) five days old (kept during this period with males) were then transferred to a vial containing normal corn meal food and allowed to lay eggs for 24 hours. These females were then discarded and replaced by 50 fertilized females and 50 males of the mutant strain bw,st. The mutant flies were allowed to lay eggs for 10 days continuously and then removed before the first flies hatched. In another set of experiments the same procedure was followed with the exception that at first 100 mutant bw,st females were used and then replaced by normal Oregon females and males. The bottles were repeatedly seeded with yeast solution so that the food supply was abundant. Fourteen days after the first females were put into the vials, the offspring started to hatch. From that moment the hatching flies were counted daily for a period of two weeks. In total, the offspring from 14 vials, seven for each experiment, were counted. The results are shown in Figure 1. The diagram on the left corresponds to the cultures with Oregon ♀♀ first, the right to those with bw,st ♀♀ first. It can be clearly seen that whichever genotype was used first has its peak for hatching flies on the second day with steady reduction on the days following. The genotype which had a delay of a day in egglaying, however, has its peak on the 9th day.




From the data presented it can be concluded that in competition the genotype of the eggs laid first is at an advantage. The period between the hatching peaks for the two groups is much longer than the period of delay in egglaying. This finding can be interpreted in two ways. Either there is an interaction between larvae in the sense that the older larvae can suppress the development of the younger ones, or females which are ready to lay eggs do not do so if there are already eggs deposited on the food surface. Egg laying, however, cannot be hindered by the females longer than a certain period. The hatching behaviour of the strains used, irrespective of the strain allowed to lay eggs first, is practically identical.

Wong, P.T., W.D. Kaplan, and W.E. Trout III.
City of Hope National Medical Center, Duarte, California. Alteration of response to a visual stimulus by a cholinesterase inhibitor.

The behavior mutant, $Hk^1$ (hyperkinetic), discovered in our laboratory, jumps and falls over when presented with a light stimulus in the form of a burst of strobe flashes. The sensitivity of these flies to such a stimulus depends on the intensity and frequency of the strobe light used. We are currently investigating the effect of various cholinesterase inhibitors on this response. The present communication provides data on the effect of eserine.

Flies were starved overnight and fed for three hours on eserine dissolved in a one % sucrose solution. Control flies were fed sucrose solution only. The mean % response of $Hk^1$ flies fed different concentrations of eserine and tested at three different light intensities is shown in figure 1. Each point on the graph represents a sample of ten flies

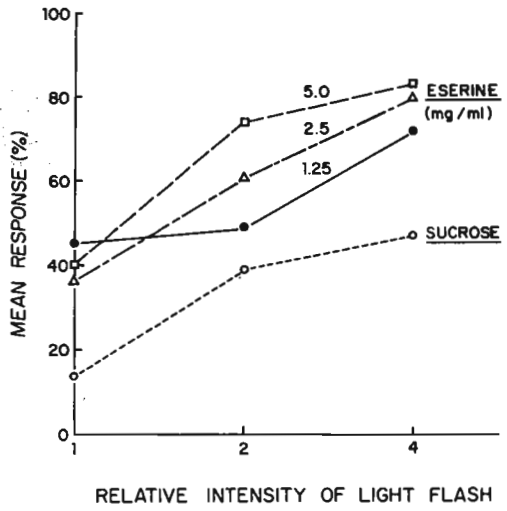



Figure 1

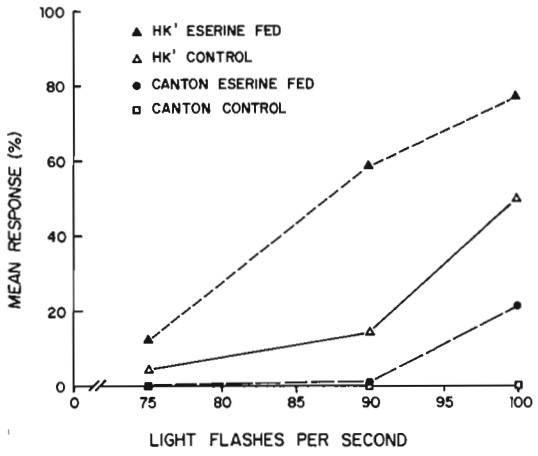



Figure 2

and each fly was tested 30 times. Each stimulus consisted of a burst of 25 light flashes at a frequency of 100 flashes per second. The intensity of the strobe light corresponding to a relative scale of four on the graph is approximately 350,000 candlepower. The eserine fed flies showed an increase in sensitivity to the light stimulus; at the two higher intensities, the mean % response appears to be directly proportional to the concentration of eserine. The increase in sensitivity of eserine-fed $Hk^1$ flies is also evident when the mean response is measured against frequency of the strobe flashes (figure 2); the strobe was set to give 25 flashes at a relative intensity scale of four.

Even Canton-S (wild type) flies, which do not normally respond to the light stimulus, can be made sensitive by feeding eserine. The type of response appears to be identical to that of $Hk^1$, so in this sense eserine produces $Hk^1$ phenocopies. However, eserine fed flies do not shake their legs while etherized, as does $Hk^1$. Thus the change in behavior may be primarily an increased sensitivity of the sensory system rather than the motor system. Dewhurst et al (1970) have suggested that acetylcholine may be a sensory transmitter in Drosophila. Therefore eserine, by blocking the degradation of acetylcholine, may increase the sensitivity of the sensory system which when stimulated sufficiently produces a startle response.

Reference: Dewhurst, S.A., McCaman, R.E., and Kaplan, W.D. Biochemical Genetics 4: 499-508 (1970).

Supported by NIH grant No. NS08014.

Thompson, S.R. and J.E. Putnam. Ithaca College, Ithaca, New York. Alteration of the rudimentary wing phenotype with Minutes and temperature.

Besides female sterility, the syndrome of abnormalities associated with rudimentary mutants includes modification of the size and morphology of the wing. The effects of second and third chromosomal Minutes in combination with $r^{39k}$ and various temperatures on wing development in $r^{39k}$ was studied. Table 1 lists the Minute allele, the arbitrary rating of the Minute, and the effect of the Minute upon the rating of $r^{39k}$ wings. Rudimentary wing phenotypes were arbitrarily rated on a scale of 3, a wing having an extreme defect was given a rating of 1, a moderate (intermediate) defect a rating of 2, and a normal wing a rating of 3. The Minutes were classified on a scale of 4, with 4 indicating extremely reduced bristle size and 0 indicating normal bristle size. In general, it seems that the stronger the Minute the more nearly normal the rudimentary wings appear. However, the Minutes appeared to have no modifying effect on the sterility associated with homozygous rudimentary females.

Table 1. Minute Interactions with $r^{39k}$.

| Minute Allele | Mean Minute Rating | Mean $r^{39k}$ Rating |
|---|---|---|
| Control (no Minute) | ---- | 1.75 |
| M(2)1$^2$ | 3.75 | 3.00 |
| M(2)S7 | 3.25 | 1.80 |
| M(2)S6 | 3.25 | 2.00 |
| M(2)B | 3.00 | 2.00 |
| M(2)173 | 3.00 | 2.50 |
| M(2)S5 | 2.75 | 2.50 |
| M(2)S3 | 2.50 | 1.75 |
| M(2)S4 | 2.25 | 1.75 |
| M(2)S10 | 1.75 | 1.75 |
| M(2)S11 | 1.00 | 1.80 |
| M(2)S9 | 1.00 | 1.75 |
| M(2)S8 | 0.50 | 1.75 |
| M(3)w | 4.00 | 3.00 |
| M(3)B$^2$ | 3.50 | 2.00 |
| M(3)1 | 3.00 | 1.75 |
| M(3)S31 | 2.50 | 2.00 |
| M(3)y | 2.00 | 2.00 |
| M(3)S34 | 1.75 | 2.50 |
| M(3)36e | 1.25 | 2.00 |
| M(3)S36 | 1.00 | 1.75 |
| M(3)B | 0.00 | 1.75 |

Since Schultz (1929) demonstrated that Minutes delay the length of time required for normal development, rudimentary wing development was studied at different temperatures, which would alter the developmental rate. White prepupae were collected, placed in shell vials and allowed to undergo metamorphosis in constant temperature water baths at three different temperatures (18°, 23°, and 28° C.). Table 2 lists the effect of these temperatures on pupal development time and on wing size in $r^{39k}$.

Because the slower development, produced by the lower temperature (18° C), yielded wing sizes which were more normal than controls (23° C), and approximated those found with strong Minutes, it seems likely that the Minute effect was due to the slowing down of development. Because the two extreme temperatures, 18° and 23° C, have no overlap in mean wing size it was possible to use this difference to determine the

Table 2. Effect of temperature on $r^{39k}$.

| Temperature °C. | Number of Flies | Mean Pupal Development Time in Hrs. | Mean Wing Rating |
|---|---|---|---|
| 18 | 60 | 192.5 | 2.47 |
| 23 | 81 | 127 | 1.75 |
| 28 | 44 | 91.5 | 1.40 |

period in which the wing development was affected by the temperature (Temperature Effective Period). Prepupae were maintained at one temperature, 18° C, for a period of time and then later transferred to the second temperature. 28° C, to complete their development. The results of these transfers demonstrated that the temperature effective period lies somewhere between 15 and 20 hours in pupal development. Further experimentation would be necessary to more closely define this critical period.

Reference: Schultz, J. 1929. Genetics 14: 366-419.

Grossfield, J. and W.L. Pak. Purdue University, Lafayette, Indiana. Localization of electroretinogram mutants.

We have reported the isolation and physiological characterization of visual mutants which show an abnormal electroretinogram (ERG). Mutants x-7 and x-14 lack the "on" and "off" transients present in the wild type ERG and thus give the appearance of an isolated receptor response. Mutants x-12, 13, 16 and 24 show a gradation of response from none at all to a slow small amplitude response. These mutants may represent altered visual pigment. Allelism tests show that at least three distinct cistrons have been functionally identified. (See Pak, Grossfield and Arnold, Nature 227: 518 (1970) for pictures of electrophysiological phenotypes and the allelism tests.)

Here we present data for the map positions of the three loci. x-7 was mapped using the multiply marked X chromosomes sc cv v f and sc cv m f; x-14 with sc cv v f and m f car; x-12 with sc cv v f and y cv v f. The remaining mutants were just mapped a single time: x-13 and x-16 using sc ec cv ptg and x-24 with y w cv m f.

Table I. Position of Visual Mutants on X-chromosome.

| Mutant | Number of Crossovers Between | | Recombinational Fraction Between Mutant and Nearest Marker | Map Position * |
|---|---|---|---|---|
| x-12 | sc and x-12 | 39 | .493 | $6.8 \pm \frac{1.4}{1.5}$ |
| | x-12 and cv | 40 | | |
| x-12 | y and x-12 | 44 | .473 | $6.5 \pm \frac{1.3}{1.4}$ |
| | x-12 and cv | 49 | | |
| x-13 | ec and X-13 | 12 | .163 | $6.8 \pm \frac{.8}{.6}$ |
| | x-13 and cv | 62 | | |
| x-16 | ec and x-16 | 6 | .176 | $6.9 \pm \frac{1.4}{.9}$ |
| | x-16 and cv | 28 | | |
| x-24 | w and x-24 | 36 | .324 | $5.5 \pm 1.0$ |
| | x-24 and cv | 111 | | |
| x-7 | cv and x-7 | 41 | .359 | $26.1 \pm \frac{2.1}{2.7}$ |
| | x-7 and v | 23 | | |
| x-7 | cv and x-7 | 60 | .439 | $26.3 \pm \frac{2.1}{2.7}$ |
| | x-7 and m | 47 | | |
| x-14 | m and x-14 | 10 | .149 | $53.5 \pm \frac{1.7}{2.1}$ |
| | x-14 and f | 57 | | |
| x-14 | v and x-14 | 58 | .236 | $51.1 \pm \frac{2.1}{2.7}$ |
| | x-14 and f | 18 | | |

* Confidence limits correspond to 5% significance level. W.L. Stevens J. Genetics 43: 301-307 (1942)

Although these mutants were generated by our phototaxis assay procedure we did not rely on phototaxis for characterization of the mutant phenotype for mapping. $F_2$ males of all recombinant classes from $F_1$ heterozygous females were individually tested for their electrophysiological phenotype. Recording of the ERG gives unequivical identification of each visual mutant and visual inspection of each male provided identification of the usual morphological markers associated with each all-or-none ERG response. The use of markers on either side of each ERG mutant provided unambiguous localization. Recombination rate in the interval containing each mutant was not significantly different from standard values.

The recombination data agree with our earlier allelism tests in delimiting three loci, x-7, which we have shown to be allelic with tan, x-14 and x-(12, 13, 16, 24). It is interesting that x-7 and x-14, which have the same phenotype are more than 20 map units apart. This indicates that there may be more than one way of blocking "on" and "off" transients.

Supported by grant GB-8140 to Dr. W.L. Pak and an NIH Health Sciences Advancement Award to Purdue University.

Mikulska, Grygoń. Department of Zoology, University of Nicholas Copernicus, Institute of Biology, Toruń, Poland. The number of ovarioles in the ovaries of females of reciprocal crosses of the types wild of Torun and vg/vg D. melanogaster Meig.

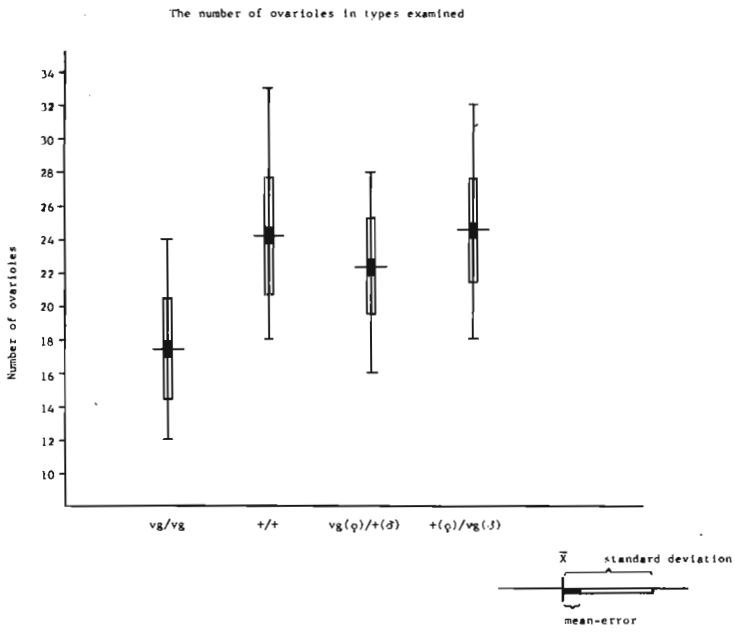
The fecundity of females heterozygous for vg originating from the cross of wild type of Toruń and vg/vg was examined by Grygoń (1970). The result of crossing was always advantageous when compared with vg/vg, and compared with +/+, it was advantageous if the female used was +/+. With reference to these studies, the anatomical verification is presented here.

The numbers of ovarioles were counted in the following types: 1. +/+ of Toruń, which has been bred since 1960 in the Genetic Laboratory of Zoology Department in Toruń, 2. vg/vg, originating from the laboratory ETH in Zürich and bred in Toruń since 1966 and 3. females originating from reciprocal crosses of these types.

All the flies were raised on cornmeal medium (500 g of water, 4 g agar, 34 g sugar, 68 g cornmeal and yeast suspension - for 10 bottles) in excess of the medium and uniform conditions. Ten pairs of virgin flies were used to start each culture. The ovaries were removed on the fifth day of life and they were stained with methylene blue in Ringer's fluid with a few drops of alcohol to harden the tissues (Melou 1961). The statistic analysis of results was based on a sample of 50 individuals and from each one ovary was prepared (Melou's method). The results are presented in the table and illustrated in the diagram. As they show, the type +/+ of Toruń has an average number of ovarioles of 24.2 ± 0.98.

In comparison to the French type Banyuls-Union and Oregon (Melou op.cit.) it is a little more fecund. The type vg/vg indicated 17.44 ± 0.44.

The number of ovarioles in types examined




The number of ovarioles in types examined

| Number of ovarioles in: | vg/vg | +/+ | +(♂)/vg(♀) | +(♀)/vg(♂) |
|---|---|---|---|---|
| Range | 12 - 24 | 18 - 33 | 16 - 28 | 18 - 32 |
| M (n=50 | 17.44 ± 0.44 | 24.2 ± 0.98 | 22.36 ± 0.39 | 24.5 ± 0.43 |

Females of the reciprocal cross in which the +/+ female was used, showed a slightly higher number of ovarioles than the type +/+, and a slightly lower number when the female used for crossing was vg/vg.

As in the initial crosses the females were vg/vg both combinations seemed to be expedient.

These anatomical relations explain the experimental data of Grygoń (op. cit.) and are in accord with them.

References: Grygoń, B., 1970, Zesz. Nauk. UMK Toruń, Nauki Mat. Przyr. 22, Biologia 12: 27-36; Melou, J.P., 1961, Ann. de Génétique 3: 25-28.

Hedrick, P.W. University of Kansas, Lawrence, Kansas. Competition experiments between D. melanogaster and D. simulans.

J.S.F. Barker tested a number of D. melanogaster strains against his ver strain of D. simulans in order to find a D. melanogaster strain of approximately equal competitive ability. He selected a yw strain that produced 45% D. melanogaster progeny when 50% of the parents were D. melanogaster.

In order to test the competitive ability of these two strains over a longer period of time, I initiated a series of serial transfer experiments. In these experiments all freshly emerged adults from several different age bottles during a certain time period were combined in a fresh bottle at the end of the time period. This procedure allows a semi-continuous population to be maintained.

Samples of the yw D. melanogaster and the ver D. simulans were obtained from Barker. An initial experiment was set up with parental D. melanogaster percentages of 10%, 50% and 90%. Unexpectedly, the D. melanogaster (Barker 1) eliminated the D. simulans (simulans 1) almost immediately (Table 1). To check these results a sample (Chicago 1) of the original yw stock was obtained from W.G. Baker. A second experiment was performed which repeated the 10% level of the first and all three levels for the second strain. Again Barker 1 eliminated simulans 1, but surprisingly Chicago 1 was eliminated by simulans 1 at 10% and 50% and greatly reduced at 90%. New samples (Barker 2, simulans 2, and Chicago 2) were obtained from both sources and a third experiment was carried out. In this experiment both the Barker 2 and Chicago 2 samples remained the same or slightly decreased in frequency from parental frequencies over a seven week period.

Table 1. Per cent D. melanogaster (yw) observed from competition with D. simulans (ver) using a serial transfer method. Values are the means of three replicates. Several initial percentages were not conducted (N.C.) each experiment.

| Experiment 1 | Bottle begun on Day | Initial percentage yw Barker 1, simulans 1 10% | 50% | 90% | Initial percentage yw 10% | 50% | 90% |
|---|---|---|---|---|---|---|---|
| | 0 | 45.6 | 97.0 | 98.4 | N.C. | N.C. | N.C. |
| | 3 | 49.8 | 93.5 | 100.0 | | | |
| | 7 | 59.2 | 95.9 | 100.0 | | | |
| | 10 | 86.2 | 98.6 | - | | | |
| | 14 | 93.4 | - | - | | | |

| Experiment 2 | | Barker 1, simulans 1 10% | 50% | 90% | Chicago 1, simulans 1 10% | 50% | 90% |
|---|---|---|---|---|---|---|---|
| | 0 | 67.4 | N.C. | N.C. | 8.9 | 31.8 | 100.0 |
| | 7 | 59.7 | | | 1.4 | 5.7 | 38.4 |
| | 14 | 95.8 | | | 4.8 | 5.6 | 89.7 |
| | 21 | 95.8 | | | 0.0 | 0.0 | 12.5 |
| | 28 | 98.8 | | | 0.0 | 0.3 | 22.0 |

| Experiment 3 | | Barker 2, simulans 2 10% | 50% | 90% | Chicago 2, simulans 2 10% | 50% | 90% |
|---|---|---|---|---|---|---|---|
| | 0 | 7.1 | 36.8 | N.C. | N.C. | 58.4 | 93.9 |
| | 7 | 11.4 | 57.5 | | | 80.2 | 94.7 |
| | 14 | 10.3 | 47.5 | | | 52.5 | 92.1 |
| | 21 | 16.7 | 44.4 | | | 79.4 | 80.6 |
| | 28 | 13.9 | 61.1 | | | 63.2 | 90.8 |
| | 35 | 26.8 | 27.7 | | | 60.4 | 86.2 |
| | 42 | 11.2 | 16.5 | | | 32.6 | 85.5 |
| | 49 | 9.2 | 21.4 | | | 29.6 | 78.8 |

The following hypothesis explains these results and is presently being tested. The

Barker 1 sample became adapted to the different conditions in my laboratory (a different food and the change from vials to bottles are probably the most dramatic differences) before experiment 1 was conducted. This adaptation increased the "competitive ability" of Barker 1 so that it quickly eliminated simulans 1. Simulans 1 also adapted in some manner so that it could outcompete the fresh Chicago 1 sample in experiment 2. The fact that experiment 3 results were as initially predicted was because all samples were obtained simultaneously and the experiment was run immediately after they were obtained. Further support of this hypothesis comes from Barker (pers. comm.) who has found in a second one-generation test that his stock in a recent retest produced approximately 40% D. melanogaster from 50% D. melanogaster parents, a value very close to his earlier results and quite unlike mine in experiments 1 and 2.

Grossfield, J. Purdue University, Lafayette, Indiana. A non-heuristic attribute of the ERG.

In conversation with several colleagues the question of electroretinograms (ERG's) being used to trace evolutionary patterns in the genus has arisen. I wish to point out that all species of Drosophila tested to date have the same waveform and time course when ERG's are recorded under comparable conditions. This has held true over the past few years when ERG's have been cursorily checked in this laboratory for members of the melanogaster, obscura, virilis, quinaria, robusta, and annulimanna species groups. Indeed, on the basis of available information, the same is true for all Diptera, with flies such as Calliphora and Musca showing larger amplitude responses. The presence of screening or accessory pigments in the eyes of various species may change the sensitivity of the response somewhat but that would be the maximal effect expected.

Hall, J.C. University of Washington, Seattle, Washington. The failure of two alleles of c(3)G to increase frequencies of X-linked lethals.

Green (Mut. Research 10:353, 1970) has discovered a putative allele of the recombination-deficient mutant, c(3)G, picked up as a mutator gene. It is a third chromosome semidominant whose locus is absent from Df(3R) $sbd^{105}$--as is true for c(3)G. The frequencies of mutations at certain X chromosome loci are increased in the presence of the mutator gene in females. In addition, recombination is somewhat reduced by this mutant (M. Green, personal communication). Green's preliminary allelism tests show that females bearing the mutator and c(3)G in heterozygous condition do not generate mutations in the relatively high frequency found for the mutator in homozygous condition. This means that a) c(3)G and the mutator are alleles, but c(3)G is not a mutator; or b) c(3)G may or may not be a mutator, but it is not an allele of Green's mutant.

Both c(3)G and $c(3)G^{68}$ -- a newly arisen allele of this meiotic mutant (mei-W22 of Sandler, DIS 47, 1971, in press) -- have been directly assayed for possible mutator properties. Parry (Ph.D. Dissertation, University of Washington, 1970) found that a meiotic mutant which lowers recombination and increases nondisjunction generates increased frequencies of sex-linked lethals. Such sex-linked lethal tests were carried out for the two alleles of c(3)G, in which the treatment of X chromosomes consisted of passage of these X chromosomes through females homozygous for either meiotic mutant. For c(3)G only one of 473 treated X chromosomes carried a lethal. And for $c(3)G^{68}$ none of 553 X's had a lethal induced. In a control, seven of 931 X's recovered from $c(3)G^{+}/c(3)G^{+}$ females bore a lethal. Four of these lethals arose from one female, and three from another, so the seven lethals probably represent only two mutations, each of which occurred at an oogonial stage and was proliferated. In any event, c(3)G does not appear to be a mutator gene.

Samples of the X chromosomes passed through these three kinds of females were examined for the presence of half chromatid (mosaic) lethals (produced in high frequency by chemical mutagens -- e.g. Carlson and Southin, Genetics 48:663, 1963). If c(3)G were generating increased frequencies of such half chromatid lethals in meiosis, after chromosome replication, they would go undetected among the $F_1$ males in a sex-linked lethal test (defining the P generation mothers as those bearing an X chromosome balancer and an X from a c(3)G female). However, of 87 X's from c(3)G, 70 from $c(3)G^{68}$, and 292 from $c(3)G^{+}$, none was found (in the $F_2$) to have originally carried a half chromatid lethal.

Stockert, J.C. Centro de Investigaciones sobre Reproducción, Facultad de Medicina, Buenos Aires, Argentina. Alteration in the spermatocyte nuclei of a testis of Drosophila hydei.

During the course of cytological studies on spermatogenesis in D. hydei we have found a morphological abnormality with characteristics we would like to present. An apparently normal larva of D. hydei showed spermatocytes with the lampbrush loops of the Y chromosome normally developed (see reviews by Hess and Meyer 1968; Hess 1969), in one testis (Fig. 1-a), while the other one showed a very heterogeneous aspect in which normal spermatocytes and spermatocytes with an abnormal organization of the nuclear structure participated (Fig. 1-b, c, d).

The most prominent characteristics of these cells were the following:

1) A variable reduction in the size of the peripheral (attached to the nuclear envelope) nucleolus.

2) The occurrence of 1 or more (sometimes 4 or 5) free spherical bodies without the appearance of any normal loop, but quite similar in optic density and structure to the peripheral nucleolar mass. The size of these free nucleolus-like bodies was variable, and when it did not appear, the peripheral nucleolus was normal in size.

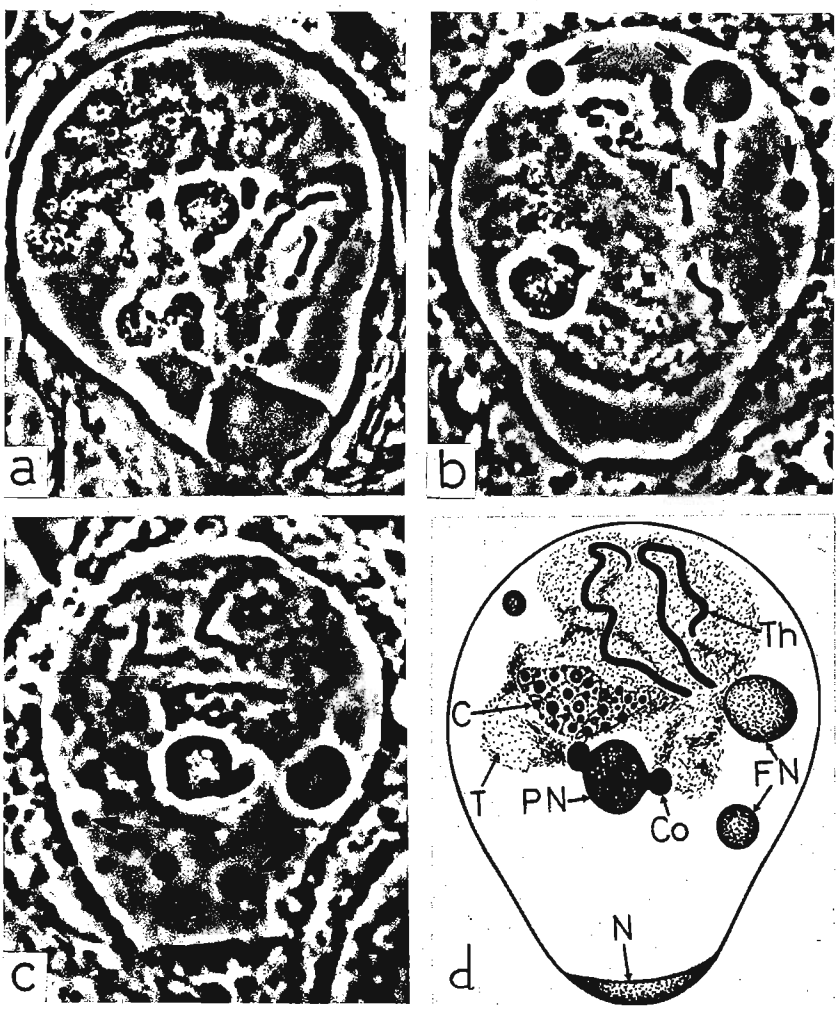



Figure 1-a: normal spermatocyte; b,c: abnormal nuclei of spermatocytes, with free nucleolus-like bodies (arrows). Living spermatocytes, phase contrast. -d: schematic representation of one of these altered nuclei. Th: compact threads; PN: pseudonucleolus; Co: cones; N: nucleolus; FN: free nucleolus-like bodies; C: clubs; T: tubular ribbons.

3) Frequently altered orientation of some loops, i.e. the compact threads or the clubs that were bound to one or more of the free nucleolus-like bodies, instead of the normal attachment to the peripheral nucleolus. However, in other cells we also observed a normal orientation of these loops in spite of the presence of free nucleolus-like bodies.

4) The total absence of loops in some nuclei of fully grown spermatocytes, and the presence of duplicate loops (i.e. two pseudonucleoli) in a few other nuclei.

Unfortunately we could not obtain more data about this testis. A possible interpretation of the cytologic abnormalities we have found could be based on the existence of a fragmentation of the Y chromosome in some cells, in early stages of development of this testis. If

each nucleolar mass has its own organizer region (belonging to chromosomes, see Hay 1968, or free and segregated from the chromosomes, see Miller and Beatty 1969), it seems logical to assume that the free nucleoli to which certain loops were attached could contain portions of the nucleolar organizer near the loci of the loop forming sites in the Y chromosome.

References: Hay, E.D., 1968, Structure and function of the nucleolus in developing cells. In: Ultrastructure in Biological Systems. The Nucleus. (A.J. Dalton and F. Haguenau, Eds.) Acad. Press, N.Y. and London; Hess, O., 1969, Genetic activities of the Y chromosome in Drosophila. Ann. Embryol. Morphog., suppl. 1: 165-176; Hess, O. and G.F. Meyer, 1968, Genetic activities of the Y chromosome in Drosophila during spermatogenesis. Adv. Genetics 14: 171-223; Miller, O.L. and B.R. Beatty, 1969, Visualization of nucleolar genes. Science 164: 955-957.

Laughnan, J.R., S.J. Gabay and I.N. Montgomery. University of Illinois, Urbana, Illinois. Genetic basis for the exceptional events in Dp(1;1)MNB-8 Drosophila melanogaster males.

Males carrying Dp(1;1)MNB-8, designated the modified long duplication (MLD), and having the genotype f($B^+$ $os^+$)(B os)car (duplicated members in parentheses), are B $os^+$ in phenotype. When mated with attached-X females, patroclinous sons are mainly of the B $os^+$ (parental) class. Not infrequently, however, three classes of exceptional sons are produced. The distribution of exceptional offspring among progeny of single pair matings of the above type indicates that the exceptional event occurs almost exclusively in germinal tissues of the MLD parent and that it often takes place at a relatively early stage in the development of germinal elements. Analysis of salivary gland chromosomes reveals that the exceptional events involve a loss of portions of the duplication.-----Genetic analyses (Gabay and Laughnan, 1970) of progenies of MLD male parents from six different strains indicate a striking variation in overall frequency of exceptional events, and in the relative frequencies of the different kinds of exceptions. There have also been instances of stabilization within sublines of strains characterized by a high frequency of exceptional events, and of changes from a relatively stable to relatively unstable or active condition.-----The existence of stable and unstable MLD strains, and the strong tendency for these traits to be inherited through many generations, suggest a genetic control over the exceptional event. The particular mating system we employ, and the fact that, except for sudden changes of the type noted above, the various strains have, over many generations, retained the differences in frequency of exceptional events which they exhibit, make it unlikely that genetic control resides in either the autosomes or in the Y chromosome. On the other hand, since the duplication-bearing X chromosome of an MLD male parent is passed from father to son in each mating cycle, it seems most likely that if exceptional events are under the control of a chromosomal gene, the latter is located in the X chromosome. This hypothesis was tested using marked females that derive one duplication-X chromosome from an MLD stock characterized by a relatively high frequency of exceptional events, and another duplication-X chromosome from an MLD strain that is stable in this regard. These f "unstable" /$f^+$ "stable" females were mated with wild-type males and f and $f^+$ MLD sons were test mated with attached-X females to search for patroclinous exceptional sons. Among the 550 progenies from matings involving the f MLD sons, 205, or 37 per cent, had one or more exceptions, while, in similar matings, 458 $f^+$ MLD sons produced only seven, or 1.5 per cent, progenies with exceptions. These results indicate that genetic control of the exceptional event is carried in the X chromosome. Since forked, the marker used here to screen for sons carrying the X chromosomes from the unstable and stable MLD sources, is close to the distal end of Dp(1;1) MNB-8, and since the screen proved to be highly effective in identifying unstable and stable X chromosomes in the test matings, it appears that the genetic element in control of exceptional events is at a site in, or not far removed from the duplication itself. As noted above, the exceptional events involve a loss of chromosomal material from the duplication; moreover, the array of deficiency types among exceptions from the MLD strains suggests that there are characteristic hot spots for breakage in these strains. Hence there is no reason to assume a separate, closely-linked controlling element in the X-chromosome. For the time being it is sufficient to consider that the X chromosome of an unstable MLD strain differs from that of a stable strain in carrying within the duplication two or more sites that are highly susceptible to breakage and consequential loss of specific chromosomal segments.

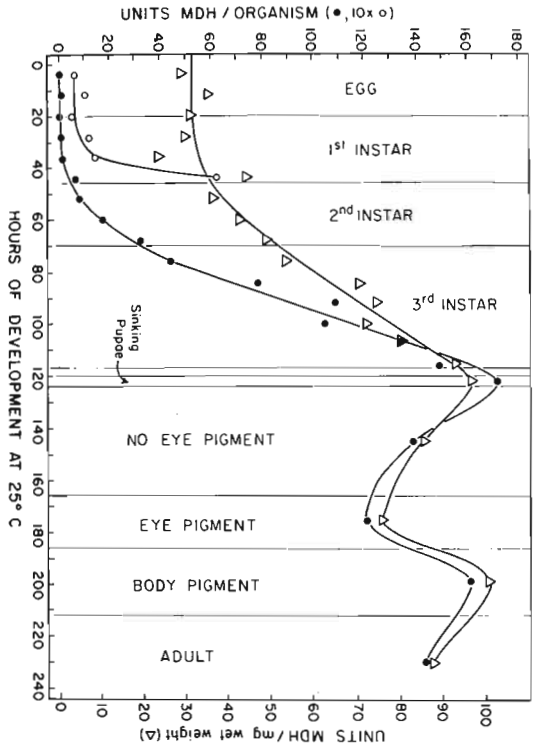
This work was supported by National Science Foundation Grant GB-7635.

Reference: Gabay, S.J. and J.R. Laughnan, 1970, Genetics 65: 249-265.

Anderson, M.* The Johns Hopkins University, Baltimore, Maryland. Variations in the level of malate dehydrogenase during development.

The variation in the level of malate dehydrogenase (L-malate: NAD oxidoreductase, EC 1.1.1.37) during development has been studied in Oregon RCH flies. The techniques used to obtain eggs of known age, to raise the eggs and larvae, and to stage pupae, have been described (Rechsteiner, 1970). At various times, eggs and/or larvae were collected by washing them into fine mesh nylon net with distilled water. An aliquot was counted and weighed; the remainder of the organisms was weighed, and the number estimated from the weight of the counted sample. They were homogenized at 100 mg/ml in 0.2 M sodium phosphate, pH 6.4, 0.1% in phenylthiourea. The homogenates were centrifuged for 20 minutes at 17,000 rpm, and the MDH activity of the supernatant solution was immediately determined. For pupal points, pupae were washed from the walls of an established bottle, staged by morphological characteristics, and counted, weighed, homogenized, and assayed. The pupae were found to be significantly lighter than the oldest larvae, presumably because they were raised under more crowded conditions on the standard food. To correct for this in determining enzyme units/organism, the experimentally determined enzymatic activity for the pupal points was multiplied by the ratio of the weight/organism of the oldest larvae to that of the youngest pupae. That the difference in body weight is not merely a reflection of differences in body water, and that some sort of correction factor need be applied, is shown by the fact that enzyme activity/mg. wet weight and enzyme activity/mg. extracted protein were similar for the oldest larvae and youngest pupae, while enzyme activity/organism and wet weight/organism in the pupae were about half of the larval figure.

MDH was assayed at 30°C on a Zeiss monochromator equipped with a Gilford Absorbance Recorder. Two to 25 µl. of homogenate were added to 3 ml. of an assay mixture containing 16.7 mM malate, 2 mM $NAD^+$, 50 mM glycine, pH 10.0, and the initial rate of change in O.D. at 340 nm. was determined.




It is apparent that the level of MDH in Drosophila undergoes stage-specific variations during development which are regulated independently of total soluble protein. Other Drosophila dehydrogenases show variations which are similar, but not identical. The levels of MDH are approximately constant up to hatching. This has been observed in alcohol dehydrogenase (unpublished observations), isocitrate dehydrogenase (Fox, 1971), α-glycerolphosphate dehydrogenase, (Rechsteiner, 1970), and glucose-6-phosphate dehydrogenase and 6-phosphogluconate dehydrogenase (Wright and Shaw, 1970). However, β-hydroxybutyrate dehydrogenase (unpublished observations) and lactate dehydrogenase (Rechsteiner, 1970) show seven- and twenty-fold increases, respectively, in enzyme units per organism during embryogenesis. The drop in MDH activity per organism during metamorphosis (30%) is much less than that observed for alcohol dehydrogenase (500%), β-hydroxybutyrate dehydrogenase (900%) (unpublished observations), and α-glycerolphosphate dehydrogenase (300%, Rechsteiner, 1970), but similar to that seen in $NADP^+$-dependent isocitrate dehydrogenase (40%, Fox, 1971). The measured activity of MDH represents the sum of s-MDH and m-MDH. Separate measurement of the two during development would provide interesting data on the control of the levels of the forms of MDH. If the two are not regulated coordinately, this could bear on the significance of the existence of two forms of the enzyme. E.g., different cell types, or a single cell type at different times

in the life cycle of the cell or organism, could require a different balance between the mitochondrial and supernatant forms of the enzyme. Experiments in other systems (Kitto, 1967; Kitto and Lewis, 1967), including D. virilis (McReynolds and Kitto, 1970), indicate that antibodies to the two forms of MDH do not crossreact; hence immunologic techniques should allow one to measure the activities of the two forms separately during development.

References: Fox, D.J., 1971, Biochem. Genet. 5:69-80; Kitto, G.B., 1967, Biochem. Biophys. Acta 139:16-23. Kitto, G.B., and R.G. Lewis, 1967, Biochem. Biophys. Acta 139: 1-15; McReynolds, M.S., and G.B. Kitto, 1970, Biochem. Biophys. Acta 198:165-175; Rechsteiner, M.C., 1970, J. Ins. Physiol. 16:1179-1197; Wright, D.A., and C.R. Shaw, 1970, Biochem. Genet. 4:385-394.

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Parzen, S.D., M.J. Kessenich, and A.S. Fox. University of Wisconsin, Madison, Wisconsin.

A method for the preparation on high molecular weight DNA from adult D. melanogaster.

Twenty-five grams of flies (wet weight) are ground with 120 ml of cold absolute methanol in an all-glass homogenizer in an ice bucket. The homogenate is centrifuged at 12,000 Xg for 10 minutes at 4° C, the supernatant discarded, and the pellet reground in 120 ml of a solution containing 0.15 M NaCl, 0.015 M sodium citrate, and 0.05 M EDTA at pH 7.0 (Solution No. 1). This is centrifuged as before and the pellet is ground again in 120 ml of Solution No. 1, followed by another centrifugation. After this centrfugation the pellet is suspended in 40 ml of 0.1 M NaCl, and 40 ml of 5% Aerosol O.T. in 0.1 M NaCl is added slowly with gentle stirring. The suspension is placed in a water bath at 50° C for one hour. It is then allowed to cool to room temperature and sufficient solid NaCl is added to raise the salt molarity to 1.0 M. After 10 minutes the preparation is centrifuged at 12,000 Xg for 10 minutes at 4° C. The precipitate is now discarded and one volume of cold 2-ethoxyethanol is added to the supernatant. This is placed in a freezer for 15 minutes, and is then centrifuged for 10 minutes at 10,000 Xg.

The precipitate is dissolved in 20 ml of 0.15 M NaCl, and is deproteinized by shaking vigorously for 10 minutes with an equal volume of a solution containing 24 parts chloroform to 1 of isoamyl alcohol. It is then briefly centrifuged to separate the phases, the aqueous phase is removed, and to the aqueous phase is added two volumes of cold ethanol. The DNA is now spooled out on a glass rod and dissolved in 9 ml of 0.015 M NaCl. When it is completely dissolved, 1 ml of 1.5 M NaCl is added.

Removal of contaminating RNA is now carried out by the addition of 1.0 ml of a 0.2% RNase solution (prepared in 0.1 M TRIS, pH 7.6, and heated for 10 minutes at 80° C to inactivate contaminating DNase). This is allowed to incubate in a 37° C water bath for one to three hours. After this, the solution is deproteinized as before. The DNA is then precipitated with either ethanol or 2-ethoxyethanol, deproteinized again, and precipitated once more with either ethanol or 2-ethoxyethanol. Following the last precipitation it is dissolved in 9 ml of 0.015 M NaCl + 1.0 ml 3.0 M NaAcetate containing 0.001 M EDTA, pH 7.0. It is then precipitated by the dropwise addition of 0.60 volumes of cold isopropanol. The DNA is spooled out and dissolved in 9 ml of 0.015 M NaCl + 1.0 ml 3.0 M NaAcetate containing 0.001 M EDTA, pH 7.0, and precipitated by the addition of 0.60 volumes of cold isopropanol. This final precipitate is dissolved in 10 ml of 0.15 M NaCl and is then typically chromatographed on a Sepharose-4B column, eluting with 0.15 M NaCl, collecting and pooling those fractions coming off the column immediately after the void volume.

Orcinol tests for RNA are negative. Melting point determinations show a Tm = 83.6° C with a hyperchromic effect of 40-45% in 0.15 M NaCl. $A_{260\ m\mu}/A_{280\ m\mu}$ = 1.8 - 2.0. Sedimentation equilibrium studies in CsCl show a single narrow main band at $\rho$ = 1.702 with a small shoulder at $\rho$ = 1.687. Prior to Sepharose-4B chromatography an additional smaller but wider band is present at $\rho$ = 1.675. Yield has been as great as 2.5 mg DNA from twenty-five g of flies.

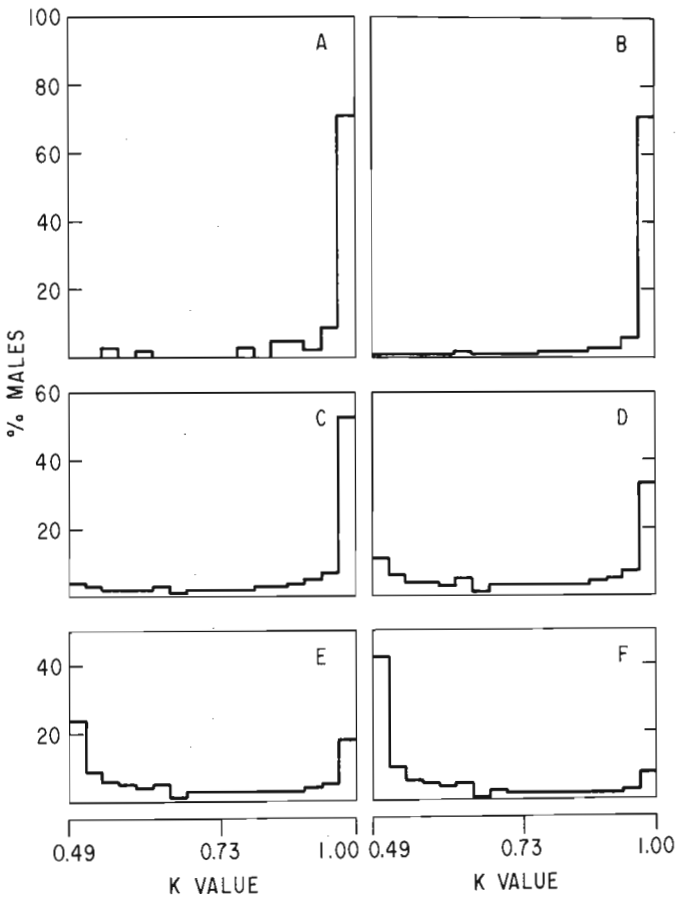
Supported by the following grants from the National Institutes of Health, USPHS: GM-11777, GM-15422, and GM-00398.

Miklos, G. L. G. University of California, San Diego, California. SD distributions and the measurement of distortion.

In Segregation-Distorter (SD) experiments, K value distributions are often encountered which have seemingly unusual shapes, and a distribution can appear to be a composite of two different histograms. It is sometimes found in such an experiment that many or most SD/SD$^+$ males exhibit very high distortion, whilst others show greatly reduced distortion or none at all. The interpretation of the shape of SD histograms can be approached using the make analysis of Miklos and Smith-White (Genetics 67:305-317, 1971). This communication presents the results of an experiment in which an unusual SD distribution is interpreted as having a high variance on the make scale. It further gives other odd shapes which can be generated by the make method, and the following examples extend the spectrum of histogram shapes initiated by the two high variance examples of Miklos and Smith-White (1971). The problems associated with the use of K value as a measure of distortion will also be clear from the examples.

A. During a series of experiments designed to investigate the phenomenon of conditional distortion, (Sandler and Hiraizumi, Genetics 46:585-604, 1961) an SD-72 chromosome was passed through females, and the SD-72/cn bw sons of the SD-72/cn bw mothers were tested for distortion in the standard way. Instead of obtaining the characteristic high K value distribution, in which all males yield values near 1.00, it was found that approximately 25 per cent of the males exhibited markedly reduced distortion. These results are shown in histogram A, in which the 65 tested males yielded 9,509 progeny. This type of histogram is frequently encountered in the SD literature, and it is due to a high level of distortion, together with large between-male variability.

The theoretical histograms B,C,D,E and F have been derived from normal distributions on the make scale. They all have the same high variance ($V_M$ = 4.0), and differ only in their means; B,C,D,E and F have means of 8,7,6,5 and 4 Probits respectively, corresponding to K values of 0.999, 0.98, 0.86, 0.67 and 0.54. It can be seen that the experimental histogram A corresponds to a theoretical one such as B, which has a high mean and a large underlying variance. The level of distortion of the SD-72 stock has thus remained unaltered during the passage of the SD-72 chromosome through females, however the between-male variance has increased. If the level of distortion is lowered, but a high between-male variance is retained, then SD distributions such as B,C,D,E and F are obtained. Histograms such as these are relatively abundant in the SD literature, and most probably occur when some form of heterogeneity is introduced into the SD stock.




It should be pointed out that if an SD distribution is highly skewed, its mean and median can differ by a large amount, and the mean will provide an inappropriate measure of distortion because it is influenced by the skewness. The median is unaffected and should be utilized in such cases.

B. These results demonstrate the deceptive nature of K value distributions. Another property is that K is not uniform over its range of measurement. The difference in distortion between K's of 0.95 and 0.90 is not the same as between 0.70 and 0.65 for example. It takes the same amount of "work" to go from K = 0.67 to K = 0.86 (ie, 5 Probits to 6 Probits), as it does to go from

K = 0.98 to K = 0.999 (7 Probits to 8 Probits). Thus changes in distortion in terms of K value can be misleading, and an unambiguous description of an SD experiment involves describing means, variances and changes in Probits.

The difficulties are increased further when tests of significance are involved. Owing to the nature of the K scale, two distributions may appear to be significantly different under standard statistical tests, but in reality, are not significantly different when compared in Probits. This is not due to a deficiency in the test itself, but rather to the unsuitable analytical properties of the K scale.

These results on the shape of SD histograms and the nonuniform changes in K over the range of distortion, further show that the phenomenon of Segregation-Distortion is best considered in terms of the make analysis.

Supported by A.E.C. Contract No. AT(04-3)-34 PA150.

Sandler, L. University of Washington, Seattle, Washington. Induction of autosomal meiotic mutants by EMS in D. melanogaster.

A scheme for the detection and isolation of autosomal meiotic mutants (i.e. mutants on either chromosome 2 or 3 that affect disjunction of either the sex chromosomes or chromosome 4 in either sex or X chromosome recombination in females) has been described by Sandler et al. (Genetics 60:525-558, 1968). They examined autosomes collected from natural populations.

This scheme has now been applied to mutagenized major autosomes. Chromosomes 2 and 3 were recovered from Canton-S males treated with EMS according to the method of Lewis and Bacher (DIS 43:193, 1968) using a treatment that produced about 10% (54/545) sex-linked recessive lethals after one additional backcross generation to resolve mosaics. The scheme of Sandler et al. for examining autosomes was also modified to resolve mosaics.

There were 35 lethal-free 2-3 complements examined for meiotic mutants -- 24 in both sexes, 8 in females only (the males were sterile) and 3 in males only (the females were sterile). Among these, two meiotic mutants were recovered: (1) mei-W5, a second chromosome recessive that causes the production, in homozygous males, of sperm lacking paternal chromosomes and has no obvious effects in females, and (2) mei-W22, a third chromosome recessive that eliminates recombination and increases nondisjunction in homozygous females and is sterile in males (for reasons not yet investigated).

From the cross, In(1LR)sc$^{VI}$, y pn v · y$^{+}$/y; spa$^{pol}$/spa$^{pol}$ females homozygous for the indicated meiotic mutant by Y$^{S}$X·Y$^{L}$, In(1)EN, v f B/O; C(4) RM, ci ey $^{R}$/O males, there were observed:

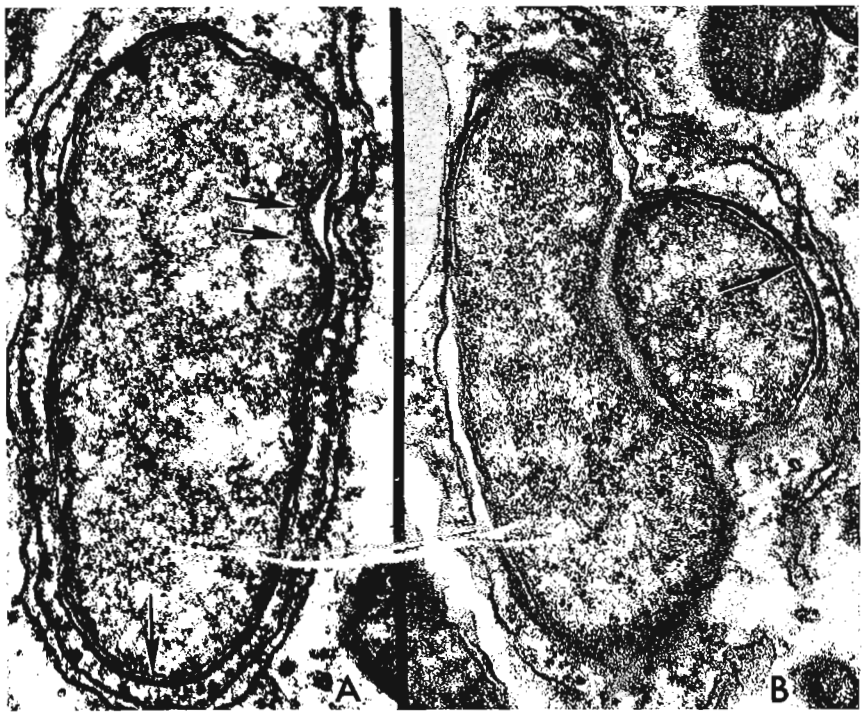
| | mei-W5 | | | | mei-W22 | | |
|---|---|---|---|---|---|---|---|
| | chromosome 4 | | | | chromosome 4 | | |
| X chromosome | + | pol | ey | X chromosome | + | pol | ey |
| (v,v$^{+}$)B/+♀♀ | 97 | 0 | 0 | (v,v$^{+}$)B/+♀♀ | 51 | 2 | 5 |
| B$^{+}$♀♀ | 0 | 0 | 0 | B$^{+}$♀♀ | 17 | 4 | 3 |
| v f B ♂♂ | 0 | 0 | 0 | v f B ♂♂ | 14 | 7 | 9 |
| pn v ♂♂ | 26 | 0 | 0 | pn v ♂♂ | 32 | 3 | 8 |
| y ♂♂ | 20 | 0 | 0 | y ♂♂ | 37 | 2 | 3 |
| y pn ♂♂ | 6 | 0 | 0 | y pn ♂♂ | 0 | 0 | 0 |
| v ♂♂ | 9 | 0 | 0 | v ♂♂ | 0 | 0 | 0 |
| y pn v ♂♂ | 11 | 0 | 0 | y pn v ♂♂ | 0 | 0 | 0 |
| + ♂♂ | 11 | 0 | 0 | + ♂♂ | 0 | 0 | 0 |
| pn ♂♂ | 2 | 0 | 0 | pn ♂♂ | 0 | 0 | 0 |
| y v ♂♂ | 2 | 0 | 0 | y v ♂♂ | 0 | 0 | 0 |

From the cross, In(1)FM6, y$^{31d}$ sc$^{8}$ dm B/y$^{+}$Y; spa$^{pol}$/spa$^{pol}$ males homozygous for mei-W5 by y pn/y pn; C(4) RM, ci ey $^{R}$/O females, there were observed: y$^{2}$ B ♀♀ = 213, pn ♂♂ = 217, B ♀♀ = 0, y pn ♂♂ = 9, y$^{2}$ B; pol ♀♀ = 0, y$^{2}$ B; ci ey ♀♀ = 33, pn; pol ♂♂ = 0, pn; ci ey ♂♂ = 28, B; pol ♀♀ = 0, B; ci, ey ♀♀ = 0, y pn; pol ♂♂ = 0, and y pn; ci ey ♂♂ = 0.

Kernaghan, R.P. SUNY at Stony Brook, New York. The ultrastructure of the organism associated with hybrid sterility in D. paulistorum.

Crosses between some semispecies of D. paulistorum produce sterile $F_1$ male offspring. Usually a cross between Mesitas females X Santa Marta males produce fertile $F_1$ male progeny but in recent years the male progeny are sterile as if the ability to produce sterile offspring has been acquired. At the electron microscope level, the testes of these males are congested with degenerating sperm and contain a microorganism or symbiont similar to that previously described by Kernaghan and Ehrman (1970). In addition, extracts prepared from such infected tissue are potent in inducing sterility in the sons of recipient females (Williamson, Ehrman and Kernaghan 1971).

High resolution analysis of testes of these sterile $F_1$ hybrid males, as well as developing eggs of their fertile sisters and mothers shows a cytoplasmic membrane limited vacuole enclosing one or more of the symbionts. In addition, each microorganism is limited by two membranes. The outer membrane may be juxtaposed to the vacuolar membrane to produce a more electron dense region (Figure A & B). The internal structure of the organism is described as a reticulate network of fibers and may or may not be accompanied by a rough peripherial granulation of ribosome-like material. Pleomorphic forms are not unusual ranging from a smaller dense granular form .1 μ in diameter to the larger reticular form .3 to .5 μ in diameter.




Electron micrograph of the reticulate form of the microorganism in the sterile $F_1$ male testis from a cross Mesitas females X Santa Marta males. Figure A, (upper arrows) show the duplicate membranes of the symbiont while Figure A, (lower arrow) and Figure B demonstrate the juxtaposition of the external membrane to the vacuolar membrane. 90,000X

Penicillin has no detectable effect on the ultrastructure of the symbiont. Both external membranes remain intact when such sterile $F_1$ hybrid males are raised on drug treated media. Large reticulate forms are common in these treated individuals. In adult tissue the symbiont has been detected only in the gonads while in some cases larval and pupal gut muscle exhibit the microorganism.

On the ultrastructural level alone, no clear assignment may be made as to the type of organism involved. Although a general similarity to mycoplasma-like morphology exists, the ultrastructure of a Rickettsia described by Briton and Burgdorfer (1971) or the fine structure of Chlamydia by Tamura et al (1971) is equally applicable.

References: Kernaghan, P. and L. Ehrman 1970 Chromosoma 29:291-304; Williamson, D., L. Ehrman and P. Kernaghan 1971 P.N.A.S. (in press); Brinton, L.P. and W. Burgdorfer 1971 J. Bacteriol. 105:1149-1159; Tamara, A., Matsumoto, A., G.P. Manire and N. Higashi, 1971 J. Bacteriol. 105:355-360.

P. Kernaghan acknowledges support of Nih Grant #AI09945.

Lefevre, G., Jr. San Fernando Valley State College, Northridge, California. Crossing over in an insertional translocation.

A cytogenetic analysis of some EMS-induced sex-linked lethals unexpectedly revealed an insertional translocation in which a segment of X extending from 1D1-2 to approximately 5C5-6, i.e., from su ($w^a$) through cv, was inserted in direct order near the tip of 3R, just before 100E1. This translocation, designated as T(1;3)SFV-70k, is illustrated in Fig. 1. The aneuploid deficiency segregant is lethal as a heterozygous female; the duplication segregant survives as a fertile female, but is lethal as a male.

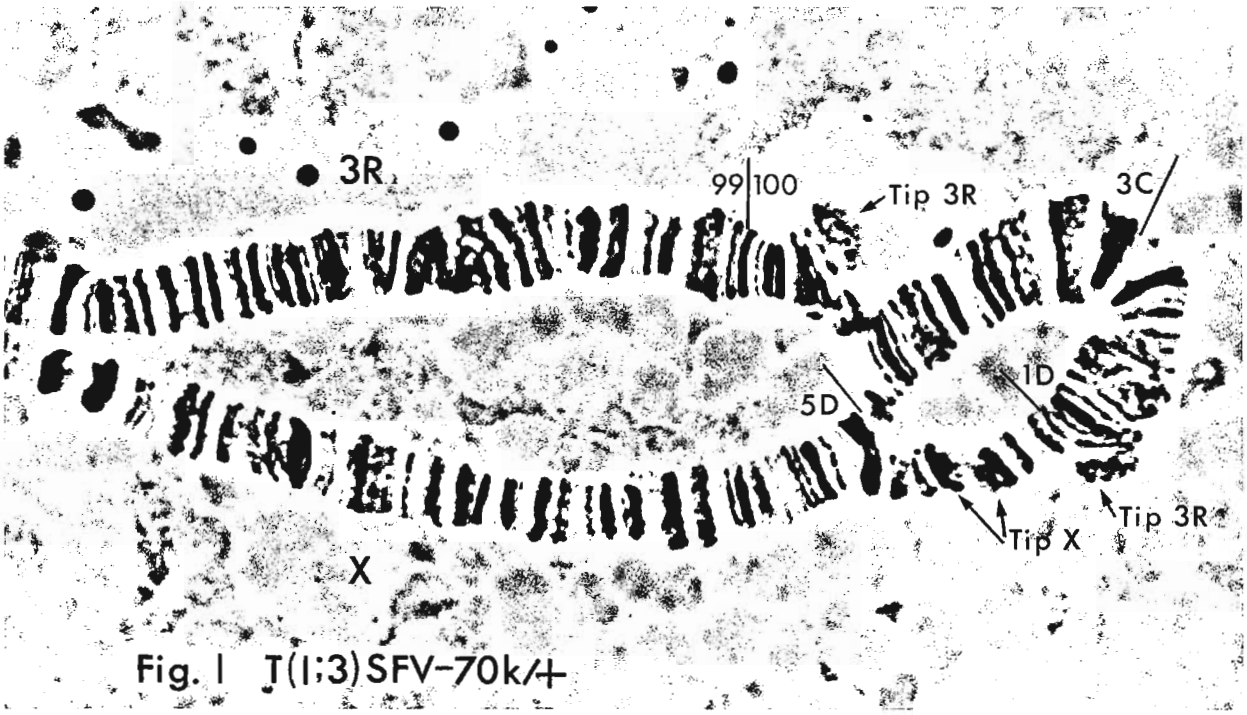



Fig. 1 T(1;3)SFV-70k/+

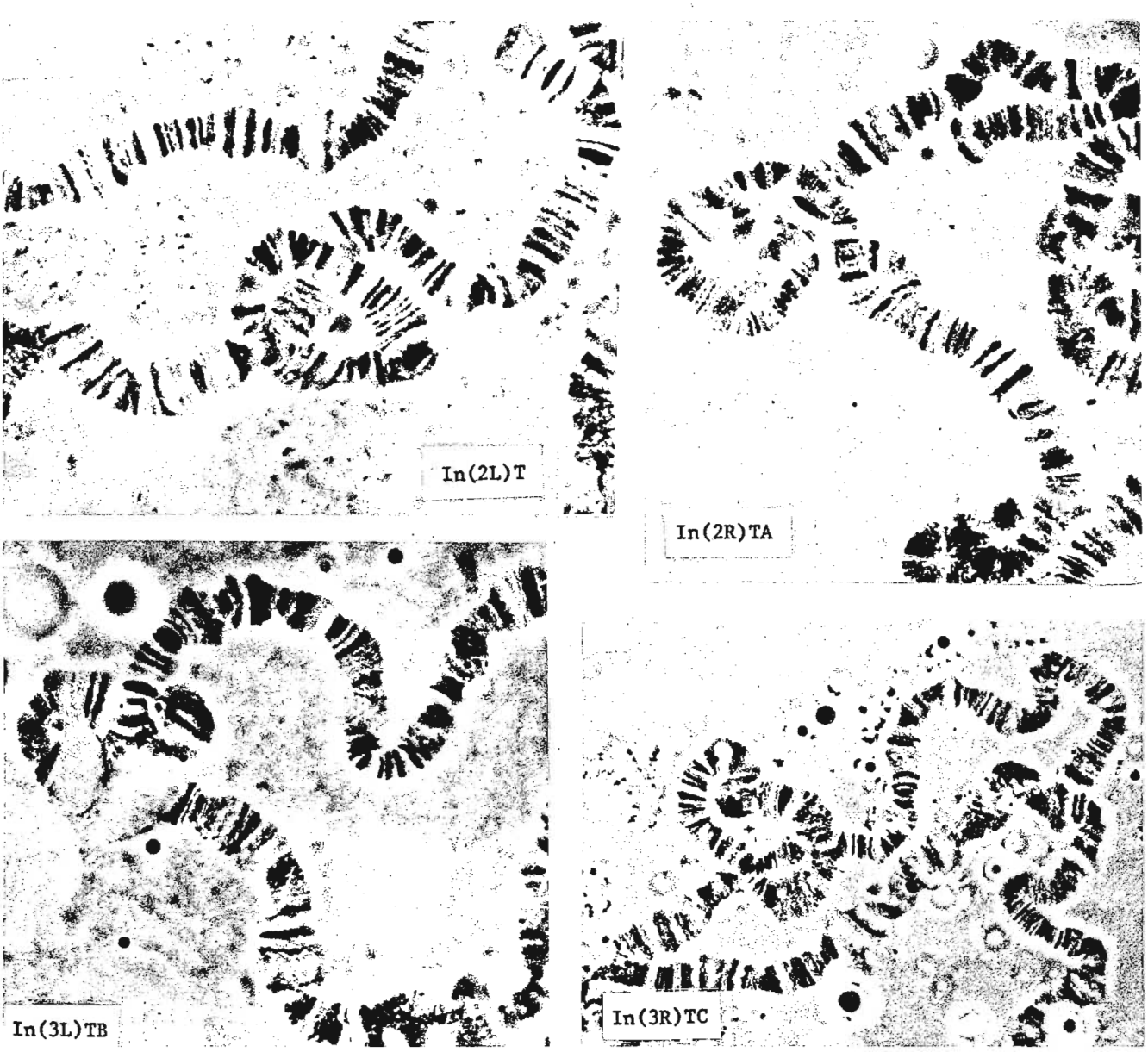
Because of the favorable orientation and location of the inserted material, an attempt was made to recover a single crossover between it and a normal, marked X. Although only a portion of such crossovers should be identifiable, a total of 4 were found among 2,551 daughters of $T(1;3)/y^2$ $w^a$ ec cv ct f females. Each of these recombinant daughters carried one T(1;3) chromosome in which the original insertional translocation had been converted by the single crossover into a reciprocal translocation. However, only a half-translocation was recovered in each recombinant fly. (Although the full reciprocal translocation can be recovered in a single individual, it should not be recognizable as a recombinant.)

The successful recovery of these crossovers demonstrates that effective synapsis does not require a zipperlike action initiated only at the telomere or centromere, but is compatible with the view of von Wettstein (PNAS 68:851-855, 1971) that precise synapsis between homologous elements can be initiated at any point.

Yang, H., K. Kojima and A. Kovarik. The University of Texas, Austin, Texas. Inversions in a Southwest Texas D. melanogaster population.

Four new and eight cosmopolitan chromosomal inversions on Chromosomes II and III were found in a year-round population of D. melanogaster in Southwest Texas. The flies were collected near Brownsville, Texas in 1970.

Break-points in these inversions were identified on the basis of the salivary gland chromosome map by Bridges (1935). The eight cosmopolitan inversions identified were: In(2L)t, In(2R)NS, In(3L)P, In(3L)M, In(3R)C, In(3R)K and In(3R)MO.




The new inversions (Fig. 1) were found in each arm of the second and third chromosomes. In(2L)T is a small single paracentric inversion between 30F and 36D. In(2R)TA is another small paracentric inversion between 50A and 53A. In(3L)TB is a small inversion between 70B

and 75A. In(3R)TC is a large paracentric one which is located between 84D and 91E. The break-points of this In(3R)TC are similar to those of In(3R)Antp$^{LC}$ which were induced by neutrons.

The relative frequencies of occurrence of these inversions are as follows, in percentages:

| | | | |
|---|---|---|---|
| In(2L)t = 14.5 | In(2L)T = 0.3 | In(2R)NS =20.9 | In(2R)TA = 0.3 |
| In(3L)P = 19.1 | In(3L)M = 1.0 | In(3L)TB = 0.3 | In(3R)TC = 0.3 |
| In(3R)K = 0.3 | In(3R)P =38.2 | In(3R)C = 3.9 | In(3R)MO = 0.3 |

The total of the genomes extracted by the Cy/Pm; Ubx/Sb method was 233.

Benner, D.B.* University of California, Riverside, California. Some evidence against the presence of suppressors of variegation on the Y chromosome.

Brosseau (1964) reports that the localized regions of the Y chromosome near the kl-2 fertility factor on Y$^{L}$ and proximal to ks-1 on Y$^{S}$ act as position-effect suppressors. I would like to present some evidence that suggests that there is no suppression of variegation by these localized regions.

The first evidence comes from an analysis of the Y-4R fragments reported by Parker (1965, 1967). The Dubinin effect (a position-effect variegation of the cubitus interruptus gene on the fourth chromosome) is observed only in those cases where 4R is located distal to kl-2. In these cases Y$^{S}$ is intact and the break in Y$^{L}$ is distal to kl-2 and therefore the presumed suppressor. The second evidence is of like nature and comes from a similar analysis of twenty Y-4R fragments that were produced using an unmarked Y obtained from a wild population. Sixteen of the fragments do not show the Dubinin effect and show no evidence of any of the KL fertility factors. The four remaining fragments have at least kl-1 and kl-2 present, and all four show the Dubinin effect.

The third bit of evidence that these specific regions of the Y may not be responsible for variegation suppression comes from an analysis of X detachments in which 4R and some portion of the Y from Parker's fragments have been attached to the X chromosome. C(1)RM, y v bb; ci ey$^{R}$ ♀♀ bearing the Y-4R, y$^{+}$ ci$^{+}$ ey$^{+}$ fragment were irradiated with 3Kr of X-rays within twelve hours of eclosion, mated to In(1)sc$^{SIL}$ sc$^{8R+S}$, sc$^{S1}$ sc$^{8}$ w$^{a}$ B/Y; ci ey$^{R}$ ♂♂, and allowed to lay eggs for four days. y v; ci$^{+}$ ey$^{+}$ ♂♂ were recovered and put into stock by mating to C(1)RM, y v bb/Y; ci ey$^{R}$ ♀♀. In all cases where the fertility factors that were present in the fragment have been lost the Dubinin effect has been lost. This suggests that the loss of variegation is accompanied by loss of the region adjacent to kl-2 and the region proximal to ks-1. In those cases where the proximal region of Y$^{L}$, and therefore the regions adjacent to kl-2, have not been disrupted the Dubinin effect persists. Likewise, in those cases where neither Y$^{S}$ nor Y$^{L}$ have been disrupted the variegation persists.

These results suggest that there is no region of the Y that specifically acts to suppress the Dubinin effect. The effect is absent when all or most of the Y is missing. It has been previously reported that the Y chromosome does not always act as a suppressor of the Dubinin effect and in fact may act as an enhancer of the effect (Panshin, 1938; Altofer, 1967). The results reported by Brosseau were obtained using a variegating B$^{S}$.

The results reported here are consistant with Brosseau's conclusion that the heterochromatic Y does not suppress variegation. The discrepancy in the results concerning the action of specific sites on the Y as variegation suppressors may mean that suppression of a particular variegation is the result of an effect on that specific rearrangement by the Y and not the pecular property of a specific region. In other words, the Y may have different effects on different rearrangements because of modifications in spatial associations within the nucleus (as suggested by Muller, 1935) not because of the special influences of specific regions

References: Altofer, N. 1967 Genetics 55:755-767; Brosseau, G.E. 1964 Genetics 50: 237; Muller, H.J. 1935 Fifteenth Int. Physiol. Congr., Leningrad; Panshin, I.B. 1938 Biol. Zh., Mosk. 7:837-868; Parker, D.R. 1965 Mutation Res. 2:523-529, 1967 Mutation Res. 4:333-337.

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Nash, W.G. The George Washington University, Washington, D.C. Deep orange and carnation: Another lethal gene combination in D.m.

The dor car lethal gene combination resembles both dor ry (Lucchesi, DIS 39: 127) and ey eyg (Hunt, Gen. Res. Camb. 15: 29-34) in that the genes involved are all recessive whose combined action results in death of the pupa. Unlike the latter two lethal combinations it is rare for dor car zygotes to develop beyond the early stages of the pupal period.

With the exception of having colorless malphigian tubules dor car larvae appear to develop in a completely normal manner. Shortly after pupating, however, normal development stops abruptly and is signaled by the formation of a large air bubble in the center of the pupa. Since larval development appears normal it seems likely that the lethal biochemical lesion is affecting some or all of the adult imaginal disks.

This hypothesis is tested using a special stock which produces gynandromorphs with a high frequency. Certain female zygotes in this stock have an unstable ring X chromosome ($X^{c2}$ $In(1)w^{vC}$, $w^{vC}$, f) which may be lost at the first meiotic division in the egg or at any later nuclear division. The genes on the remaining X chromosome are thus unmasked in a hemizygous condition. For example, dor car/$X^{c2}$ $In(1)w^{vC}$, $w^{vC}$ f female zygotes will express the dor car phenotype in any tissues which are derived from a cell line having lost the unstable ring X chromosome.

The mating scheme used to produce the type of female zygote in the above example follows.

Balanced lethal stock

FM-6, $y^{31d}$ dm B/y dor car x FM-6, $y^{31d}$ dm B

FM-6, $y^{31d}$ dm B/y dor car x $X^{c2}$ $In(1)w^{vC}$ $w^{vC}$ f/Y

Type A ♀ y dor car/$X^{c2}$ $In(1)w^{vC}$

Type B ♀ FM-6, $y^{31d}$ dm B/$X^{c2}$ $In(1)w^{vC}$ $w^{vC}$ f

The yellow alleles in type A and B females makes it possible to recognize any male cuticular tissues in the resulting gynandromorphs. In type A zygotes dor car male tissue should result only from imaginal disks in which this gene combination is viable. Type B zygotes act as controls in the sense that any gynandromorphic patterns possible should be unrestricted in their appearance among these zygotes. The results of this experiment are shown in Table 1.

Table 1. A comparison of gynandromorphic tissue patterns between type A and type B female zygotes.

| | Total No. of flies | Total No. of gynandromorphs | Hemizygous tissue found in gynandromorphs | | |
|---|---|---|---|---|---|
| | | | Head | Thorax | Abdomen |
| Type A zygotes | 624 | 24 | 14 | 6 | 9 |
| Type B zygotes | 640 | 69 | 36 | 42 | 38 |

Of the 69 gynandromorphs which appear among the type B adult progeny, the hemizygous FM-6 chromosomal markers express themselves randomly in the head, thorax and abdominal tissues. Only 24 gynandromorphs appeared among the type A adult progeny. This might result from a random killing tendency among all kinds of type A gynandromorphs or from the specific elimination of certain kinds of gynandromorphic patterns. The latter explanation appears to be correct in that complete bilateral gynandromorphs are common in type B adults but absent amoung type A adults. It is also interesting that in the six cases where the whole head of type A adults is dor car no part of the thorax or abdomen is dor car. If only half of the head is dor car then parts of the thorax are sometimes also dor car. The kinds of gynandromorphic patterns among type A adults suggest that the dor car gene combination is lethal due to an abnormal interaction between the different regions of the fly and not due to the lack of development of any single region.

Bennett, J. and J.F. Hughes. Northern Illinois University, DeKalb, Illinois.

Behavioral correlates of the w, $w^+$ gene substitution, observations without ether.

A pair of isogenic, inbred, Oregon-R lines, differing only at the white locus, were examined for behavioral differences. The lines were described more fully in DIS 45: 140-141. In the earlier study ether was used to separate the flies and place them in the small petri-dish type observation chambers. In this study ether was dispensed with and smaller more convenient observation chambers were utilized (Figure 1). These chambers (#BGS4, 16mm dia. x 3mm deep, $3.75/100, The Blister Co., 845 3rd Ave. East, Kalispell, Montana 59901) known as Blister™ slides, have sliding covers over a circular, flat bottomed cavity. It is possible to place a chamber at the end of a vial, shake the fly into the cavity and slide the cover in place before removing the vial. When turned over under a stereoscope the chamber is easily visible and fills the field at 10X magnification.

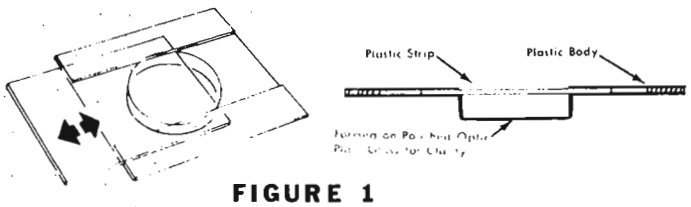



**FIGURE 1**

This procedure made anesthesia unnecessary and allowed direct observations without the ether trauma involved in the earlier study. A preliminary effort with Nitrogen anesthetization indicated that it, too, was traumatic in that many flies emerged with wings locked over their backs and did not recover for several hours. Thus complete avoidance of anesthesia seemed preferable.

Preliminary observations revealed a somewhat different set of characteristic behavioral patterns. Fourteen, many similar to the earlier study, were selected to record. One hundred flies of each sex and of each line were observed for 10 minutes each. An observed behavior pattern was recorded only once in each observation period. Flies were generally from two to four days of age and from stock cultures.

Of the observed traits five displayed significant differences in frequency between lines, or sexes, or both. Rubbing Forelegs: The fly stands on rear four legs and rubs front legs together rapidly. Rubbing Antenna: The fly used one front leg to brush antenna from base to tip many times in rapid succession. Rubbing Head and Eyes: The fly turns head and brushes top of head and eye and the neck region with one foreleg. Combs Wings: The fly stands on five legs, depresses tip of abdomen, combs top of wing with rear leg on the same side. Pulls Anus: The fly stands on front four legs uses rear pair of legs to pull anal region posteriorly.

The table indicates the observed numbers and the $\chi^2$ probabilities for the differences, either between lines $w^+$ & w, or between sexes (across lines). In addition an activity value

| Line & Sex | Rub Forelegs | Rub Antenna | Rub Head | Combs Wing | Pulls Anus |
|---|---|---|---|---|---|
| $w^+$ ♀ | 98 | 87 | 83 | 75 | 1 |
| $w^+$ ♂ | 100 | 77 | 78 | 78 | 1 |
| w ♀ | 92 | 77 | 80 | 72 | 1 |
| w ♂ | 87 | 59 | 55 | 62 | 14 |
| w vs $w^+$ | <0.001 | <0.004 | <0.002 | <0.04 | <0.004 |
| ♂ vs ♀ | | <0.001 | <0.002 | | |

was obtained by summing all recorded activities. This score showed significant difference between the sexes but not between lines.

The wing combing behavior is closely similar to the earlier study. There does not seem to be any other parallelism with the behavior following etherization. In either case it appears that the substitution of the w gene for its normal allele does result in a number of measurable changes in behavioral tendencies. In several of the measures it appears to result in a lessening of the probability of the activity, and usually a more extreme reduction among males than females.

Hoenigsberg, H.F. Universidad de los Andes, Bogotá, Colombia. A new environmental variable that changes the rate of development of some members of the willistoni group of species.

The usual laboratory conditions for Drosophila paulistorum cluster of species (Dobzhansky and Spassky 1959), D. willistoni, D. tropicalis and D. equinoxialis, have been up to now cultivated in the same banana-agar medium utilized to grow and study other species of the genus Drosophila. In our laboratory as elsewhere little attention has been paid (see recent issues of Evolution and Genetics) to culturing conditions. Most geneticists and zoologists in general think that to speak of the adaptive optimum or more specifically of Darwinian fitness (any component can be drastically affected by different culturing conditions as one can see in what follows) do not implicate the judicious testing of those laboratory conditions that will show the most abundant egg laying and larval survival the species or the simispecies have. Thus testing how natural selection changes the paths of gene frequencies under environmentally poor conditions brings forth very biased results and conclusions, in some cases producing even contradictory data of what the gene pool can do to approximate the average phenotype to a particular ecological exigency. Very often while studying the environmental variance in D. willistoni (experiments on the genetic load) we were puzzled by the apparent impossibility of reducing it under our laboratory conditions. After testing various ways of doing our crosses, changing incubating practices, rotating our culture media so that each tray could have the same amount of exposure to each condition, and after following the usual statistical recomendations, the environmental variance was still relatively prominent. By simply changing our laboratory culturing medium from bananas to guayaba (Psidium guajaba) we were able to reduce the variance to approximately one fourth of what it was before, and as expected the statistical analysis became twice as effective as before.

The following table shows the rate of development in D. paulistorum, D. willistoni, D. tropicalis and D. equinoxialis, in both banana - agar and guayaba - agar media. The reader can see how the rate of development from egg laying to adult emergence as imagoes were reduced considerably in our new laboratory medium.

Table 1. Rate of development of different species of the willistoni group in different laboratory media. The numbers represent days of development from egg laying to adult emergence from pupae.

| | Banana - agar | | Guayaba - agar | |
|---|---|---|---|---|
| | Pupa | Adult | Pupa | Adult |
| D. paulistorum | | | | |
| Andean | 10 | 16 | 8 | 12 |
| Caripe | 12 | 18 | 8 | 12 |
| Llanos 13A | 10 | 18 | 8 | 12 |
| Transitional | 10 | 16 | 8 | 12 |
| Amazonian | 11 | 16 | 8 | 12 |
| Central American | 11 | 16 | 9 | 12 |
| D. willistoni | | | | |
| Yaguaracaca | 10 | 17 | 7 | 12 |
| Valparaiso | 10 | 17 | 7 | 13 |
| D. Tropicalis | | | | |
| Valparaiso | 10 | 17 | 8 | 13 |
| D. equinoxialis | | | | |
| Mitú 2A | 10 | 18 | 8 | 12 |

Acknowledgment: We wish to thank the Colombian National Science Foundation (Colciencias) for their support.

Reference: Dobzhansky, Th. and B. Spassky, 1959, Proc. Natl. Acad. Sc. 45: 419-428.

McCrady, E. University of North Carolina at Greensboro, North Carolina. Wing disc tracheotomy prior to pupation in D. virilis.

In order to establish the relative roles of the tracheal supply and the hypodermal stalk in the development of the wing disc during metamorphosis, we have compared the results of three distinct types of operations with mock operated controls. Mature third instar larvae of D. virilis were etherized and dissected with a sterile Aloe #115 ultra-micro dissecting hook. The tip of the hook was inserted through the dorso-lateral body wall in the meta-thoracic segment so that four distinct results were obtained:

a) unilateral cutting of the ventral branch of the dorsal segmental trachea, the only tracheal supply to the disc,

b) severance of the hypodermal stalk, including its internal tracheal branch,

c) operations in which both "a" and "b" were accomplished on the same side in one animal,

d) mock operations consisting of insertion of the dissecting needle without the cutting of either the tracheal trunk or the hypodermal stalk.

The preliminary results of these operations are summarized below:

| Operation Category | Number | Survivors (%) | Normal Wings (%) | Abnormal Wings (%) |
|---|---|---|---|---|
| a | 117 | 77(66) | 66(86) | 11(14) |
| b | 108 | 37(34) | 35(94) | 2(06) |
| c | 46 | 19(41) | 12(63) | 7(37) |
| mock | 44 | 41(93) | 40(98) | 1(02) |

A majority of the wing abnormalities found following category "a" and "b" operations consisted of wings which were not properly expanded following emergence. Five operated animals in category "b" which had normal wings lacked one or more macrochaetae, most often the anterior notopleural. The relatively large number of wing abnormalities observed after category "c" operations, in which the mortality was also the highest, consisted of wings in which a major failure of cell differentiation had occurred, similar to earlier results obtained in the transplantation of whole discs into mature larvae[1,2]. Since the majority of operated discs were able to develop normally, it appears likely that the previously observed failure in wing development in transplanted discs could be associated with the metamorphosis of such discs in the absence of the normal association of their thoracic areas with the other discs ordinarily contiguous with them. The nature of this association and its influence are being investigated further. (Supported by grant 153 from the North Carolina Board of Science and Industry).

References: 1) Glancy, E.A. and R.B. Howland, 1938, Bio. Bull. 75: 99-105; 2) McCrady, E., 1970, Amer. Zoologist 10: 320 (Abst.)

Tarantul, V.Z., V.T. Kakpakov and V.A. Gvozdev. Kurchatov Institute of Atomic Energy, Moscow, U.S.S.R. Protein, RNA and DNA synthesis in the established line of diploid cells of Drosophila melanogaster in vitro.

The synthesis and intracellular content of macromolecules were determined in the established diploid line of embryonic cells of Drosophila melanogaster (Genetika, Russ., 1969, 5, 12, 67; DIS 1970 45: 110). The quantity of DNA per diploid cell measured chemically by diphenilamine reaction according to Burton was $1.9 \times 10^{-12}$g. The quantity of RNA and protein per cell was $7\text{-}14 \times 10^{-12}$g and $5\text{-}10 \times 10^{-11}$g correspondingly. Actinomycin D (3 µg/ml) inhibited incorporation of $C^{14}$-uracil or $C^{14}$-uridine to 5-10% of the control. The protein synthesis measured by the $C^{14}$-lysine incorporation stops in 8-9 hours after the actinomycin addition. The half life of messenger RNA evaluated by the actinomycin induced inhibition of $C^{14}$-lysine incorporation is about 3-3.5 hours. The RNA synthesis is resistant to α-amanitin (20 µg/ml), although the same sample of the drug effectively inhibits the RNA synthesis in the isolated rat liver nuclei. The RNA synthesis in presence of rifampicin (100 µg/ml) decreases by 30%. Puromycin (100 µg/ml) inhibits $C^{14}$-lysine incorporation by 85%. After 6-12 hours of puromycin treatment the increase of the number of cells accompanied by the decrease of cell size was observed although the normal mitotic figures were absent. The presence of purumycin leads probably to the abnormal cell division or cell fragmentation. Hydroxyurea in concentrations of 100 µg/ml and 1 mg/ml inhibits the $H^3$-thymidine incorporation to 15% and 3% of the control respectively.

Hoenigsberg, H.F. Universidad de los Andes, Bogotá, Colombia. New culturing conditions for several Drosophila species.

Cheap, safe and easily prepared culturing media is one of the necessary instruments to do field work (experimental stations, collecting work, field research etc.) of population genetics of Drosophila in the tropics. Often the usual banana-agar medium appears contaminated with fungi which impedes the normal growth of first generation larvae. Furthermore, banana-agar, although a good laboratory medium, if periodically seeded with dry yeast, proves to have a hard surface for Drosophila eggs to survive casual mechanical contact when not seeded with yeast. The reason is that timely fermentation propitiates the structure of a soft surface.

The author has made and tested three new culture media where difficulties such as those mentioned above are obviated. Moreover, outside of ground Agar and Tegocept, easily found tropical fruits are the only important ingredients. Common fruits such as those prescribed here are necessary in order to avoid bringing from long distances baskets with bananas to prepare adequate culturing conditions for Drosophila. Bananas are not as easily found in the tropical forest as thought in various genetic laboratories up north.

Table 1 presents the kitchen formulae for those new culturing conditions where papaya (Carica papaja) piña (Ananas sativus) and guayaba (Psidium guajaba) are used instead of bananas. Table 2 presents the list of species tested for adequate growth in such new culturing conditions.

Table 1. Kitchen formulae for three new culturing conditions for several Drosophila species.

| | PAPAYA - AGAR | GUAYABA - AGAR | PIÑA - AGAR |
|---|---|---|---|
| Water | 2000 cc. | 2000 cc. | 2000 cc. |
| Agar | 87.5 gm. | 87.5 gm. | 87.5 gm. |
| Tegocept | 40 cc. | 40 cc. | 40 cc. |
| | 3 medium sized papayas liquified with 100 cc of water. | 30 large sized quayabas liquefied in 250 cc. of water. | 2 small sized piñas liquefied in 45 cc. of water. |

Table 2. A list of species which grow well in the above mentioned agar media.

1. Saltans Group
   - D. saltans
   - D. prosaltans
   - D. sturtevanti
   - D. cordata
   - D. neosaltans
   - D. pseudosaltans
   - D. parasaltans
   - D. subsaltans
   - D. neocordata

2. Willistoni Group
   - D. willistoni
   - D. tropicalis
   - D. insularis
   - D. paulistorum
   - D. equinoxialis

3. Melanogaster Group
   - D. melanogaster
   - D. simulans

Acknowledgment: We wish to thank the Colombian National Science Foundation (Colciencias) for their support.

Oshima, C. and T.K. Watanabe. National Institute of Genetics, Misima, Japan. Sterility genes in natural summer and autumn populations of D. melanogaster.

Frequencies of sterile male and female flies among several hundred flies collected simultaneously from the summer and autumn populations of 1970 in Katsunuma locality were found to be 4.3, 4.8 per cent and 3.4, 7.8 per cent respectively. Among 342 second chromosomes extracted from each male fly in the summer and autumn populations, 69 chromosomes were found to be sterility chromosomes. The results are shown in Table 1.

Table 1. Frequency of sterility chromosomes and frequencies of male, female and both sexual sterility chromosomes

Collection time: 1970 (July, October)

| Class of viability | Semilethal | Subvital | Normal | Total |
|---|---|---|---|---|
| No. of tested chromosomes | 46 | 41 | 255 | 342 |
| No. of sterility chromosomes | 13 | 12 | 44 | 69 |
| Frequency (%) | (28.3) | (29.3) | (17.3) | (20.2) |
| No. of male sterility chromosomes (%) | | 32 | (46.4) | |
| No. of female sterility chromosomes (%) | | 27 | (39.1) | |
| No. of both sexual sterility chromosomes (%) | | 10 | (14.5) | |

The frequency of sterility chromosomes was higher than 12.6% in 1968.

By half diallel crosses between sterility lines, which have been maintained by the Cy-sterility balanced system, the frequency of allelism was determined in the summer and autumn populations as Table 2 shows.

Table 2. Results of allelism between sterility genes

| Collection time | July 31 | | October 12 | |
|---|---|---|---|---|
| Sex | ♀ | ♂ | ♀ | ♂ |
| No. of sterility chromosomes | 11 | 19 | 24 | 23 |
| No. of crosses | 55 | 171 | 276 | 253 |
| No. of allelic crosses | 10 | 1 | 14 | 26 |
| Frequency of allelism (%) | (18.2) | (0.6) | (5.1) | (10.3) |
| Frequency of finding | No. of sterility genes | | | |
| 1 | 6 | 17 | 14 | 10 |
| 2 | - | 1 | 1 | - |
| 3 | - | - | 1 | 2** |
| 5 | 1 | - | 1* | - |
| 7 | - | - | - | 1 |

**, * persistent gene

Table 3. Persistent and allelic sterility genes during two years in Katsunuma locality

| Collection date | Oct. 2, 1968 | July 31, 1970 | Oct. 12, 1970 |
|---|---|---|---|
| Female sterility gene | FS 801 A - F (6 chromosomes) | FS 102 (1 chromosome) | FS 201 A - E * (5 chromosomes) |
| Male sterility gene | MS 801 A - M (13 chromosomes) | MS 113 (1 chromosome) | MS 202 A **<br>MS 202 B·FS 206<br>MS 202 C·FS 217<br>(3 chromosomes) |
| | | MS 114 | MSS 203 A |
| | | MS 103 | MSS 203 B |
| | | MS 102 | MSS 203 C |

:allelic relationship

**, * persistent gene

The sterility strains, which were extracted from a natural population in Katsunuma locality in 1968 and have been maintained in our laboratory, were crossed diallelly with new sterility strains extracted from the summer population of 1970. On the other hand, diallel crosses between the sterility strains of the summer and autumn populations were performed.

One male sterility gene (MS 801) and one female sterility gene (FS 801) have persisted for two years. The male sterility genes (MS 202, B, C) in the autumn population of 1970 were linked with different female sterility genes (FS 206, FS 217). Three different male sterility genes in the summer population of 1970 were found to be combined in double sterility chromosomes (MSS 203 A - C). On the other hand, a female sterility gene (FS 102) has increased in the autumn population. The results are shown in Table 3.

The breeding pattern of D. melanogaster in Katsunuma locality has been scarcely known, but the places of hibernation and the dispersion of flies were probably localized in the district.

Ehrie, M.G. and R.C. King. Northwestern University, Evanston, Illinois. The anatomy of the larval ring gland of Drosophila melanogaster and its associated organs.

During histological processing Drosophila larvae are generally punctured to insure penetration of the fixative. The drop in hydrostatic pressure caused by puncturing often alters the three dimensional interrelations of adjacent organs. In 1966 F.G. Gottlieb published the details of a procedure that avoids puncturing (J.Roy.Mic.Soc. 85: 369-373). We have adapted this technique for plastic embedments. Larvae were relaxed, fixed, and dehydrated following Gottlieb's directions. The Dioxane used as the dehydrating solvent was replaced by propylene oxide, and the larvae were then infiltrated with the following resin mixture: Araldite 502 monomer, 8 ml; dodecenyl succinic anhydride hardener, 8.5 ml; and DMP-30 accelerator, 0.32 ml. The monomer and hardener must be mixed thoroughly before the accelerator is added. All components were obtained from Polysciences, Inc., Paul Valley Industrial Park, Warrington, Pa. 18976. Larvae were infiltrated with resin using the following schedule: 12 hours each in (1) 3 parts propylene oxide (P): 1 part resin mixture (R), (2) 1 P:1 R, (3) 1 P:3 R, and (4) 100% R. The specimens were transferred to embedding capsules containing fresh resin and were left to polymerize at 60°C, for 24 hours.

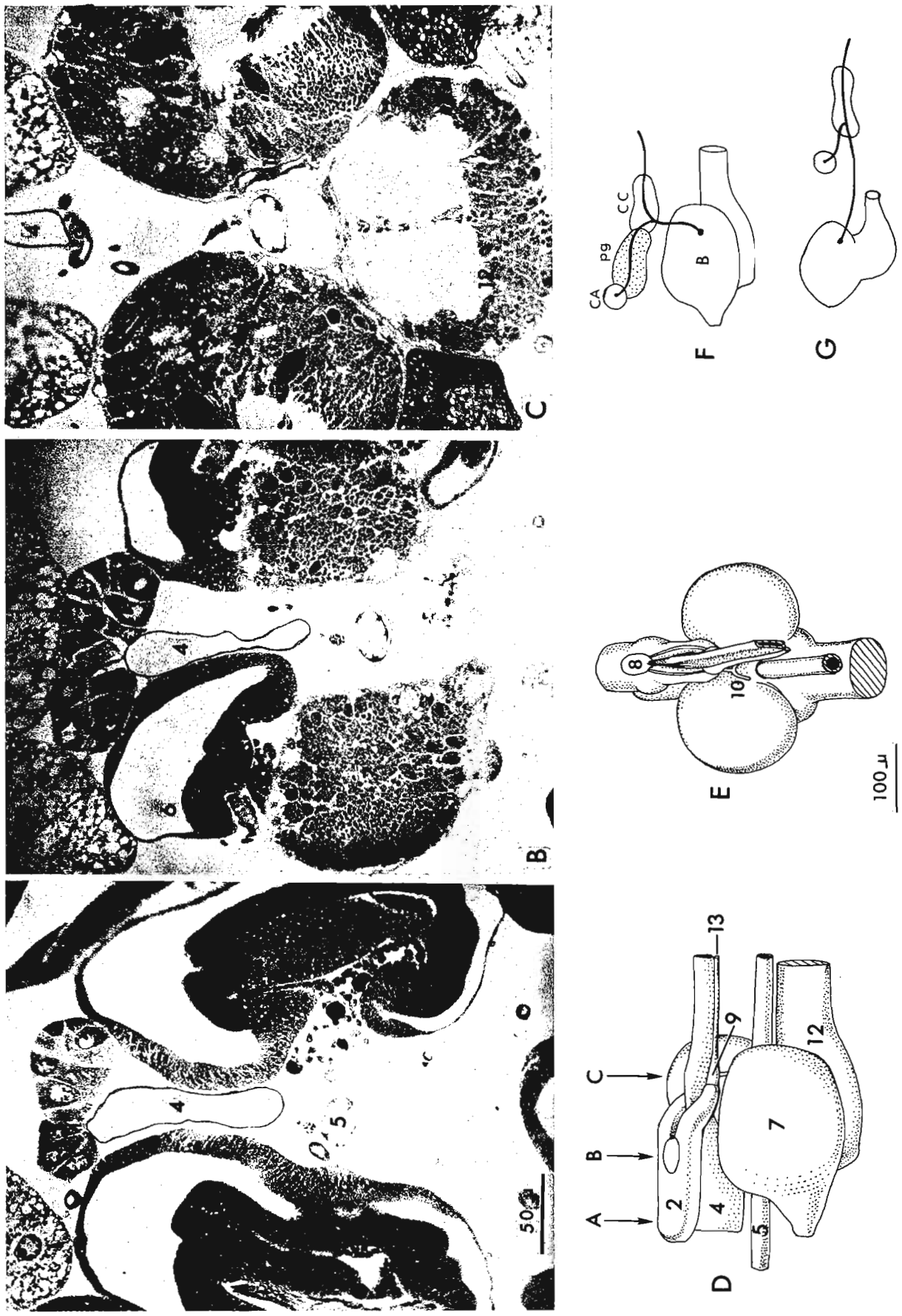
The female sectioned was in the terminal portion of the third instar (92 hrs. after hatching). One micron sections were cut with glass knives mounted in a Leitz Fernandez-Moran microtome. The serial transverse sections were stained with Azure B. Outline drawings of the sectioned brain, ring gland, aorta, and oesophagus were made on sheets of cardboard using a Wild M20 microscope equipped with a drawing tube. The tracings were cut out and glued together to form a three dimensional model of these organs at 350X.

The accompanying photomicrographs and drawings illustrate the results. The following labeling system is used: 1. fat body cell; 2. prothoracic gland cell; 3. tracheal cell; 4. aorta; 5. oesophagus; 6. antennal imaginal disc; 7. brain hemisphere; 8. corpus allatum cell; 9. corpus cardiacum; 10. afferent nerve to corpus cardiacum; 11. salivary gland cell; 12. ventral ganglion; 13. efferent nerve from corpus cardiacum. (See next page.)

The ring gland lies above the brain, straddling the aorta (Figs. D and E). Fig. A shows a section through the anterior portion of the ring which contains prothoracic gland cells clustered in paired longitudinal strips along the dorsal surface of the aorta. Two tracheae enter the prothoracic gland to the left and right where its ventral lateral surfaces rest upon the paired antennal discs (Figs. A and B). The anterior tips of the brain hemispheres and the corpus allatum are included in the section shown in Fig. B. The ventral ganglion and the corpus cardiacum are included in section shown in Fig. C. An afferent nerve to the corpus cardiacum is sectioned where it leaves the right brain hemisphere. It is joined by a nerve from the left hemisphere (Fig. E). A left and right nerve leave the corpus cardiacum and pass through the arms of prothoracic gland to the corpus allatum.

The positions of the brain (B), corpus allatum (CA), and corpus cardiacum (CC) are contrasted for the larva and adult in diagrams F and G. During metamorphosis the prothoracic gland (pg) degenerates and the corpus allatum and corpus cardiacum move posteriorly and ventrally relative to the brain.

(M.G. Ehrie held an Undergraduate Research Participation Award from the N.S.F. during the summers of 1968 and 1969.)


A
B
C
D
E
F
G
2
4
5
7
9
12
13
8
10
CA
pg
CC
B
50 µ
100 µ

Baird, M.B., H.V. Samis and H.R. Massie. Masonic Medical Research Laboratory, Utica, New York. Changes in Drosophila catalase activity associated with preadult development.

Catalase (EC 1.11.1.6) activity was determined in preadult D. melanogaster at various time intervals after oviposition at 25°C. Ore-R females were permitted to lay eggs on enriched yeast plates for four-hour intervals, following which the eggs were transferred to standard corn meal-agar-molasses medium (1). This technique results in cultures of developing flies which are temporally synchronous within ± 2 hours.

Samples of eggs were collected directly from the yeast plates at the indicated times after oviposition and washed thoroughly with insect saline to remove yeast and media contaminants. Samples of larvae were collected by flotation in 1M NaCl, followed by thorough rinsing with insect saline. Samples of pupae were manually collected from the sides of the culture bottles.

All samples were homogenized in water, and dechitinized by a filtration technique described elsewhere (2). Catalase assays were performed at 22.5°C by modification of the spectrophotometric technique of Price et al. (3), utilizing a Perkin-Elmer 139 spectrophotometer equipped with an externally thermostated constant temperature cuvette chamber. 0.050ml of sample was added to a cuvette containing 3.0ml of 0.02M phosphate buffer, pH 6.8. The cuvette was blanked to zero absorbance, and 0.030ml of 0.98M hydrogen peroxide was added to the cuvette. Helium gas was immediately bubbled through the contents of the cuvette for five seconds, and the disappearance of hydrogen peroxide was recorded at 230 mμ for an additional 30 seconds with a chart recorder. Units of catalase were calculated as described by Lück (4), and protein was determined by the method of Lowry et al. (5).

No appreciable catalase activity was found in Drosophila embryos [Fig. 1]. However,

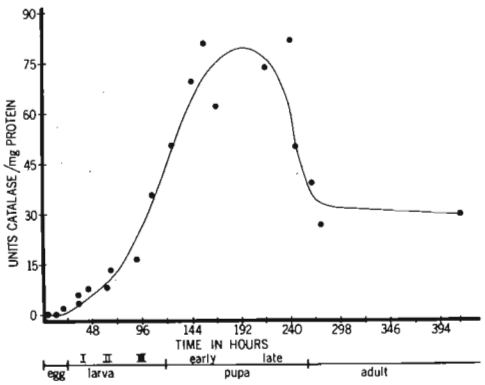



Figure 1. Catalase activity in preadult D. melanogaster at various times after oviposition at 25°C. Data expressed as units/mg. protein, where one unit is that amount of enzyme necessary to liberate half the peroxide oxygen from a hydrogen peroxide solution of given concentration in 100 seconds at 22.5°C.

appreciable enzyme activity appeared during the early larval stages of development, increasing 70-fold to maximal activity during mid-pupal development. This increase was followed by a sharp decline in enzyme activity prior to eclosion.

These preliminary results indicate a stage specific activation (e.g. differential gene action) of those genes in D. melanogaster which code for the normally ubiquitous catalase.

References: 1. Ursprung, H. 1967. In Methods in Developmental Biology (F.H. Wilt and N.K. Wessells, eds), Thomas Y. Crowell, Co., New York, p486; 2. Samis, H.V., Jr., and F.C. Erk. 1969. DIS 44:132; 3. Price, V.E., Sterling, W.R., Tarantola, V.A., Hartley, R.W., Jr., and Rechcigl, M., Jr. 1962. J. Biol. Chem. 237:3468; 4. Lück, H. 1965. In Methods of Enzymatic Analysis (H. Bergemeyer, ed), Academic Press, New York, p885; 5. Lowry, O.H., Rosebrough, N.J., Farr, A.L., and Randal, R.J. 1951. J. Biol. Chem. 193: 265.

Minamori, S. and K. Ito. Hiroshima University, Hiroshima, Japan. Effects of delta on fertility in D. melanogaster.

The $ID^b$-45 chromosome line usually carries an appreciable amount of delta b, but it is not susceptible to the killing action of this delta (Minamori et. al. 1970). The productivity of this line was examined when it carried various amounts of delta b. More than one-third of $Cy/ID^b$-45

males and females tested became sterile when they carried cytoplasm of Cy/Pm stock which is considered to carry no delta b. The number of progeny was smaller when flies of this line were raised at 25° C than raised at 28° C at which temperature the multiplication of delta is accelerated. The progeny number was appreciably reduced when the flies were raised at 18° C at which temperature the multiplication of delta is suppressed. This line could not be maintained at that temperature, since both males and females became sterile (Table 1).

Table 1. The number of progeny (average) recovered from Cy/ID$^b$-45 flies which were raised for successive generations at 18° C

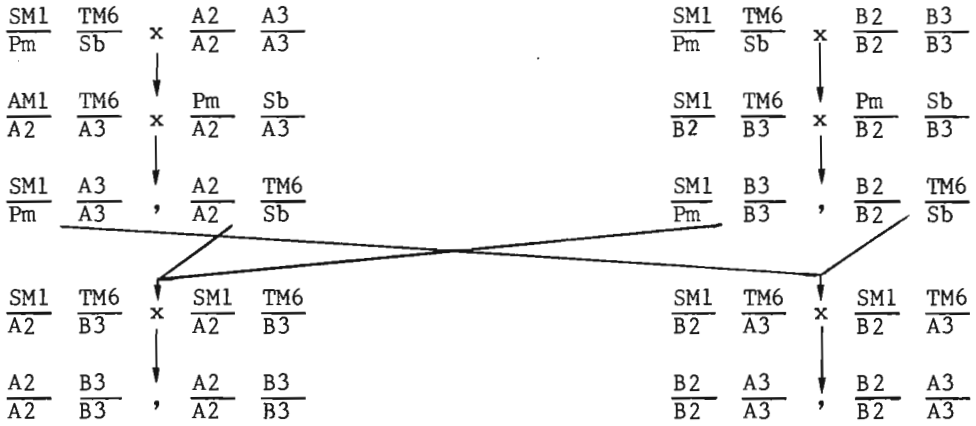
| Subline | Raising temperature for progeny (C) | Generations raised at 18° C | | | |
|---|---|---|---|---|---|
| | | 1 | 2 | 3 | 4 |
| 0-9 | 18° | 0 | - | - | - |
| | 25° | 0 | - | - | - |
| y-9 | 18° | 5.9 | 15.0 | 3.4 | 0 |
| | 25° | 33.5 | 0 | - | - |

Thus, the conclusion drawn may be that the presence of an appreciable amount of delta b is necessary for the gametogenesis of the Cy/ID$^b$-45 flies.

Reference: Minamori, S., Fujioka, N., Ito, K., and Ikebuchi, M. 1970. Evolution 24: 735-744.

Moree, Ray. Washington State University, Pullman, Washington. A method for the construction chromosomal interchange lines.

The following scheme has been found useful for the construction of chromosomal interchange lines used in heterozygosity studies, where only the 2nd and 3rd chromosomes are interchanged.

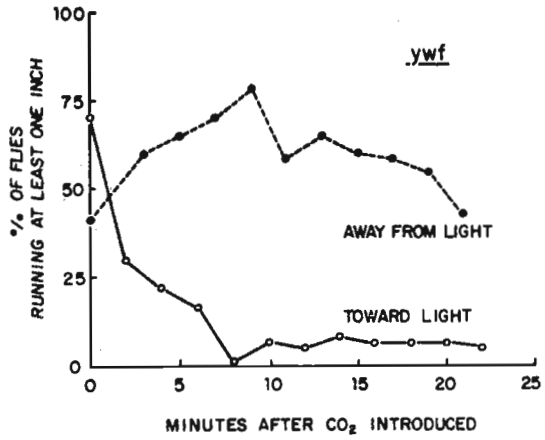


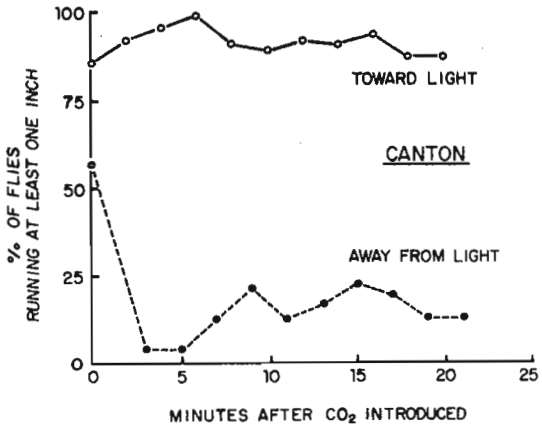

A and B designate different wild type stocks; 2 and 3 designate chromosomes 2 and 3. All other chromosomes are those described in Lindsley and Grell (Carnegie Institution of Washington Publication No. 677, 1968) except that TM6, obtained from E.B. Lewis, has a new marker, Ubx$^{P15}$. Males used in the fourth cross can of course carry Pm instead of SM1 and Sb instead of SM1 and Sb instead of TM6, which sometimes makes this cross easier to set up. The X chromosomes consist of material from the double balancer line, from line A, and from line B in the approximate ratio of 4:1:1, respectively. If lines A and B are made isogenic prior to making the interchanges, then maximum heterozygosity contrasts are possible. (Aided by funds from the State of Washington Initiative Measure No. 171 for the Support of Biological and Medical Research.)

Kaplan, W.D., and B. Hanstein. City of Hope National Medical Center, Duarte, California.
A mutant stock showing negative phototaxis in the presence of $CO_2$.

Flies of a ywf stock carried in this laboratory for the past several years show a normal response to light. Placed in a 5-7/8 inch test tube containing air, and given one minute to run horizontally toward a fluorescent light, 70% move at least one inch from the bottom of the tube toward the source of the light in an otherwise darkened room. 85% of the flies of a Canton-S stock exhibited this positive phototactic response.

When placed in an atmosphere of 20% carbon dioxide, however, the ywf stock responds by moving away from the light source. Placed at the end of the tube closest to the light, they begin to move away from the light. At nine minutes after the introduction of $CO_2$, 78% move at least one inch away from the light in a minute. At 8 minutes less than 10% run one inch toward the light. This is in contrast to the Canton-S stock which in the presence of 20% $CO_2$ continues to run toward the light and not run away from it. (see graphs). The zero




minute point on the graph is before $CO_2$ was introduced and the flies are in air. The curves are for the same groups of flies run on alternate minutes toward and away from light, and given one minute to run. Total number of flies in the four ywf groups was 60, and in the four Canton-S groups, 61.

White-eyed flies of a bw st stock, and just w by itself did not show this reversal. The reason for this behavior is currently under investigation.

Supported by N.I.H. grant No. NS08014.

Lefevre, G., Jr. San Fernando Valley State College, Northridge, California.
A cytological analysis of X-ray-induced recessive sex-linked lethals.

Salivary chromosome preparations of sex-linked lethals recovered following exposure of mature sperm from wild-type males to 2000r and 3000r doses of X rays were analyzed for the presence of detectable rearrangements in the euchromatic portion of the X chromosome (1A through 19F). Among 190 lethal X chromosomes recovered in the 2000r experiment, 70 (36.8%) carried rearrangements; among 125 lethals from the 3000r experiment, 55 (44.0%) were detectably abnormal. These values are not significantly different. It would appear that, insofar as mature sperm are concerned, the proportion of recovered X-ray-induced lethal effects that are associated with rearrangements is not dose dependent.

Ouweneel, W.J. Hubrecht Laboratory, Utrecht, Netherlands. Homoeotic mutants in Drosophila: interaction during development.

A given mutation not only affects a specific type of normal tissue, but also the same kind of tissue whenever this arises at an abnormal location in the fly under the influence of a homoeotic mutation. For instance, dachs not only shortens the normal legs but also the legs formed instead of the aristae in aristapedia ($ss^a$);[1] multiple-wing-hairs not only effectuates a characteristic hair pattern on the normal wing but also on the wing outgrowths appearing in the eyes under the influence of ophthalmoptera (opht).[2] It turns out that also homoeotic mutations themselves affect allotypic tissues produced by other homoeotic mutations. Three classes of genetic combinations are suitable to demonstrate this type of interaction. First, $ss^a$ was found to change not only the normal arista, but also the arista produced from the proboscis by proboscipedia (pb) (at low temperature) into a tarsus, whereas pb has no influence on the antenna. This confirms earlier results of Vogt,[3] although I feel her explanation (sequential activity of $ss^a$ and pb) is irrelevant. Secondly, bithorax (bx) changes the haltere into a wing-like structure, while tetraltera (tet) strongly reduces the wing and produces a hypodermal (leg-like?) protrusion along with, or instead of the wing. In bx tet combinations tet seems to reduce not only the normal wings but also the wing-like structures produced by bx; however, no leg-like structures replacing the halteres were observed so far. The same class of interactions is exemplified by the effect of Contrabithorax, which heterotopically changes the posterior mesothorax produced by bithoraxoid from the first abdominal segment into a posterior metathorax.[4] Thirdly, in combinations of tet with either eyeless-opht or loboid-opht it was found that tet not only affects the normal wings but also changes the wing-like outgrowths from the eye area produced by opht into hypodermal bristle-bearing protrusions. Therefore, homoeotic mutations act not only in one specific imaginal disc, but also at any other place in the developing organism where the genome allotypically determines a tissue to follow a given developmental pathway with which the mutations concerned can interfere.

References: 1. Braun, W. 1940, Genetics 25: 143-149. 2. Ouweneel, W.J. 1970, Genetica 41: 1-20. 3. Vogt, M. 1946, Z. Naturforsch. 1: 469-475. 4. Lewis, E.B. 1963, Am. Zoologist 3: 33-56.

Krimbas, C.B. Agricultural College of Athens, Athens (Votanikos), Greece. Gene arragement frequencies in Pindos population of D. subobscura.

A small sample of D. subobscura taken in the summer of 1968 in a Quercus forest near the village of Korydallos, 34 km from Kalambaka, on the road of Kalambaka-Metsovon, in Pindos Mt. was analysed for the gene arrangement frequencies in all five chromosomes, by crossing wild males and sons of wild females to a standard strain. Table I reports these frequencies as well as the numbers of chromosomes studied. This is the first sample reported from the West of North Greece and does not differ strikingly from the mainland (North and South) Greek samples. It seems that local differentiation between not very far remote populations of D. subobscura is not great.

Table I

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| $A_{st}$ .33 | $A_I$ .38 | $A_2$ .29 | N 58 | | | | |
| $J_I$ .74 | $J_{3+4}$ .01 | $J_{St}$ .25 | N 85 | | | | |
| $E_{1+2+9}$ .58 | $E_{st}$ .19 | $E_8$ .19 | $E_{1+2}$ .03 | $E_{1+2+9+12}$ .01 | N 86 | | |
| $U^*_{1+2+6}$ .54 | $U_{1+2}$ .29 | $U_{st}$ .13 | $U_{1+2+8}$ .02 | $U_{1+2+7}$ .02 | N 84 | | |
| $O_{3+4}$ .48 | $O_{3+4+1}$ .21 | $O_{st}$ .12 | $O_{3+4+22}$ .06 | $O_{3+4+7}$ .02 | $O_{3+4+2}$ .06 | $O_{3+4+2}$ .05 | N 87 |

*$U_{1+2+4}$ included

Khishin, A.F. and M.M. Megaheid. University of Assuit, U.A.R. Storage of germ cells and process of mutation in Drosophila melanogaster.

This research is designed to study the effects of storing Drosophila melanogaster male germ cells on the spontaneous and induced sex-linked and second chromosome lethal mutations. Three different storage periods of 3, 6 and 9 days were used. Adult males 3 days old were irradiated with 2352 r of X-rays.

The Muller-5 (M-5) and the Curly Lobe (Cy/L) methods were used for the determination of the mutation rates for sex-linked and second chromosome lethals respectively. The result obtained suggests that:

The frequency of the spontaneous recessive sex-linked lethal mutations are not different statistically for different storage periods, or when compared with the unstored.

X-ray induced sex-linked lethal mutations may increase slightly after storing male germ cells in untreated females for 3, 6 and 9 days; the difference, however, is not statistically significant.

The percentage of spontaneous second chromosome lethals increases by storage. The difference is significant when storage continues for 6 and 9 days.

Storage of irradiated sperm for different periods increases the rates of second chromosome lethals over the rates obtained from the unstored irradiated sperm.

The effect of storage is more pronounced in the case of irradiated than in the case of untreated sperm.

The present study shows that the ratio between the induced sex-linked and second chromosome lethals increases by sperm storage.

Rosenfeld, A., A. Carpenter, and L. Sandler. University of Washington, Seattle, Washington. A nonchromosomal factor causing factor causing female sterility in D. melanogaster.

A homozygous pr cn stock in our laboratory appears to carry a nonchromosomal factor which will sterilize females that carry specific chromosomes contributed by the male parent. This system has features similar to the case of $CO_2$ sensitivity studied by L'Héritier and his collaborators and to the delta-factor-induced lethality studied by Minamori, and is perhaps the same as the "maternally inherited factor" reported by Picard and L'Héritier (DIS 46:54, 1971).

The standard experiment, here, is to cross the two stocks to be examined such that each serves as female parent. $F_1$ females are tested for fertility by crossing to Canton-S ♂♂. $F_1$ daughters of pr cn mothers are fertile when the male is pr cn, Canton-S, or Muller-5; sterile when the male is $y^+$; abo/Cy or Muller-$5_A$ (=Muller-5/$y^+Y$; +/+; +/+; $spa^{pol}/spa^{pol}$ ♂♂ from a stock kept as Muller-5/Muller-5; Sm1/+; +/+; $spa^{pol}/spa^{pol}$ x Muller-5/$y^+Y$; Sm1/+; Ly Pr/+ +; $spa^{pol}/spa^{pol}$) and Cy daughters are fertile, but $Cy^+$ daughters are sterile, when the male parent is y; abo/Cy. All other pairwise crosses gave fertile $F_1$ females (Muller-$5_A$ was not tested with $y^+$; abo/Cy, y; abo/Cy, or Muller-5).

These data indicate: (1) that both parents much contribute something to the female-sterility phenotype; (2) that chromosome 2 may be of especial importance (from the results of pr cn ♀♀ x y; abo/Cy ♂♂), and (3) that abo (Sandler, Genetics 64:481-493, 1970) is not specifically involved (from the results of pr cn ♀♀ x Muller-$5_A$ ♂♂).

To examine the nature of the maternal contribution from the pr cn ♀♀, $F_1$ ♀♀ from the crosses: (A) pr cn ♀♀ X Muller-5 ♂♂ and (B) pr cn ♀♀ x Canton-S ♂♂ were crossed to $y^+$; abo/Cy ♂♂ and $F_2$ ♀♀ tested for fertility (by mating with Canton-S males). In cross A, 52 B; $Cy^+$ and 50 $B^+$; $Cy^+$ $F_2$ females were tested and all were sterile; in cross B, 59 $Cy^+$ $F_2$ females were tested and all were sterile. The pr cn maternal contribution, therefore, appears to be non-chromosomal since 1/16 of the sterile females should have received no chromosomes from the pr cn stock (except for the B; $Cy^+$ ♀♀ from cross B, where 1/8 would lack such chromosomes).

Further evidence on the nature of the non-chromosomal element in the pr cn stock comes from the results of the following experiment. $F_1$ females from a cross of pr cn ♂♂ by Canton-S ♀♀ were backcrossed to pr cn males. From this cross, 173 $F_2$ females were crossed to $y^+$; abo/Cy males and the $F_3$ $Cy^+$ female progeny tested for fertility. In these crosses, pr cn ♀♀ were not involved; nevertheless 21 females were sterile, 81 were semisterile (producing one or occasionally two larvae), and only 71 were normally fertile. These data strongly suggest some transmission of the nonchromosomal element through the sperm and the existence of quantitative effects; the parallel with the unstable state of sigma in the $CO_2$ sensitivity system is striking.

Band, H.T. Michigan State University, East Lansing, Michigan. Four decades of natural selection.

Darwinian natural selection typically implies directional selection. To date reported instances of genetic changes brought about by directional selection pressures exerted by climatic changes have been rare. In biology, man is still considered to be the primary agent effecting environmental changes (Crow, 1971). As noted by Lamb (1966) between World War I and World War II it was widely believed that climate was static except on the geological time scale. This was the period of classical genetics and the development of classical mathematical population genetics, which was incorporated into population ecology.

The study of the South Amherst, Mass. D. melanogaster natural population now emerges as the study of a population in a slowly changing climate. Regular shifts in decade averages for daily temperature range in summers (Band, 1971) reflect a trend toward more days in the wider range categories.

| | 1930's | 1940's | 1950's | 1960's |
|---|---|---|---|---|
| Narrow (2-20°F) | 334 | 289 | 261 | 225 |
| Intermediate (21-25°F) | 243 | 254 | 248 | 234 |
| Wide (26-44°F) | 343 | 377 | 411 | 461 |

Periods of genetic changes reflecting changes in lethal and semilethal frequencies are: 1938-1946, 1947-1961, 1962-1966, post-1966. In the first, second chromosome lethal and semilethal frequencies fluctuated around 48.8%, in the second around 33-34%; the third was marked by increased developmental homeostasis and resistance of le + sle frequency to decline followed by plunge to 16-17%. Post-1966 le + sle frequency has been rising. The number of days in the different temperature range categories in the different periods in summers are given in Table 2.

Table 2. Mean number of days in different temperature range categories in the different periods of genetic changes in the Amherst D. melanogaster population.

| | 1930-1946 | 1947-1961 | 1962-1966 | 1966-1969 |
|---|---|---|---|---|
| Narrow | 32 | 26 | 19 | 25 |
| Intermediate | 25 | 25 | 21 | 24 |
| Wide | 35 | 41 | 52* | 43 |

*P<05 that significantly more days are in the wide range category. This is the case for all 5 summer.

Heterozygotes containing drastics have been found to have higher viability in narrow temperature ranges (Band, 1963; Oshima, 1969), those free of drastics to have higher viability in wide range conditions (Band, 1963, 1969). The behavior of the population 196?-1966 has provided an example of genetic homeostasis and population adaptation to a more severe climate (Band, 1971). The environmental data thus give support to the hypothesis that selection can be disruptive within summers, directional over the longer term (Band, 1971). They also provide further evidence that such recessive deleterious variants may actually be adaptive in heterozygous condition, hence are maintained in the population in response to the dynamic environment.

References: Band, H.T. 1963. Evolution 17:307-319; Band, H.T. 1969. Japan. J. Genet. 44, Suppl. 1; 200-208; Band, H.T. 1971. American Naturalist (in press). Crow, J.F. 1971. BioScience 21:107. Lamb, H.H. 1966. The Changing Climate (Methuen and Co., Ltd, London). Oshima, C. 1969. Japan. J. Genet. 44, Suppl. 1:209-216.

Band, H.T. Michigan State University, East Lansing. On the negative relation between summer rainfall and average daily temperature range.

Genetics studies on recessive lethal and semilethal frequencies in the S. Amherst D. melanogaster population have usually been made in the Fall until the discovery of a breeding site enabled investigations throughout the breeding season, May-October (Ives, 1970). A negative significant relationship between lethal and semilethal frequencies and average daily temperature range of the week prior to collection (Band and Ives, 1961) and a positive significant relation between these frequencies in Fall and total summer rainfall (Band and Ives, 1968) has been observed. Studies on the relation between climate and genetic changes further indicated changes in general level of precipitation and average daily temperature range had occurred several times in the past 40 years, May-October, although decade averages for summer temperature range increased regularly (Band, 1971). To explore the rainfall-average daily temperature range relation in summer, data back to 1889 have been analyzed.

Table 1 shows a negative relation between these two climatic variables. Rainfall declines spanning 4 or more summers are noted in the 1890's, 1910's, and 1960's. To a lesser extent rainfall in the 1930's was also reduced. Rowan (1954) reports that although the Bruckner cycle has a periodicity of approximately every 35 years, variations from 20 to 50 years are noted. Average maximum daily temperature can be judged to be about 1°F higher at the Amherst College Weather station (data past Nov. 1948) than at the old U. Mass. weather station site (1889- Nov. 1948) which was moved in 1960. Although low rainfall in summers was most prolonged in 1907-1913, Ives' father recalled that Spring rains and cooler summers mitigated against drought. The number of days with 90° maxima or above have increased since the 1930's.

Table 1. Summer rainfall, average daily temperature range and average daily maximum temperatures in the same season, Amherst, Mass., 1889-1970

| Interval | no. years | Rainfall in inches | Daily T° range | Max T° |
|---|---|---|---|---|
| 1889-1892 | 4 | 14.71 | 22.5 | 79.0 |
| 1893-1896 | 4 | 8.96 | 24.7 | 80.9 |
| 1897-1906 | 10 | 14.23 | 23.0 | 79.2 |
| 1907-1913 | 7 | 8.15 | 25.4 | 80.7 |
| 1914-1922 | 9 | 13.09 | 22.6 | 79.7 |
| 1923-1924 | 2 | 6.35 | 26.3 | 81.4 |
| 1925-1928 | 4 | 13.45 | 21.9 | 78.2 |
| 1929-1936 | 8 | 10.11 | 23.3 | 80.7 |
| 1937-1946 | 10 | 12.74 | 22.7 | 80.6 |
| 1947-1961 | 15 | 11.40 | 24.0 | 82.0 |
| 1962-1966 | 5 | 8.60 | 26.1 | 82.1 |
| 1967-1970 | 4 | 12.60 | 4.0 | 82.2 |

Table 2 shows decade averages for daily temperature range in summers since the 1890's. Singer (1970) has commented that nothing is known about climatic stability. Although an apparent stability of temperature range is noted in the first few decades of recorded observations in that area, the data in Table 1 indicate there have been fluctuations in temperature range in association with rainfall levels. We may only speculate that had the tools for Drosophila genetics research been available then, genetic changes in the population in relation to climatic shifts might have been observed in the population as have been witnessed since studies began on recessive lethal and semilethal frequencies in 1938.

Table 2. Average daily temperature range per summer in the past 8 decades in Amherst

| 1890's | 1900's | 1910's | 1920's | 1930's | 1940's | 1950's | 1960's | 1970's(?) |
|---|---|---|---|---|---|---|---|---|
| 24.0 | 23.9 | 23.8 | 23.3 | 22.5 | 23.3 | 24.1 | 24.9 | 25.7(?) |

References: Band, H.T. 1971. Amer. Nat. (in press); Band, H.T. and P.T. Ives. 1961 P.N.A.S., Wash. 47:180-185; Band, H.T. and P.T. Ives. 1968. Evolution 22:633-641; Ives, P.T. 1970. Evolution 24:507-527; Rowan, W. 1954. J. Wildl. Mgt. 18:52-60; Singer, E.F. 1970. Science 170:125 (editorial).

Band, H. T. Michigan State University, East Lansing, Michigan. Was there a drought in the northern U.S. in the 1960's?

Genetic changes in the South Amherst, Mass. D. melanogaster population in the 1960's appear to have been initiated after the onset of a severe decline in rainfall, July, 1961, which lasted to June, 1966. In a study of meteorological drought in Michigan, Strommen, van den Brink and Kidder (1969) commented that drought had been an increasing problem in the Northeast in the 1960's. Their study over the past 4 decades indicated prolonged drought effects in both the 1930's and 1960's in many areas of the state. The 1930's drought appears more evident in Michigan data than in Amherst, Mass. data.

Kendeigh (1961) indicates a low rainfall cycle has been observed and effects on duck numbers noted. Bruckner (1890) detected a cycle approximately every 35 years in data back to the 1700's. Rowan (1954) correlated low duck numbers in the 1820's, and 1860's, and 1890's and 1930's with this cycle. Rowan had been at the University of Alberta, Edmonton. A study recently compiled by the U.S.D.I. (1971) indicates that drought was widespread in the prairie provinces of Manitoba, Saskatchawan and Alberta in Canada and upper Great Plains states of Montana, the Dakotas and western Minnesota in the late 1950's and early 1960's, and again in the summer and fall of 1967. About 50-75% of the important game ducks come from this region. Duck numbers, 1955-1970, appear to follow the effects of the drought on numbers of suitable prairie pothole habitats. The following table has been compiled from the survey of breeding size of populations reported annually for the past 15 years and included in the report.

Mean breeding size of duck populations for the past 15 years. Numbers in millions.

| 1955-1960 | 1961-1965 | 1966-1968 | 1969-1970 |
|---|---|---|---|
| 42462.3 | 31254.6 | 34605.3 | 42355.0 |

Duck breeds surveyed include mallards, gadwall, American widgeon, green winged teal, blue winged teal, shoveler, pintail, redhead, canvasback and scaup.

The continuing relationship between drought cycle and duck numbers in that area indicates that population numbers do not fluctuate at random, somewhat at variance with the conclusions of Cole (1954). It also raises the question, was there a widespread reduction in rainfall throughout the northern United States in the 1960's?

References: Bruckner, E. 1890. Klimaschwankungen seit 1700. Vienna; Cole, L.C. 1954. J. Widl. Mgt. 18:2-24; Kendeigh, S.C. 1961. Animal Ecology (Prentice-Hall, Inc., Englewood Cliffs, N.J.); Rowan, Wm. 1954. J. Wildl. Mgt. 18:52-60; U.S. Department of the Interior, Bureau of Sport Fisheries and Wildlife. Migratory Game Bird Briefing Book. Prepared by the Division of Management and Enforcement. Jan. 1971.

Gabay, S.J. University of Illinois, Urbana, Illinois. Recombination at the bar locus in a reverse attached-X system in D. melanogaster.

Recombination at the bar locus in Drosophila melanogaster was studied in a reverse attached-X stock synthesized by Dr. E. Novitski. The males of this stock were of the constitution $XY^S.Y^L$. The females of the stock carried a compound reversed metacentric X chromosome. They were homozygous for y and B, and heterozygous for v, f and $os^o$. The X chromosomes were attached at the "yellow" end rather than at the usual centromere end. This arrangement facilitates a higher frequency of homozygosis for the bar region and thus permits analysis of the marker constitution of exceptional females, that is, females which are double-bar or half-bar in phenotype. The likelihood of recovery of sister chromatids involved in exceptional events is increased as well, a prerequisite to the study of the reciprocity of intrachromosomal events.-----Exceptional females were analyzed to determine the bar genotype and the combination of marker genes carried by each of their X chromosomes. The results were interpreted in regard to exchange between obliquely synapsed members of the duplication, which is associated with exchange of outside markers and is presumed to be reciprocal. Results were also interpreted in regard to the hypothesis of intrachromosomal exchange (Laughnan, 1961) in which the markers are expected to be nonrecombinant and which proposes a reciprocal sister chromatid event.-----A total of 59,836 females, and therefore 119,672 X chromosomes, were examined for changes at the bar locus. A total of 193 recombinant exceptions was observed. Fifty-seven of these carried bar changes in both X chromosomes and were $B^+/BB$ in genotype with the exchange of outside markers which is predicted on the basis of reciprocal products of crossing over between obliquely synapsed members of the bar duplication. The reciprocity of the exchange between obliquely synapsed duplication members was thus demonstrated. One hundred of the recombinant exceptions were of the genotype $B^+/B$ and thirty-six were BB/B. The three classes of recombinant exceptions are expected to be equal in frequency and the discrepancy is ascribed to the reduced viability of the BB females.-----Eleven females carrying nonrecombinant exceptional strands were observed. Eight of these were half-bar in phenotype, one had round eyes and two were double-bar in phenotype. One of the BB nonrecombinant strands was recovered along with its sister strand and offered an opportunity to test the prediction that intrachromosomal exchanges between sister strands produce BB and $B^+$ strands as reciprocal types. Analysis showed this exceptional female to carry bar on one strand and double-bar on the other. While females of this type are not expected on the hypothesis of intrachromosomal exchange, the number of BB nonrecombinant females is insufficient to refute the hypothesis. More cases in which the nonrecombinant BB exceptional strand is recovered along with its sister are needed before any final conclusions concerning the model can be drawn.-----Four females carrying aberrations associated with bar changes were also observed. Three of these carried deletions and the fourth is still in the process of analysis.-----All 193 recombinant-exceptional females obtained from these studies exhibited normal genetic behavior in that all exceptional strands were homozygous viable. The frequency of these recombinant bar changes was one per 480 X chromosomes. The eleven females carrying nonrecombinant exceptional strands were analyzed cytologically as well as genetically and the frequency of nonaberrant changes was one per 10,000 X chromosomes. The frequency of X chromosomes carrying bar changes associated with aberrations was one per 24,000 X chromosomes.

This work submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy in Botany in the Graduate College of the University of Illinois, 1969.

This work was supported by National Science Foundation Grant GB-7635.

Reference: Laughnan, J.R., 1961, Mutation and Plant Breeding, NAS-NRC 891: 3-29.

Würgler, F.E. Swiss Federal Institute of Technology, Zürich, Switzerland. Synthetic female sterile factors in two combinations of X-chromosomes with dp bw ; st $p^p$ autosomes in D. melanogaster.

During the last few years we tried to construct some new multipurpose stocks with marked sex-chromosomes and the autosomal markers dp bw ; st $p^p$. The autosomes were always derived from the Inscy ; dp bw ; st $p^p$ stock obtained from I.I. Oster, Bowling Green, Ohio, USA (stock number j 419 in the 1971 stock list). In a first set of experiments (U. PETERMANN, Mutation Res. 5, 397-410, 1968) we tried to replace the Inscy chromosome by a crossover product with the markers y $sc^{S1}$ B f In-49 v $w^a$ $sc^8$ ($=X^x$) obtained from females heterozygous for the following

chromosomes: y $sc^{Sl}$ B In-49 v $w^a$ $sc^8$ / $sc^{Sl}$ f In-49 v $w^a$ $sc^8$. It turned out that none of the crossover chromosomes obtained gave fertile females in combination with the dp bw ; st $p^p$ autosomes. A similar result was recently found in an attempt to replace the Inscy chromosome by the XY(Parker 110-8) $y^?$ su($w^a$) $w^a$ KS·KL $y^+$ chromosome. The following table summarises our results:

| | |
|---|---|
| Inscy/Inscy ; dp bw ; st $p^p$ | fertile |
| $X^*$ / Inscy ; dp bw ; st $p^p$ | fertile |
| $X^*$ / $X^*$ ; dp bw ; st $p^p$ | sterile |
| XY (Parker 110-8)/Inscy ; dp bw ; st $p^p$ | fertile |
| XY (Parker 110-8)/XY(Parker 110-8);dp bw ; st $p^p$ | sterile |

Since the $X^*$ and the XY(Parker 110-8) in homozygous condition in combination with other autosomes (including heterozygosity for dp bw ; st $p^p$) give fertile females "synthetic sterility factors" seem to result in the particular combinations listed in the table. Male fertility was not affected.

Work supported by Schweizerischer Nationalfonds zur Förderung der wissenschaftlichen Forschung.

Jones, A.M. University of California, La Jolla, California. The cytological localization of cd and wo by means of deficiency mapping.

X/T(Y;3)/cd males were produced using translocations B93, D100, B27, and H173 with autosomal breakpoints in 93F-94A, 94A, 94E, and 95E, respectively. These males were crossed to stock females carrying translocations with adjacent autosomal breakpoints. In this manner it was possible to produce interstitial deficiencies for the segments between the autosomal breakpoints of the two translocations in combination with the normal third chromosome 3 carrying cd. These heterozygotes are recognizable on the basis of the phenotype with respect to y, Hw, $B^S$, and Ubx as outlined below:

$$\frac{Y^S X \cdot Y^L,\ In(1)EN,\ y\ B}{A^D Y^P 94E,\ y^+};\ \frac{In(3LR)TM6,\ Ubx^{67b}}{Y^D A^P 94E,\ B^S}\ \varphi \quad X \quad \frac{+}{+};\ \frac{cd}{cd}\ \sigma$$

$$\frac{C(1)M3,\ y}{A^D Y^P 94A,\ B^S};\ \frac{In(3LR)TM6,\ Ubx^{67b}}{Y^D A^P 94A,\ y^+}\ \varphi \quad X \quad \frac{+}{A^D Y^P 94E,\ y^+};\ \frac{cd}{Y^D A^P 94E,\ B^S}\ \sigma$$

adjacent I in both | disjunction parents

$$\frac{C(1)M3,\ y}{A^D Y^P 94E,\ y^+};\ \frac{cd}{Y^D A^P 94A,\ y^+}\ \varphi$$

The $y^+y^+$ (extreme hairy wing) non B phenotype of this female shows unambiguously that she carries the $A^D Y^P 94E$ and the $Y^D A^P 95E$ elements and is therefore deficient for 94A to 94E. The fact that she has cardinal eyes places the cd locus between 94A and 94E. Similar crosses placed wo (white ocelli) in the same cytological interval. The exact status of wo is uncertain, however, as both cd/cd and cd/wo flies have white ocelli. By the same procedures obt (obtuse) and bar-3 were found to lie outside the 94A-95E interval.

The sterility of some X/T(Y;A) males impairs the general utility of the above method; this difficulty can be circumvented by first constructing males of constitution $Y^S X \cdot Y^L$/X; autosomal recessive/autosomal balancer for use in the first generation indicated above to produce $Y^S X \cdot Y^L$/T(Y;A)/autosomal recessive in place of X/T(Y;A)/autosomal recessive in the second generation of the crossing scheme.

Wargent, J.M. University of Sheffield, Sheffield, England. Position-effect variegation in wings of D. melanogaster.

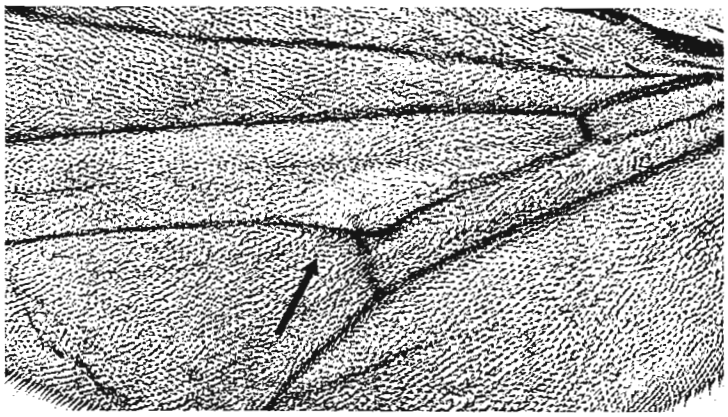
The wings of the mutant miniature of Krivshenko[1] ($m^K$) at normal temperatures (25°C) are sometimes crumpled and may vary in size from fully wild type to fully 'miniature'. The 'miniature' wings show the distinctive morphology of the miniature (m) mutant.

Reciprocal crosses, $m^K/m^K$ x m/Y and m/m x $m^K$/Y were set up, and the $F_1$ bred at 14°C. Microscopic examination of the wings of the female heterozygote, $m^K$/m, showed that they contain patches in which the hairs appeared closer together than in surrounding areas. Measurements were made of the distances between hairs in patches (p) and also in surrounding areas (s). For comparative purposes measurements were also made of the inter-hair distances in the Amherst wild type and in a homozygous m strain, both bred at 14°C. The results are shown in the table below.

| Strain | Number of Measurements | Mean inter-hair distances ± standard error |
|---|---|---|
| $m^K$/m (p) | 50 | 62.19 ± 0.93 |
| $m^K$/m (s) | 50 | 88.55 ± 0.98 |
| +/+ | 50 | 92.08 ± 1.51 |
| m/m | 50 | 53.40 ± 0.90 |

Statistical comparison of the difference between means shows that the wild type and the m mutant are significantly different at the 1% level. The difference between $m^K$/m (s) and +/+ is not significant and although a significant difference was obtained between $m^K$/m (p) and m/m the inter-hair distances of the former resemble those of the m strain rather than those of the wild type. The difference between the means for $m^K$/m (s) and $m^K$/m (p) is significant at the 1% level. These results indicate that the wings contain both wild type and m-like cells.

There is considerable variation in the size of the patches observed; some wings contain predominantly wild type cells while others consist mainly of m-like cells. The photograph shows a predominantly 'miniature' wing with an area of wild type cells. It is assumed that the crumpled phenotype is caused by the presence of patches, and that the number and size of the patches determine the size of the wing.



The strain contains an inversion with breakpoints in section 10E4-5 and section 20B of the salivary X chromosome. The m locus, in section 10E1-2, is relocated next to broken heterochromatin in the rearranged chromosome. The presence of mutant cells in the wing is therefore probably due to a variegation-type position effect[2] at the m locus; this conclusion is supported by the observation that the mottling is enhanced by low temperature.

References: 1. Krivshenko, J., 1956, DIS 30: 75; 2. Lewis, E.B., 1950, Advances in Genetics 3: 73-115. This work was supported by Grant No. 68/1317 from the Science Research Council of Great Britain.

Novitski, E., E. Ehrlich and H. Becker*. University of Oregon, Eugene, Oregon and University of Munich, Germany*. A terminal attachment region on 2L.

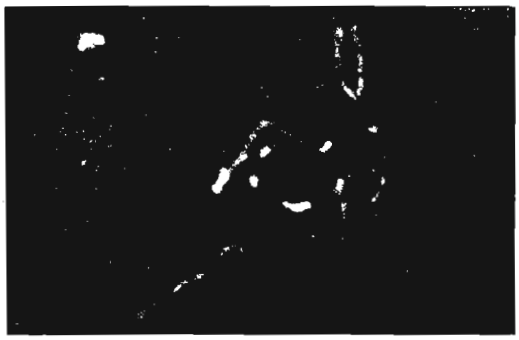
Compounds involving the X and Y chromosomes or several X-chromosomes have been put together in virtually every combination, but compounds involving the autosomes have been limited to the five cases where homologous arms of an autosome have been attached to the same centromere as reversed metacentrics. There are two reasons for this difference in versatility of the sex chromosomes as opposed to the autosomes.

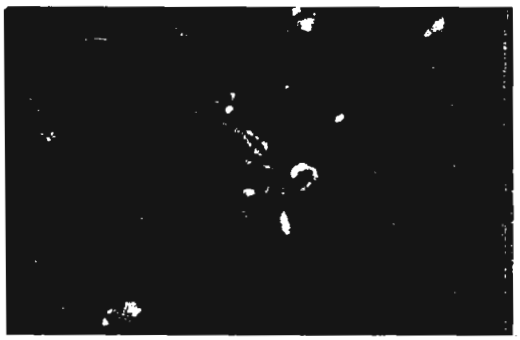
1. The sex chromosomes are much simpler to handle in single and compound forms because of the manner of their transmission and because of the prior existence of certain specialized chromosome types.

2. The occurrence of a complete X-chromosome with a terminal heterochromatic (and dispensable) section, first found in In(1)EN, made it possible to tack X-chromosomes together in serial order into compounds of all possible combinations. Since the construction of compound autosomes is limited by the absence at any of the ends of any useful terminal heterochromatic piece with an associated dispensable marker, it was decided to synthesize such chromosomes as a first step in making more useful autosomal compounds.

The hope that such an attempt might be successful stems from the suspicion that on one or more of the chromosome tips there may be small regions that are essentially heterochromatic (or a few loci which can be readily dispensed with in the heterozygote). This thought is based in part on the occurrence of the two ring chromosomes, R(1) and R(1)2, from attached X-chromosomes. The simplest and perhaps only explanation for their origin is that one of the tips of the attached X's underwent a rare "exchange" with the base of the other arm of the attached X. It seemed a good possibility that a similar situation might exist at the tips of one or more autosomes and that a search for a similar exchange that would add a larger piece of heterochromatin along with a good marker to an autosome would give positive results.

The procedure involved irradiating (3,000r) females carrying a doubly marked Y-chromosome along with an attached X, and looking for cases among the $F_1$ where the two markers, $y^+$ and Bar, have been separated, indicating that some kind of "exchange" had taken place, but where the attached X was not involved. Almost without exception, each female so treated produced one or more progeny showing such a separation and it was necessary to limit the analysis to one exceptional progeny per parental female, in order to avoid duplication. The exceptions selected were tested for segregation of the X-chromosome markers from the major autosomes. Two cases were found of attachment of Bar to the left end of the second chromosome. In both cases, results of the tests of this chromosome against a normal second showed for it to be .01 units to the left of the locus of al. Tests for the presence of Y-chromosome fertility factors at the left end of X have showed that in the first case (B3) none of the fertility factors is present and in the second case (B5) two (S1 and L5, and possibly L4) are. B3 is viable and fertile when homozygous; tests are not yet complete for B5.





Ganglion metaphases stained with quinacrine hydrochloride confirm the presence of some of the Y-chromosome at the tip of the second chromosome of B5. As can be seen on the photographs, there are two bright fourth chromosomes and XY chromosome with two bright regions at one end (undoubtedly corresponding to the short arm of the Y) and three at the other (the long arm of the Y). One of the autosomes has two bright blobs at one end, with a less well stained piece distal to them. This must represent the tip of 2L to which part of Y long, along with Bar, have been attached.

With these chromosomes, it should now be possible to make up certain additional compounds, by hooking another autosomal arm (or an X, or a Y) to the tip of 2L.

Additional runs are being made to try to synthesize chromosomes in which a marker is similarly placed on the tips of the other autosomal arms. If no other cases occur, an attempt will be made to use certain Y-autosome translocations (see the Washington-La Jolla report in this issue).

Bremner, T.A., W.L. Douglas and G.O. Ogonji. Howard University, Washington, D.C. Substrate-specific differences of alcohol and octanol dehydrogenases in eight species of Drosophilidae.

Of the ten cathodally migrating isozymes of alcohol dehydrogenase (ADH) detected by Ursprung and Leone (1965) in D. melanogaster, the slowest three showed stronger formazan staining with n-octanol than with ethanol. These three bands were shown, on the basis of linkage relationships, substrate specificity, and differential elution from DEAE cellulose columns. to belong to a separate enzyme system, octanol dehydrogenase (ODH) which shows strong formazan staining with n-hexanol, n-heptanol, and n-octanol (Courtright, et al., 1966). Isopropanol and sec-butanol are equally good substrates or better than ethanol for ADH in D. melanogaster (Johnson and Denniston, 1964; Grell et al., 1965).

In an attempt to differentiate these two enzyme systems further and to ascertain whether there might be species-related differences in substrate specificity within each system, a comparison of the substrate requirements of both ADH and ODH in eight members of the family Drosophilidae was undertaken. The eight species belong to two genera, Drosophila and Zaprionus. Of the seven Drosophila species four, robusta, camargoi, metzii, and unipunctata belong to the subgenus Drosophila, while D. lebanonensis casteeli belongs to the subgenus Pholadoris (primitive), D. busckii to the subgenus Dorsiphola, and D. melanogaster to the subgenus Sophophora. The single Zaprionus species is Z. multistriata.

The substrates fall into four categories, primary unbranched alcohols, secondary alcohols, branched primary alcohols, and a cyclic alcohol, cyclohexanol. The method of agar gel electrophoresis and formazan staining of Ursprung and Leone (1965) as modified by Pipkin (1968) was used to assay crude homogenates of single female flies cultured on an enriched medium, and aged according to the following schema: D. busckii, 4-6 days; D. melanogaster, 5-6 days; D. metzii, 5-8 days; D. unipunctata, 7-9 days; D. camargoi, D. robusta, D. l. casteeli, and Z. multistriata, 9-11 days. These were the ages at which the respective species attained their optimum levels of enzyme activity as measured by the intensity of formazan staining. To compensate for the very small size of D. busckii 2-4 females were homogenized in a drop of distilled water.

From Tables I-IV it can be seen that both the ADH and ODH of D. unipunctata show more intense staining when secondary alcohols are used as substrates. The ADH of D. busckii is aberrant in that it shows a preference for the short chain unbranched primary alcohols while its ODH activity is quite low. Both ADH and ODH show a moderate preference for the long chain, unbranched primary alcohols in five of the species assayed. Although the two enzymes have overlapping substrate specificities, that of ODH is distinctly narrower and comprises

Table I Unbranched Primary Alcohols

| Substrate | Enzyme Activity | mel. | metz. | busc. | rob. | uni. | car. | Dlc. | Z.mult. |
|---|---|---|---|---|---|---|---|---|---|
| Methanol | ODH | - | * | - | - | - | ** | - | * |
| | ADH | *** | - | *** | *** | ** | *** | *** | *** |
| Ethanol | ODH | - | - | - | - | - | ** | - | * |
| | ADH | ** | - | * | *** | * | *** | *** | *** |
| N-propanol | ODH | ** | - | - | * | *** | ** | ** | ** |
| | ADH | *** | - | *** | *** | * | *** | *** | *** |
| N-butanol | ODH | * | * | - | ** | ** | ** | ** | * |
| | ADH | *** | - | * | *** | * | *** | *** | *** |
| N-amyl | ODH | ** | ** | * | * | - | *** | *** | *** |
| | ADH | *** | - | ** | *** | * | *** | *** | ** |
| N-hexanol | ODH | ** | *** | ** | ** | - | | *** | *** |
| | ADH | *** | - | ** | ** | * | | *** | ** |
| N-heptanol | ODH | ** | *** | * | *** | *** | *** | *** | *** |
| | ADH | *** | - | * | *** | * | *** | *** | *** |
| N-octanol | ODH | *** | *** | ** | ** | - | *** | *** | *** |
| | ADH | *** | - | - | *** | * | ** | *** | *** |
| Nonyl alc. | ODH | * | * | * | ** | * | ** | *** | *** |
| | ADH | *** | - | * | *** | * | *** | *** | *** |
| Decyl alc. | ODH | * | * | * | - | - | ** | ** | ** |
| | ADH | *** | - | * | *** | * | *** | *** | ** |

Table II Secondary Alcohols

| Substrate | Enzyme Activity | mel. | metz. | busc. | rob. | uni. | car. | Dlc. | Z.mult. |
|---|---|---|---|---|---|---|---|---|---|
| 2-butanol | ODH | - | * | * | * | ** | ** | - | *** |
| | ADH | *** | - | * | ** | ** | *** | *** | *** |
| 2-hexanol | ODH | - | - | - | - | *** | * | - | *** |
| | ADH | *** | * | ** | *** | * | *** | *** | *** |
| 4-heptanol | ODH | - | - | - | - | ** | *** | - | - |
| | ADH | *** | - | * | *** | ** | *** | * | * |
| 2-octanol | ODH | - | - | * | *** | *** | *** | *** | *** |
| | ADH | ** | - | * | * | ** | ** | *** | ** |

Table III Branched Primary Alcohols

| Substrate | Enzyme Activity | mel. | metz. | busc. | rob. | uni. | car. | Dlc. | Z.mult. |
|---|---|---|---|---|---|---|---|---|---|
| Iso-propanol | ODH | - | * | - | * | - | - | - | - |
| | ADH | *** | * | * | *** | ** | *** | *** | *** |
| Iso-butanol | ODH | * | * | - | *** | * | *** | ** | ** |
| | ADH | *** | * | * | ** | *** | * | *** | *** |
| Iso-amyl | ODH | * | ** | * | * | ** | * | ** | ** |
| alcohol | ADH | *** | - | * | * | ** | ** | ** | ** |
| Tert-butanol | ODH | * | - | * | - | - | ** | - | - |
| | ADH | ** | * | - | *** | * | ** | *** | ** |
| Tert-amyl | ODH | * | - | - | * | - | - | * | * |
| alcohol | ADH | *** | - | *** | * | *** | * | *** | ** |

Table IV Cyclic Alcohol

| Substrate | Enzyme Activity | mel. | metz. | busc. | rob. | uni. | car. | Dlc. | Z.mult. |
|---|---|---|---|---|---|---|---|---|---|
| Cyclohexanol | ODH | * | - | - | * | ** | * | - | * |
| | ADH | *** | - | ** | *** | * | *** | * | * |

Legend, Tables I - IV.

Species: mel. = D. melanogaster; metz. = D. metzii; busc. = D. busckii; rob. = D. robusta; uni. = D. unipunctata; car. = D. camargoi; Dlc. = D. l. casteeli; Z. mult. = Z. multistriata.

Intensity of formazan staining:

*** = strong; ** = moderate; * = trace; - = negative

primarily the 5 to 8-carbon alcohols, with n-heptanol giving the highest intensity of staining. The ADH of D. unipunctata shows no staining with n-hexanol, but moderate staining with cyclohexanol; its ADH uses all the substrates tested. The ADH of D. metzii does not use any of the unbranched primary alcohols, while that of D. melanogaster uses all of them.

The above findings suggest that the same enzyme in different species shows small but significant differences in substrate specificity which may be related to minor evolutionary differences in the structure of the molecule resulting in different stereochemical requirements for enzyme activity.

This work was supported by National Science Foundation Grant GB 8779, and NIH Grant 1 R 101 GM 18409-01.

References: Courtright et al, 1966, Genetics 54:1251-1260; Grell et al, 1965, Science 149:80-82; Johnson and Denniston, 1964, Nature 204:906-907; Pipkin, 1968, Genetics 60: 81-82; Ursprung and Leone, 1965, J. Exptl. Zool. 160:147-154.

Forman, M. and S.K. Majumdar. Lafayette College, Easton, Penn. Studies on the effects of monosodium glutamate on development and productivity of D. melanogaster.

Monosodium glutamate (MSG) is a widely used food additive. The first indication of possible ill effects from consumption of MSG was recorded in what has come to be known as the Chinese Restaurant Syndrome (Schaumberg, 1968) in man. Administrating high doses of MSG subcutaneously Olney (1969) and Olney and Sharpe (1969) produced brain lesions in the hypothalamous of mice and Rhesus monkey. As a result of these findings baby food manufacturers were asked to remove MSG from their products. Recent studies by Bazzano et al (1970) found no clinical or pathological changes in adult humans and adult gerbils when MSG was administered orally. Similarly Turner and Wright (1971) reported that 1% and 3% solutions of MSG caused no change in the development of D. melanogaster. Because of the conflicting reports the present investigation was undertaken to study the effects of MSG on the development and productivity of D. melanogaster. After initial work, the possibility of a lethal recessive sex-linked mutation was studied.

Eight female and eight male two-day-old Oregon-R flies were placed in vials containing Carolina Biological instant Drosophila medium. To the medium, one of the three solutions was added, with each solution being used in eight vials except for the sucrose where only four vials were used. One solution was pure distilled water, used as a control. A second control was a 0.7 M solution of sucrose. The third solution contained 0.7 M MSG. The $P_1$ flies were removed after seven days and the $F_1$ adults were counted on the seventeenth day according to sex and total number.

To determine the effect of MSG on the productivity, two-day-old Oregon-R male and female flies were allowed to drink 0.7 M solution of MSG for 24 hours. The drinking procedure involved soaking lens paper in the MSG solution and placing it in a bottle. Both sexes were kept together in order to insure mass culturing during consumption of the chemical. The control flies drank distilled water. After 24 hours the flies were transferred to vials containing instant medium and distilled water--two males and one female in each. The five-day brood system was used and four broods were obtained. Seventeen days after a brood was set up, the offspring were counted and the sex ratio was recorded.

It is apparent from Table 1 that 0.7 M MSG had an inhibitory effect on the development of the flies; as a result fewer $F_1$ offspring were produced. Table 1 shows that the average number of $F_1$ adults in the control (494.4) was greater than twice the average number of $F_1$ flies in the MSG (215.1). However, the numbers of the $F_1$ adult flies developed in the sucrose (519.5) and in the control were quite similar. The results of Turner and Wright (1971) seem to be contrary to the findings presented here. However, since they used only 1% and 3% solutions, it is possible that there is a threshold point somewhere between 3% and 10% solutions (0.7 M MSG is approximately 10%) at which the chemical produces an effect on the flies. An abnormal sex ratio occurred in the MSG cultures. There were 60.1% females in the MSG cultures compared with 49.95% in the control and 50.05% in the sucrose. A chi-square performed for determining the probability of the sex ratio obtained in the MSG cultures showed a significant difference ($p<0.001$).

Table 1. Effects of monosodium glutamate (MSG) on the development of D. melanogaster.

| Treatment | No. of vials | Total no. of $F_1$ flies produced | Average no. of $F_1$ flies produced per vial | % female produced |
|---|---|---|---|---|
| Control (Water) | 8 | 3955 | 494.4 | 49.95 |
| Control (0.7 M Sugar) | 4 | 2078 | 519.5 | 50.05 |
| MSG (0.7 M) | 8 | 1724 | 215.1 | 60.1 |

The results of the brood experiment are summarized in Table 2. In brood 1 (0-5 days), there was a control:MSG ratio of 2:1 for the average number of $F_1$ progeny. In the second and third broods, the ratio fell to approximately 1.75:1:00 and 1.50:1.00 respectively, and in the last brood, the ratio was almost 1.0:1.2 with the MSG flies producing a greater number of offspring in the last brood. The total average number of flies in the control (423.52) was greater than the total in the MSG (261.94) by nearly 60%. It appears that MSG has more noticeable effect on the productivity in the first and second broods. The sex ratio in the MSG culture was not significantly altered.

Table 2. Five-day brood showing comparative productivity between control and 0.7 M MSG treated D. melanogaster

| Treatment | Brood 1 0-5 | Brood 2 5-10 | Brood 3 10-15 | Brood 4 15-20 | Total | Sex Ratio |
|---|---|---|---|---|---|---|
| Control | | | | | | |
| Total | 3982 | 2887 | 1068 | 267 | 8204 | |
| No. of vials | 21 | 21 | 16 | 9 | | |
| Average | 189.62 | 137.48 | 66.75 | 29.97 | 423.52 | |
| Percent of total | 44.77 | 32.46 | 15.77 | 7.00 | | |
| Monosodium Glutamate | | | | | | |
| Male | 1035 | 890 | 302 | 135 | 2362 | 1:1.017 |
| Female | 1058 | 834 | 287 | 144 | 2323 | |
| Total | 2093 | 1724 | 589 | 279 | 4685 | |
| No. of vials | 21 | 21 | 13 | 8 | | |
| Average | 99.67 | 82.10 | 45.31 | 34.88 | 261.94 | |
| Percent of total | 38.05 | 31.34 | 17.30 | 13.31 | | |

The Muller-5 method was used to determine the mutagenic activity of MSG. The male Oregon-R flies drank 0.7 M MSG for 24 hours and the flies were tested for sex-linked recessive lethal mutations. This test on 474 $F_1$ females yielded no recessive mutations.

References: Bazzano, G., J.A. Delia, and R.E. Olson 1970 Science 169: 1208-1209; Olney, J.W. 1969. Science 164: 719-721; Olney, J.W. and L.G. Sharpe 1969. Science 166: 386-388; Schaumberg, H.H. 1968. New Eng. J. Med. 278: 1122-1124; Turner, D.C. and C.P. Wright 1971. DIS 46: 118.

Thomas-Orillard, M. Faculté des Sciences de Paris VI, France. Influence of the culture medium on the number of ovarioles in D. melanogaster.

The phenotypic expression of the number of ovarioles is influenced by the environment. The rearing temperature (David et Clavel, 1967) and the food (Saviliev, 1928; David, 1960) have an important effect on this character. Control of the temperature is always possible: all the experiments are carried on at 25±0.5° C. It is more difficult to appreciate the quantity of food which is necessary for a perfect ovary development. A study of the influence of the food on the phenotype expression of the ovarioles number is necessary to establish the experimental conditions for a study of the action of genes. Culture medium nature, density of population, sensibility of each instar larva to feeding were examined on two laboratory strains with very different geographical origins: one from France, the other from Japan and also on crosses between French and Japanese strains.

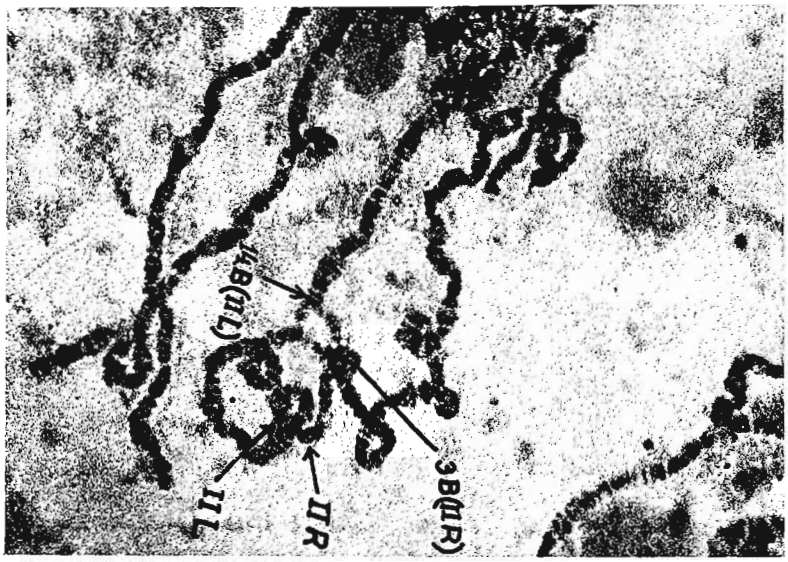
The phenotypic expression of the number of ovarioles is different on cornmeal medium and on yeast medium: t test with 98 degrees of freedom gives a significant value for t; P = .05. It depends also on the quantity of culture medium available for each larva. The biometrical characteristics of a strain are stable when all the larvae are well fed during the three instars. When first, or first and second or three instars are not well fed the mean of ovarioles number decrease significantly (F test between effect of the culture medium at different instar and residual variation gives F = 4.15 for 3 and 41 degrees of freedom).

When the density of adult population does not exceed more than 50 animals for each culture bottle, the mean of the strain is stable; but when the density is bigger than 50 the mean decreases significantly from 36 ovarioles per female to 28. The ovarioles number of the females of the first generation is significantly greater than the arithmetic mean when the number of imagos is lower than 50 in the rearing bottles. It is not far from the arithmetic mean value when the density of the population varies from 100 to 150. We conclude, in the first case that there is evidence of heterosis, in the second case that genes have additive effects.

We see that in fact, this orientates the conclusions about the genes action. In all experiments on the genetic control of ovarioles number it is necessary to work with rearing bottles where the population density is maintained around 50. By this way, controlling the effect of feeding and working at constant temperature we can expect to run into the purely genetic problems.

Singh, V.K., M. Mishra and A.P. Jha. Bhagalpur University, Bhagalpur-7, Bihar, India. A new pericentric inversion in D. ananassae.

D. ananassae a member of the melanogaster species group, is highly polymorphic due to inversions in its natural populations (Kikkawa 1938; Dobzhansky and Dreyfus 1943; Freire-Maia 1960; Ray-Chaudhuri and Jha 1965 and Futch 1966). We are reporting, herewith, for the first time, a new pericentric inversion on the second chromosome of D. ananassae from its Bahadurpur population. Bahadurpur is a sparsely situated village within a dense forested area known as Samtha forest in the State of Bihar. Breakage points in this inversion were determined from the reference map prepared by Ray-Chaudhuri and Jha (1965). One of the breaks has occurred in region 14A of IIL and other in 2B of IIR as shown




above. Freire-Maia (1960) and Futch (1966) respectively reported pericentric inversions on the second chromosome of D. ananassae from Brazil and South pacific islands. Our report is a new one in that its breakage points are located on the regions different from those reported by them.

Voss, R. Hebrew University of Jerusalem, Israel. A common suppressor for a lethal mutation and a forked mutation.

Recent experiments with lethal $1^{3DES}$, a suppression of which was described in DIS#46, had revealed that the suppression of forked was not induced simultaneously with the suppression of the lethal, but was associated with the lethal $3^{DES}$ originally. This is in accord with A. Schalet's findings in mapping the proximal X chromosome region (DIS 46, 131). However, the reversion of the lethal does not cause reversion of su-f. The revertant flies still show suppression of forked. Therefore it seems necessary to call the new suppressor su-$1^{3DES}$ and not su-$f^{V}$ as was suggested before. All features of the suppressor which were described previously still hold. To this may be added that the suppressor seems to be a Y suppressed lethal as XO males are inviable and homozygous females $1^{3DES}$, su$1^{3DES}$ with a Y chromosome are viable, although sterile. The independent reversion may be interpreted to mean that $1^{3DES}$ covers more than the su-f locus only, or that there is more than one function associated with su-f which can be separated from it.

Jha, A.P., M. Mishra and V.K. Singh. Bhagalpur University, Bhagalpur-7, Bihar, India. Abnormal sex ratio in Darjeeling Drosophila population.

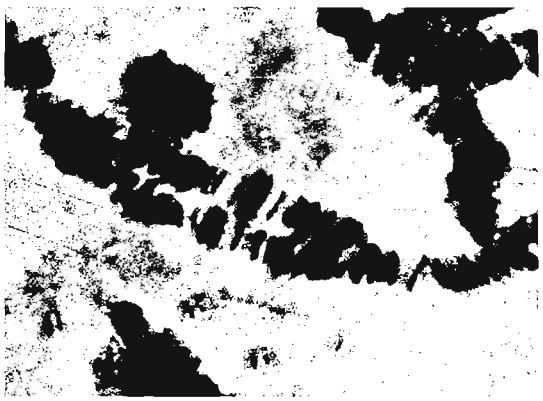
Attempts were made to collect Drosophila at Darjeeling at an altitude of 7000 ft. in India from October 5 to October 10, 1970. Our collection of 776 flies comprised seven good species (table below). Out of them only 276 were females. The collection was made by using fermented banana, orange and pineapple mesh as baits. Traps were suspended in air by hanging them upwardly. Morning and evening collections were made. The population was so thin that we could collect only 2 to 3 individuals of different species on the 5th and 6th of October from each trap. Probably it was due to extreme winter (50-55°F). Then we put some traps in the evening of October 7th inside some bakeries whose temperature was higher than outside. From the morning of October 8th the flies started pouring into the traps. Specimens were identified in collaboration with Dr. J.P. Gupta, Drosophila Laboratory, Banaras Hindu University, India. D. bipectinata was the dominating species. D. ananassae and D. melanogaster were next in number to visit the traps.

Table 1. Data showing the sex ratio of Drosophilia flies collected at Darjeeling

| Species | Males | Females | Total |
|---|---|---|---|
| D. bipectinata | 148 | 76 | 224 |
| D. ananassae | 129 | 93 | 222 |
| D. melanogaster | 141 | 56 | 197 |
| D. busckii | 41 | 13 | 54 |
| D. kikkawai | 23 | 18 | 41 |
| Chaetodrosophila quadrilineata | 12 | 17 | 29 |
| D. malerkotliana | 6 | 3 | 9 |
| | 500 | 276 | 776 |

An ideal sex ratio in a population is 50M:50F. But this equality of the number of sexes has not been favoured in any of the Drosophila species, herewith reported in the environmental conditions of Darjeeling. If two or more males are necessarily required to inseminate a female Drosophila, in that case the males may preponderate in the population. We do not know if any male producing tendency has acquired in the heredity of Darjeeling populations of these Drosophila species. The problem is intricate and we leave its solution for the future.

Krimbas, C.B. Agricultural College of Athens, Athens (Votanikos), Greece. A newly spontaneously formed chromosome arrangement in one salivary gland cell of D. subobscura.



In one of the salivary gland cells of an individual of D. subobscura a small inversion or deletion has been detected in heterozygous condition near the centromere end of chromosome O (region 76 of the map of Kunze-Mühl and E. Müller, Chromosoma 1958) shown in the photograph. All other cells of the salivary gland of the same individual did not show heterozygosity in that region. It is apparently a newly formed chromosomal arrangement in only one salivary gland cell of this individual.

Marengo, N.P. and S.H. Vernick. C.W. Post College of Long Island University, Greenvale, New York. Virus-like particles in nuclei of muscle fibers of genetically "rotated" prepupae of D. melanogaster.

The gene abdomen rotatum (ar) was discovered and named by Beliajeff (1931). Marengo and Howland (1942) described its effect on the development of the fly and established that the actual rotation of the imaginal abdomen coincided with the termination of the prepupal period and the movements attendant on the change from prepupa to pupa. In addition to the rotation of the pupal abdomen within the completely symmetrical pupa case, abnormalities of the puparium appeared which were clearly attributable to exaggeration of the function of the persisting larval muscles. Robertson (1936) states that these muscles are responsible for the contraction of the larval cuticle in puparium formation and also the movements of the individual as it changes from a prepupa to a pupa, with a clearly defined head, thorax and abdomen. Marengo and Howland (1942) suggested that these muscles were also responsible for the actual rotation of the pupal abdomen since the time of the first recognition of actual rotation coincided with the movements characterizing the change from prepupa to pupa. The most clearly recognizable puparial abnormalities were persistent segmentation of the hardened cuticle and abnormally large lateral bulges of the puparial wall between the attachments of the dorso-ventral muscles.

MATERIALS AND METHODS: Since abdominal rotation prevents normal copulation, the stock used in the 1942 study was a balanced lethal with eyeless Dominant ($ar/ey^D$). This stock apparently has not survived, and the current study was made possible through the courtesy of Pollards Wood Research Station of the Institute of Cancer Research, Royal Cancer Hospital, Buckinghamshire, England, which supplied a new balanced lethal with cubitus interruptus ($ar/ci^D$).

Since the structural abnormalities of the puparia of "rotated" flies appeared to be a direct consequence of abnormal muscle function, and since paraffin sections for light microscopy revealed no consistent structural muscle abnormality, it was decided to examine the prepupal muscles of "rotated" (ar/ar) individuals and normals (+/ar and +/+) by electron microscopy.

Prepupae with puparial abnormalities identifying them as genetically "rotated" were opened in 3% buffered glutaraldehyde and the muscle fibers dissected out. After three hours these were washed overnight in a rinsing buffer consisting of 10 gm. sucrose in 100ml Sorenson's stock buffer, post-fixed two hours in Palade's buffered osmium fixative, prestained three hours in .5% uranyl acetate, dehydrated and embedded in epon. Sections were cut with a diamond knife on a Reichert OMU-2 ultramicrotome, stained in uranyl acetate and lead citrate, and examined with an Hitachi HS-7 electron microscope. Identical treatment was given to muscle fibers of normal ($ar/ci^D$) individuals and +/+ individuals of Oregon "R" stock.

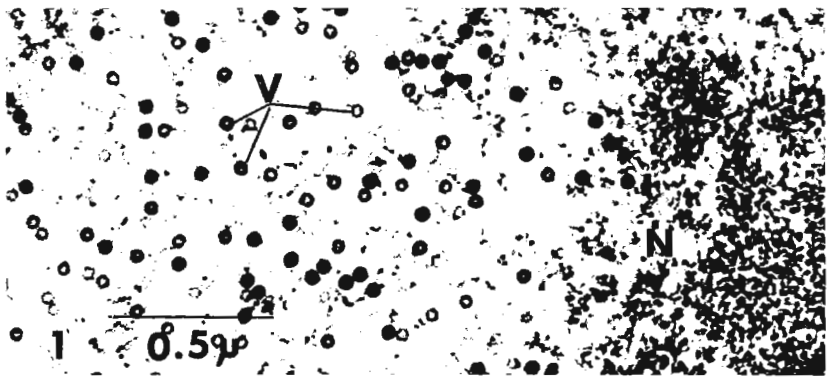



FIG. 1. Section through nucleus of muscle cell from prepupa homozygous for abdomen rotatum (ar/ar). Virus-like particles (V) are apparent in the nucleoplasm which has a characteristic empty appearance. Part of the nucleolus (N) is visible. X49,000.

RESULTS: At the present stage in this study, no readily identifiable ultrastructural difference was found between the fibrillar organization of the muscles from the "rotated" prepupae and those of the normal individuals. However, a high percentage of the muscle nuclei of genetically "rotated" prepupae contained virus-like particles approximately 48mμ in size (Fig. 1).

In a number of cases, they appear to be budding from a local electron-dense region of the nucleoplasm (Fig. 2). No particles were observed in nuclei from the muscles of either the normal heterozygotes (ar/+) or the +/+ Oregon "R" wild type.

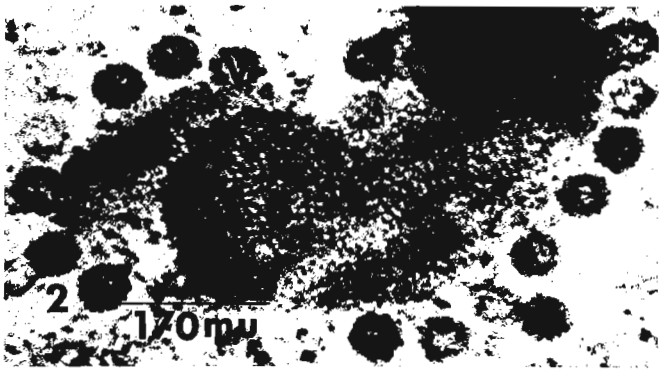



FIG. 2. Section through nucleus showing particles (V) which appear to be budding from a faintly crystalline nucleoplasmic condensation. X145,000.

DISCUSSION: The classification and significance of the observed particles is at present not known. Since abdomen rotatum as originally described by Beliajeff (1931) was a mutant of chromosome IV it might be speculated that the presence of a virus may relate to the carrying of information from the mutant DNA of chromosome IV to the larval muscles. These muscles bring about through exaggerated contraction both the puparial abnormalities described and the rotation of imaginal abdomen which is characteristic of the adult phenotypic expression of this gene.

If these particles are carriers of the ar codon, their isolation from genetically "rotated" individuals might provide a means of experimental transformation of normal larvae capable of metamorphosis into abnormal prepupae, pupae and "rotated" adults.

SUMMARY: Nuclei of muscle fibers of genetically "rotated" (ar/ar) prepupae of D. melanogaster examined by electron microscopy were found to contain virus-like particles not previously described. These particles were not observed in identical preparations of Oregon "R" prepupae and normal heterozygotes of the ar/ci$^{D}$ stock.

References: Beliajeff, N.K. 1931 Erbliche Asymmetrie bei Drosophilia. Ein neues Gen im IV chromosom von D. melanogaster. Biol. Zbl. 51:701-709; Marengo, N.P., and R.B. Howland. 1942 The effect of the gene abdomen rotatum on the development of D. melanogaster. Genetics 27:604-611; Robertson, C.W. 1936 The metamorphosis of D. melanogaster, including an accurately timed account of the principal morphological changes. J. Morph. 59:351-359.

Supported in part by a faculty research grant from C.W. Post College.

Doane, W.W. Yale University, New Haven, Connecticut. Isoamylases in Drosophila hydei: a system for the analysis of gene-specific puffing activity.

α-Amylase activity in the larval gut of D. hydei is essentially restricted to a small region at the anterior end of the posterior midgut. The region is characterized by large secretory cells with polytene chromosomes and, unlike the situation in D. melanogaster, polyteny here is of a magnitude readily open to cytological investigation of gene-specific puffing activities. As a secretory protein whose activity may be manipulated by dietary starch, amylase is an ideal subject for such a study (Doane, W.W., 1969, pp. 73-108 in "RNA in Development" E.W. Hanly, ed., Univ. of Utah Press).

A single structural gene for Amylase (Amy) is located on chromosome 5 in hydei, between cn and vg. Making use of the latter markers and two electrophoretic Amy variants, a screening program was set up to select for potential X-ray induced deficiencies of the Amy locus, and so position the locus on the cytological map of the chromosome (Doane, W.W., 1971, Isoz. Bull. 4:46-48). Of the strains selected, one is particularly useful: it contains a fifth chromosome with an inversion near the center. The inverted section is apparently accompanied by deficiencies of the Amy and vg loci, perhaps at opposite ends. Tentative location of the break points are: proximally, in 107A before the doublet, and, distally, in 109C, between bands 7 and 8 (according to the map of H. Berendes, 1963, Chromosoma 14: 195). The inversion has been examined in polytene chromosomes from salivary glands and midguts (anterior and posterior regions) taken from larvae reared on starch- and/or sugar-yeast diets with promising results. (Supported by grant NSF GB 8607.)

Sunanda Mahajani, Division of Genetics, Indian Agricultural Research Institute, New Delhi-12, India. Crossing over in the inversion carrying second chromosome of a D. melanogaster male.

Two facts are known which strongly suppress crossing over in D. melanogaster. These are: (a) sex and (b) chromosome inversions. Crossing over in D. melanogaster males is a very rare event. Likewise, pairing difficulties imposed by inversions effectively prevent crossing over in chromosomes carrying them; infrequently, however, cross-overs in inversion heterozygotes of D. melanogaster have been reported (Kaufman and Gay, 1970, DIS, 45:81). In this communication I report a rare cross-over event that has occurred in the second chromosome of a male bearing the In(2L)Cy and In(2R)Cy inversions.

Three cinnabar-eyed mutant females were obtained among D. melanogaster larvae reared on basal medium containing 0.03% nitrosoguanidine. Mutant virgins were mated to Cy/Bl $L^2$ males with two objectives in view: (a) to confirm the second chromosome location of the cinnabar mutation and (b) to keep the treated chromosome intact. Two types of flies should normally be expected in the progeny - Curly (Cy) and Bristle Lobe (Bl $L^2$). Actual results, however, showed that in addition to the above classes, Curly Bristle Lobe (Cy Bl $L^2$) and wild type (+++) flies were also obtained (3.4% and 1.1% respectively; 172 flies were observed).

The recovery of these unusual types of flies can be explained on the basis of a cross-over between the Cy and Bl loci, presumably in the region of the chromosome lying between the two inversions. This would bring the Cy, Bl and $L^2$ markers on one chromosome and their respective (+) alleles on the other.

The recovery of cross-over products observed by me is unusual in two ways. Not only has a cross-over occurred in a male, a rare event in itself, but it has taken place inspite of the inversions that should have normally prevented it.

Mahowald, A.P. Institute for Cancer Research, Fox Chase, Philadelphia, Pennsylvania. Intracellular symbionts of Drosophila.

Many workers have demonstrated with the electron microscope the presence of Rickettsia or bacteria within cells of Drosophila. King (1970) has recently reviewed these findings and presented a detailed account of hithertofore unpublished work from his laboratory. Since many laboratories are actively studying the nucleic acids of eggs of Drosophila, it is important to know whether there are bacterial contaminants within the cells. King points out the widespread occurrence of these organisms, which he terms "A-bodies", so that one gains the impression that they could be found in any stock examined with the electron microscope. But in extensive ultrastructural studies of oogenesis in two strains of D. melanogaster (Oregon R and Cochaponsett, obtained from T.R.F. Wright), D. hydei (New Haven and Zürich strains, obtained from S.J. Counce-Nicklas), D. virilis (Johns Hopkins), and D. immigrans, they have not been found. Furthermore, they have not been detected in my stocks of fs(1)N, dor 1(1)X2, 1(1)ffll (obtained from S.J. Counce-Nicklas). On the other hand, they are present in high concentration within the oocytes of D. willistoni, Barbados III obtained from D.F. Poulson. In this species the distribution differs considerably from previous reports. Ullmann (1965) found ε-granules (these appear to be the same as A-bodies) only in Stage 13 oocytes and later stages, and suggested that they may form from the oolemma. Furthermore, she only found them at the posterior tip of the embryo. In the strain of D. willistoni I have studied extensively, these structures are found in the oogonia and all cells derived from these cells, i.e., the nurse cells and oocytes. They have not been found in follicle cells. A brief survey of other adult and larval tissues also failed to detect any outside the germ tissue. During embryogenesis they are at first found throughout the ooplasm, but gradually they become concentrated in the posterior portion of the egg. Most of these A-bodies are included in the pole cells. A few A-bodies are also found in the blastoderm cells, even at the anterior end. Since apparently there are few, if any, A-bodies in somatic tissue, there must be some restriction on their growth outside the germ line. The role of these A-bodies or Rickettsia is unknown.

Reference: King, R.C. 1970, Ovarian Development in Drosophila melanogaster. Academic Press, New York. 227 pp.

Félix, R. and M.E. de la Rosa. Genetics and Radiobiology Program of the National Commission of Nuclear Energy, Mexico. Cytogenetic studies with sodium cyclamate in D. melanogaster females.

It is now almost 30 years since the first highly effective mutagens were detected. Since then, much interest has been concentrated on the agencies which produce mutations and chromosome breaks. By the application of chemical compounds, some of whose effects on the constituents of the cell and nucleus are already known, it may be possible to obtain more information on the nature of the mechanisms involved in the production of genic and chromosomal alterations. Many agencies produce mutations and chromosome breaks. Their variety is such that it was wondered whether they do not achieve the same end effect by different means. Several mechanisms of mutagenesis were imagined, such as energy transfer to the chromosomes, chemical reactions with the genetic material, and interference with chromosome synthesis. Few of these would act equally well at all stages of the cell cycle. Thus, interference with chromosome synthesis should be restricted to interphase. Within the last two decades much interest has been concentrated on the variations in differential sensitivities exhibited by cells during gametogenesis in both plants and animals. These variations may be inherent in that they depend on processes or physiological states in the test object which, while sometimes recognized, cannot always be controlled, or they may be induced by different chemical and physical agents.

The reports of Sax and Sax (1968), D. Stone et al. (1969) and Legator et al. (1969) pointed out the cytogenetic damage induced by cyclamate and by its degradation product, cyclohexylamine, preceding the announcement of cyclamate's possible carcinogenic capabilities and the subsequent restrictions on dietary consumption.

Stone et al. (1969) demonstrated that cyclamate, in a minimum concentration of 200 microgram/ml can stiumlate chromosome breakage in human cells in vitro. Whereas a high dosage (equivalent to 15 g/175 Kg) was required to obtain a demonstrable increase in chromosome breaks, Stone pointed out that there is some evidence of synergistic actions on chromosome damage between X-irradiation and radiomimetic chemicals (Merz et al., 1961); between the chemical agents and virus (Nichols et al., 1965), and between the chemical agents themselves (Moutschen-Dahem, 1962).

A considerable number of workers are now engaged in testing compounds for their mutagenic activity. Since in many cases the publication of negative data is not warranted, there is often a repetition of effort with similar negative results.

This study is concerned with the production of X chromosome loss and non-disjunction in Drosophila females by sodium cyclamate. Obtaining identifiable meiotic stages is possible in the case of the female, since the meiotic divisions do not occur until after the oocyte is laid. To obtain fairly uniform samples of a single stage it is necessary to limit the period of egg collection, so that not more than one oocyte is recovered from each ovariole. On the other hand, in the male, meiosis occurs long before the completion of sperm development and the insemination of the female, and at best one can distinguish meiotic stages from pre and post-meiotic ones, with little hope of subdividing the various stages of development of the spermatocyte.

Radiation studies to date have largely been restricted to older stages of oocytes in the vitellarium. King et al. (1956) have described the structure of the ovariole in the adult female, and have designated 14 developmental stages of the oocyte. They recognize three sensitive groups on the basis of recessive lethal mutations, X chromosome losses, and dominant lethal effects. However, much of the work of others has been concerned with but two of the stages they describe, stages 7 and 14 (Parker, 1963), which are, respectively, the oldest stages in the newly emerged females and the fully mature, chorionated oocyte found in females ready to begin egg laying (usually during the second day of adult life). Apparently strains differ in the rate of egg production, as well as in the number of stage 14 oocytes in each ovariole in 4-day old females (Williamson and Stubblefield, 1970). In the control group of our experiment an average of 30 eggs were obtained in 24 hours which correspond mainly to stage 14 oocytes.

Bridges (1913) identified non-disjunction by the recovery of exceptional females and males among the progeny, by their being matroclinous and patroclinous, respectively, in phenotype for sex linked characters. In the present work an improved method for detecting non-disjunction and chromosome X loss that gives particularly reliable evidence concerning the origin of each exceptional female and male makes use of a tester male stock with attached $Y^{S}X.Y^{L}$ chromosomes. This stock was derived from translocations between the X and Y chromo-

somes and has the markers (y) yellow and (B) Bar.

The females were taken from the cross of stocks having the $sc^8Y$ chromosome: $y^2\ w^a/y^2\ w^a$; e/e x $y^2\ w^a/sc^8Y$; e/e. The existence of any secondary exceptions (from XXY mothers) among the $y^2\ w^a/y^2\ w^a$ females was made unlikely by the $sc^8Y$ chromosome which covers the effect of yellow in XXY females. The males have an attached $Y^SX{\cdot}Y^L$ chromosome of the genotype: $In(1)EN,Y^S$ B $y{\cdot}Y^L$. When virgin females are isolated, the marker ebony insures the virginity of such females, as the genotype of the females from the cross: $y^2\ w^a/y^2\ w^a$: e/e x $In(1)EN,Y^S$ B $y{\cdot}Y^L$; +/+ is heterozygous for the ebony marker. The fertilization of an XX egg with an XY spermatozoon would produce meta-females with low viability which were excluded from the following analysis, whereas the fertilization of eggs of the same non-disjunctional chromosomal constitution with a non X chromosome bearing spermatozoon would produce matroclinous yellow, white apricot females of the same genotype as their mothers. These females are easily identified from their normal Bar eyed sisters. The no-X egg when fertilized with an SY bearing sperm would become and XY patroclinous male, which can be identified by the Bar, yellow eyes in its phenotype.

Virgin females of the genotype $y^2\ w^a/y^2\ w^a$ were aged from 4 to 6 days and fertilized in cultures containing agar-cornmeal medium with sodium cyclamate. In each culture an aged female was mated with 3 attached $Y^SX{\cdot}Y^L$ males, and eliminated after 24 hours. All the cultures were kept at 25±1°C throughout the experiment.

The $F_1$ flies were scored for X-loss and non-disjunction from 13 to 15 days after the treatment with cyclamate of the P females. The female fruit fly has remarkable synthetic abilities, since during the period of maximum egg production it ingests a daily amount of yeast which approximately equals its body weight and manufactures from the raw material a quantity of eggs which approximately equals 1/3 its weight (King and Wilson, 1955).

Table 1. Progenies obtained from 28 cultures after feeding Drosophila adult females with media containing sodium cyclamate.

| NaCy mg/ml | Control | 0.05 | 0.10 | 0.20 | 0.40 | 0.80 | 1.60 |
|---|---|---|---|---|---|---|---|
| ♂ (m.p.c.) | 17.14 | 16.57 | 17.75 | 13.21 | 12.50 | 13.25 | 11.32 |
| ♀ (m.p.c.) | 12.46 | 9.79 | 13.46 | 10.82 | 8.29 | 9.11 | 7.82 |
| ♂/♀ (s.r.) | 1.37 | 1.69 | 1.32 | 1.22 | 1.51 | 1.45 | 1.45 |
| ♂ (total) | 480 | 464 | 497 | 370 | 350 | 371 | 317 |
| ♀ (total) | 349 | 274 | 377 | 303 | 232 | 255 | 219 |

NaCy mg/ml, sodium cyclamate, milligram/milliliter; (m.p.c.) mean per culture; (s.r.), sex ratio.

As no exceptional progeny were obtained among 4,858 flies, a second experiment was started, maintaining during 6 days, 1 female previously aged during 4 days and mated to 3 males, from the stocks described above, in each culture. The agar-cornmeal medium contained no sucrose. After six days the P flies were eliminated. The feeding of the progenitors as well as the egg laying, embryonic and larval development of the progeny occurred in the medium with cyclamate. The results of this second experiment are contained in Table 2.

Table 2. Progenies obtained from adult and larval feeding during six days in media without sucrose and several concentrations of sodium cyclamate.

| NaCy mg/ml | Control | 10.00 | 40.00 | 100.00 |
|---|---|---|---|---|
| ♂(m.p.c.) | 45.74 | 25.00 | 31.33 | 34.89 |
| ♀(m.p.c.) | 25.16 | 11.39 | 12.00 | 17.33 |
| ♂/♀(s.r.) | 1.82 | 2.19 | 2.61 | 2.01 |
| ♂ total | 869 | 575 | 658 | 314 |
| ♀ total | 478 | 262 | 252 | 156 |
| (n.c.) | 19 | 23 | 21 | 9 |
| (n.d.p.) | - | - | - | 4 |
| (c.l.p.) | - | - | 2 | 9 |

NaCy mg/ml, sodium cyclamate, milligram/milliliter; (m.p.c.), mean per culture; (s.r.), sex ratio; (n.c.), number of cultures; (n.d.p.), non-junctional progeny (c.l.p.), chromosome loss progeny.

From Day and Grell (1966) the non-disjunction frequencies are calculated by the expression:

$$\% \text{ non-disjunction} = \frac{4\ (\text{exc.}\ \varphi\varphi \times 100)}{\text{total} + \text{exc.}}$$

In multiplying the exceptional females by 4 and adding the exceptions to the denominator it is assumed that the number of exceptional males arising from non-disjunction is equivalent to the number of exceptional females and that one half of all XX and O oocytes are lost due to lethality.

The following expression was applied to calculate the frequencies of X chromosome loss among the

progenies: $\%\ \text{loss} = \frac{2\ (\text{exc. } ♂♂ - \text{exc. } ♀♀) \times 100}{\text{Total + exc.}}$

The excess of exceptional males over exceptional females is considered to arise from loss of an X-chromosome during meiosis and not from non-disjunction, as the excess of exceptional male progeny is best explained as arising from spontaneous loss (Mavor, 1924; Paterson et al., 1932; Sturtevant and Beadle, 1936; Uchida, 1962). Oocytes lacking an X will lead to viable progeny only when fertilized by an X sperm; hence a correction factor of 2 is used to account for the nullo-X oocytes fertilized by a Y sperm (Day and Grell; 1966).

From table 2, exceptional progenies was restricted only to the concentrations of 40 mg/ml and 100 mg/ml. The computation of such data gives the following percentages of non-disjunction and chromosome loss:

| NaCy mg/ml | 40 | 100 |
|---|---|---|
| XO | 0.44 | 2.07 |
| XX·Y | - | 3.31 |

A comparison among the data from Stone et al. (1969) on induced chromosome breakage in human cells in vitro with a minimum concentration of 0.20 mg/ml of cyclamate and the minimum effective dose from this experiment (40 mg/ml) demonstrates a difference of sensitivity, equivalent to two orders of magnitude. Although the two experimental designs are hardly comparable, it seems evident that Drosophila oocytes are much less sensitive to cyclamates than human cells in vitro.

In a third experiment female flies aged for 3 days were fed during 24 hours with sodium cyclamate dissolved in distilled water at concentrations of 50 mg/ml, 100 mg/ml, and 160 mg/ml. (See Technical Note by R. Félix). Each treated female was mated afterwards with 3 males in individual cultures containing normal agar-cornmeal medium. After 6 days of egg-laying the flies were eliminated and the progenies were scored after 13 to 15 days (Tab. 3)

Table 3. Progenies obtained from the feeding of adults with concentrated solutions of sodium cyclamate.

| NaCy mg/ml | 50.00 | 100.00 | 160.00 |
|---|---|---|---|
| ♂ (m.p.c.) | 30.75 | 36.25 | 35.31 |
| ♀ (m.p.c.) | 21.78 | 21.15 | 22.95 |
| ♂/♀ (s.r.) | 1.41 | 1.71 | 1.54 |
| ♂ (total) | 861 | 725 | 671 |
| ♀ (total) | 610 | 423 | 436 |
| (n.c.) | 28 | 20 | 20 |

NaCy mg/ml, sodium cyclamate, milligram/milliliter; (m.p.c.), mean per culture; (s.r.), sex ratio; (n.c.) number of cultures.

No exceptional progeny among 3,726 flies were obtained after the feeding of Drosophila with sodium cyclamate solution, in spite of the 160 mg/ml concentration, which is close to the saturation point.

Comparing the 100 mg/ml concentration in the food medium with the same concentration in solution, the size of the progenies are remarkably alike.

An aspect of the experiments, which called our attention, is the deviation of the sex ratio, indicating a lowered proportion of the X·XY females ($y^2w^a$/In(1)EN, $Y^S$, By·$Y^L$) as compared with the XO males ($y^2w^a$). The deviation that is also found in the control groups, is considerably magnified by the prolonged treatment of adults with cyclamate, as shown in Table 2.

References: Bridges, C.B., 1913. J. Exptl. Zool. 15: 587-606; Bridges, C.B. and K.S. Breheme, 1944. The mutants of Drosophila. Carnegie Inst. Washington, Pub. 552; Day, J.W., and R.F. Grell, 1966. Mutation Res. 3:503-509; King, R.C. and L.P. Wilson, 1955. V.J. Exptl. Zool. 130:71-82; King, R.C., A.C. Rubinson, R.F. Smith, 1956. Growth 20:121-157; Legator, S., K.A. Palmer, S. Green, K.W. Petersen, 1969. Science 165:1139; Lindsley, D.L. and E.H. Grell, 1967. Genetic Variations of D. melanogaster. Carnegie Inst. Washington, Pub. 627; Mavor, J.W., 1924. J. Exptl. Zool. 39:381-432 Mertz, T., C.P. Swanson, N.S. Cohan, 1961. Science 133:703; Moutschen-Dahem, J. and M. Moutschen-Dahem, 1962. Chem. Abstr. 56, 13270a; Nichols, W.W., A. Levan, W.K. Henen, M. Peluse, 1965. Hereditas 54: 213; Parker, D.R., 1963. In: Sobels, F.H. (Ed.) Repair from Genetic Radiation Damage, Pergamon Press, Oxford:11-19; Patterson, J.T., W. Brewster, A.M. Winchester, 1932. J. Heredity 23:325-333; Sax, K. and H.J. Sax, 1968. Jap. J. Genet. 43:89; Stone, D., E. Lamson, Y.S. Chang, K.W. Pickering, 1969. Science 164:568; Sturtevant, A.H. and G.W. Beadle, 1936. Genetics 21:554-604; Uchida, I.A., 1962. Can. J. Genet. Cytol. 4:402-408; Williamson, J.H. and P. Stubblefield, 1970. DIS 45:191.

Félix, R., J. Guzmán and A. de Garay Arellano. Genetics and Radiobiology Program. National Commission of Nuclear Energy. Mexico City, Mexico. Distribution of $CO_2$ sensitivity (sigma virus) in an urban population of D. melanogaster from Mexico City. II. Low dispersal, a factor which explains differences among locations.

Wright's (1955) model for optimum evolutionary opportunity is one of partially isolated sub-groups within which there is some opportunity for gene frequency shifts due to environmental differences between sub-populations or due to random processes (Crow, 1955). Chance gene frequency changes will be greater as populations become smaller, whereas fluctuations in selection coefficients have a similar effect in larger populations. Kimura (1955) worked on the mathematically difficult problems of constructing the stochastic process of gene frequency change under various assumed conditions. The general conclusions of Kimura's work were anticipated by the earlier writings of Wright. The fluctuations in gene frequencies in sub-populations may be provided by random shift in small populations or by random changes in selective values in larger ones.

Wright (1951) considered a model in which the population has a homogeneous structure, but offpring travel only a certain distance from their parents. If the range of dispersal is restricted in such a way that the parents of a particular individual may be assumed to be drawn at random from a neighborhood of a certain size, the amount of local differentiation may be related to the effective size of the neighborhood.

In the study of Wallace (1966a) of the allelism, a model was applied, which predicts that for lethals collected at different times, the logarithm of the frequency of allelism due to inbreeding, should decline linearly with time. On a strictly empirical basis, Wallace (1966b) substituted the square root of the distance for time in the original model. The relationship was suggested by the dispersal of flies from a point of release: the logarithm of the numbers of recaptured flies decreases linearly with the square root of distance from the point of release.

Wallace analyzed the data of local collections of several species of Drosophila given by Dobzhansky and Wright (1943), by Burla et al. (1940) and by Timofeeff-Ressovsky (1941 a,b), which show that, despite its seemingly well developed powers of dispersal, (revealed by the migration of individual flies for several hundred meters in two days), more than one-quarter of the D. pseudoobscura captured at a given point have probably arisen within a radius of 25 meters from that point; one-eight of such collection of flies have probably arisen within a radius of ten meters. The data for D. willistoni reveal that this species is ever more sedentary; nearly one third of a collection of these flies have probably arisen within a radius of some ten meters from the point of capture. The data of Dobzhansky and Wright on the dispersal of D. melanogaster, suggest that the dispersal of this species resembles that of D. funebris and D. willistoni more closely than that of D. pseudoobscura (Wallace, 1966b).

When the linear relationship between the logarithm of the frequency and the square root of distance is plotted on a conventional arithmetical scale, the transformed curve clings to both the vertical and the horizontal axes. Wallace gives the following interpretation of this theoretical curve: immigrants form a small fraction of any population but, given that an individual is an immigrant, he is very nearly as likely to have come from any one distance as from another. The tall vertical portion of the curve, which hugs the Y axis means that the bulk of all individuals found at one spot has arisen initially at or very near that spot. Bateman (1947) refers to this feature of the dispersal pattern, pointing out its effect of subdividing what would otherwise appear to be a continuous population.

In this report, data are given, which suggest the low dispersal of D. melanogaster flies, when the percentage of sensitive flies receovered from neighboring collection sites is recorded.

Brun and Sigot (1955) pointed out the differences among stabilized and non-stabilized $CO_2$-sensitive strains of Drosophila. In stabilized strains there is a maternal effect which assures the sensitivity to all the progeny, whereas stabilized males transmit the sigma virus in his sperm to a fraction of the offspring. Non-stabilized lines, on the other hand, typically produce resistant and sensitive progeny. The role of infectious virus sigma may be clarified by future investigations of the relationship between sigma and the Drosophila sensitivity to $CO_2$ (Seecof, 1962).

L'Héritier (1958), L'Héritier and Plus (1963) made an extensive research on the variations of the genotype of the virus, as well as on the genetic factors of the host which affects his hereditary transmission. Every quantitative aspect of the relationship between virus sigma and its host seems to be more or less genotype dependent.

The $CO_2$-sensitivity itself, is not a selective factor to be considered in population studies, as the concentration of the gas required to produce anoxia in Drosophila, is never found in natural conditions. However, the presence of sensitive, resistant, and "refractory" flies in the same population, points out that the presence of the hereditary virus is not a completely neutral character.

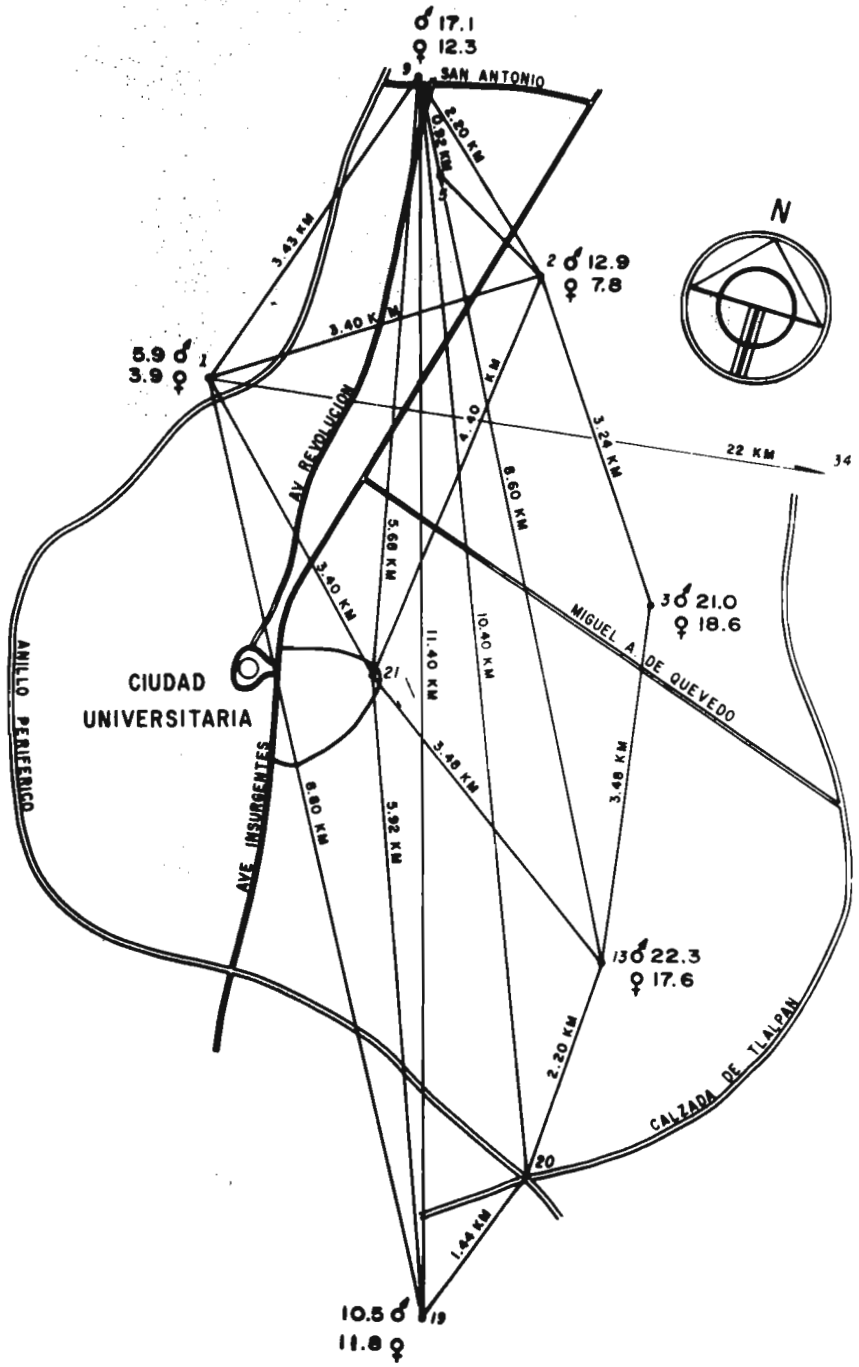



Fig. 1. Distribution of $CO_2$ sensitivity of D. melanogaster. Percentages of sensitive males and females are indicated at six locations.

Seecof (1964) investigated the deleterious effects of the sigma virus infection of D.

melanogaster, demonstrating a direct relationship among deaths of the infected progeny and the logarithm of the number of infectious units inherited by the progeny. Seecof tested the effect of the variation of environmental conditions, looking for a selective advantage of the flies when they are infected. As a result, the infection proved to be a handicap under all the conditions tested. The presence of the virus in wild populations of flies, in absence of any known vector, is therefore, unexplained. However, the crowding of flies did not reduce the proportion of $CO_2$-sensitive flies in the cultures. There is to be found, a compensating selective advantage for sigma-infected flies, in order to explain the proportion and persistence of such flies in wild populations.

In the present survey, the proportion of $CO_2$-sensitive flies from six locations in Mexico City was tested by submitting them to the treatment with pure $CO_2$ at 8°C during 15 minutes. Collections were made from June 1969 to May 1970, in a southwest section of Mexico City. Flies were attracted to 1/4 liter containers of decaying cantaloupe. The spatial distribution of $CO_2$-sensitivity, as well as the recorded averages of the monthly percentages of sensitive males and females from six locations are shown in Fig. 1. Only the data from the locations where the collections were significant, are included. The minimum and the maximum distances among locations, were respectively, 2.20 and 11.40 Km. The extreme difference of $CO_2$-sensitivity was found among locations 1, and 3, approximately 4.70 Km. apart.

Monthly percentages of $CO_2$-sensitivity in both sexes, from each location, are given in tables 1 and 2. The annual averages of the monthly percentages of sensitive males and females are shown in Table 3. These averages, are constantly larger for males than for females (ex-

Table 1. Percentage of $CO_2$-sensitivity of males collected at six locations from Mexico City.

| | June | | July | | August | | September | | October | | November | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Loc. | N/S | %S | N/S | %S | N/S | %S | N/S | %S | N/S | %S | N/S | %S |
| 1 | 106/6 | 5.6 ±2.02 | 60/5 | 8.3 ±3.56 | 575/28 | 4.8 ±0.88 | 1426/139 | 9.7 ±0.78 | 261/33 | 12.6 ±2.05 | 60/1 | 1.8 ±1.33 |
| 2 | 215/32 | 14.8 ±2.39 | 198/30 | 15.1 ±2.54 | 213/28 | 13.1 ±2.31 | 43/5 | 11.6 ±4.88 | 18/4 | 22.2 ±9.79 | - | - |
| 3 | 117/9 | 7.6 ±2.44 | 339/32 | 9.4 ±1.69 | 130/25 | 19.2 ±3.45 | 149/16 | 10.7 ±2.53 | 150/0 | 0.0 | - | - |
| 9 | - | - | 75/11 | 14.6 ±4.07 | - | - | 61/3 | 4.9 ±2.76 | 26/1 | 3.8 ±3.74 | - | - |
| 13 | 60/23 | 38.3 ±6.27 | - | - | 93/14 | 15.0 ±3.70 | 18/5 | 27.7 ±10.54 | - | - | - | - |
| 19 | - | - | 70/10 | 14.2 ±4.17 | 52/4 | 7.6 ±2.01 | 158/11 | 6.9 ±2.01 | 101/6 | 5.9 ±2.34 | 46/1 | 2.1 ±2.11 |
| | 498/70 | 14.06 ±1.66 | 742/88 | 11.86 ±1.49 | 1063/99 | 0.3 ±0.99 | 1855/179 | 9.64 ±0.75 | 556/44 | 7.91 ±1.33 | 106/2 | 1.89 ±1.31 |

| | December | | January | | February | | March | | April | | May | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Loc. | N/S | %S | N/S | %S | N/S | %S | N/S | %S | N/S | %S | N/S | %S |
| 1 | 126/3 | 2.3 ±1.75 | - | - | 90/3 | 3.3 ±1.88 | 73/2 | 2.7 ±1.87 | 259/21 | 8.1 ±1.69 | 658/39 | 5.9 ±0.91 |
| 2 | 68/0 | 0.0 | - | - | - | - | 71/3 | 4.2 ±2.38 | 146/12 | 8.2 ±2.51 | - | - |
| 3 | 15/2 | 13.3 ±8.76 | - | - | - | - | - | - | 45/13 | 28.8 ±6.75 | 14/8 | 57.1 ±13.22 |
| 9 | - | - | - | - | - | - | - | - | 114/26 | 22.8 ±3.92 | 38/15 | 39.4 ±7.92 |
| 13 | 21/0 | 0.0 | - | - | - | - | - | - | 14/1 | 7.1 ±6.34 | - | - |
| 19 | - | - | - | - | - | - | - | - | 19/5 | 26.3 ±10.10 | - | - |
| | 230/5 | 2.17 ±1.77 | | | 90/3 | 3.33 ±1.88 | 144/5 | 3.47 ±1.52 | 597/78 | 13.07 ±1.55 | 710/62 | 8.73 ±1.78 |

N, number of collected flies; S, number of sensitive flies; %S, percentage of $CO_2$-sensitive flies ± standard error.

Table 2. Percentage of $CO_2$-sensitivity of females collect at six locations from Mexico City.

| | June | | July | | August | | September | | October | | November | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Loc. | N/S | %S | N/S | %S | N/S | %S | N/S | %S | N/S | %S | N/S | %S |
| 1 | 75/0 | 0.0 | 41/2 | 4.8 ±3.34 | 629/33 | 5.2 ±0.88 | 1247/51 | 4.0 ±0.55 | 255/20 | 7.8 ±0.60 | 41/2 | 4.8 ±3.33 |
| 2 | 216/19 | 8.7 ±1.92 | 177/21 | 11.8 ±2.42 | 222/34 | 15.3 ±2.42 | 31/3 | 9.6 ±5.29 | 14/1 | 7.1 ±6.86 | - | - |
| 3 | 58/4 | 6.9 ±3.32 | 337/36 | 10.6 ±1.63 | 39/5 | 12.8 ±5.35 | 152/18 | 11.8 ±2.61 | 9/1 | 11.1 ±10.47 | - | - |
| 9 | - | - | 58/5 | 8.6 ±3.68 | - | - | 54/2 | 3.7 ±2.56 | - | - | - | - |
| 13 | 53/29 | 54.7 ±6.83 | - | - | 65/11 | 16.9 ±4.64 | 17/0 | 0.0 | - | - | - | - |
| 19 | - | - | 51/2 | 3.9 ±4.81 | 48/2 | 4.1 ±2.66 | 107/8 | 7.4 ±2.5 | 137/11 | 8.0 ±2.31 | 18/0 | 0.0 |
| | 402/52 | 12.99 ±2.12 | 664/71 | 10.69 ±1.15 | 1003/85 | 8.47 ±0.71 | 1608/116 | 7.21 ±0.65 | 415/33 | 7.95 ±1.37 | 59/2 | 3.39 ±2.16 |

| | December | | January | | February | | March | | April | | May | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Loc. | N/S | %S | N/S | %S | N/S | %S | N/S | %S | N/S | %S | N/S | %S |
| 1 | 82/0 | 0.0 | - | - | 93/3 | 3.2 ±1.82 | 56/2 | 3.4 ±2.46 | 164/5 | 3.0 ±1.33 | 487/34 | 6.9 ±1.14 |
| 2 | 42/1 | 2.3 ±2.31 | - | - | - | - | 108/2 | 1.8 ±1.28 | 212/11 | 5.1 ±1.69 | - | - |
| 3 | - | - | - | - | - | - | - | - | 31/7 | 22.5 ±7.5 | 29/17 | 58.6 ±9.14 |
| 9 | 13/1 | 7.6 | - | - | - | - | - | - | 103/22 | 21.3 ±4.03 | 49/10 | 20.4 ±5.75 |
| 13 | 30/1 | 3.3 ±3.26 | - | - | - | - | 11/1 | 9.0 ±8.62 | 23/1 | 4.3 ±2.05 | - | - |
| 19 | - | - | - | - | - | - | - | - | 27/7 | 25.9 ±8.43 | - | - |
| | 167/3 | 1.80 ±1.58 | | | 93/3 | 3.23 ±1.82 | 175/5 | 2.86 ±1.60 | 560/53 | 9.46 ±1.46 | 565/61 | 10.80 ±1.90 |

N, number of collected flies; S, number of sensitive flies; %S, percentage of $CO_2$-sensitivity ± standard error.

Table 3. Total number of male and female flies collected at six locations from Mexico City and percentage of $CO_2$ sensitivity ± standard error.

| | Males | | Females | |
|---|---|---|---|---|
| Loc. | Totals | %S | Totals | %S |
| 1 | 3,694 | 5.92 ±0.38 | 3,170 | 3.92 ±0.33 |
| 2 | 972 | 12.99 ±1.08 | 1,022 | 7.89 ±0.84 |
| 3 | 959 | 21.09 ±1.32 | 655 | 18.62 ±1.52 |
| 9 | 314 | 17.14 ±2.13 | 277 | 12.32 ±1.97 |
| 13 | 206 | 22.36 ±2.89 | 199 | 17.64 ±2.70 |
| 19 | 446 | 10.51 ±1.45 | 388 | 11.82 ±1.64 |
| | 6,591 | 14.99 ±0.44 | 5,711 | 12.04 ±0.44 |

cluding location 19). Such a difference suggests that Y bearing spermatozoa are more effective in transporting the virus than X bearing spermatozoa, or that there exists a differential egg-adult survival depending on sex of infected flies among the progenies.

The differences of sensitivity among adjoining trapping sites may be explained by the relative isolation of the micropopulations, resulting from the low dispersal of Drosophila. The dispersal rate is perhaps further diminished in these densely inhabited urban areas, as compared with the field populations previously studied.

A Drosophila population which has been decimated during the winter and builds up again from a few new founder individuals will have a vastly different genetic structure than a population at the height of population density. There must be a "bottleneck" in the winter months in Mexico City, as it was impos-

sible to collect any flies during January.

The variations of the percentages of $CO_2$-sensitivity in two localities (3 and 13), 2.48 Km. apart are shown in Fig. 2 and 3. The collections included the winter months, when the dwindling of the population took place. The pattern of variation of the proportion of sensitive flies differs considerably in the two trapping sites. Such differences may be due to a drift during the rebuilding of the populations, starting with a small population density, and to the low dispersal rate of urban Drosophila flies.

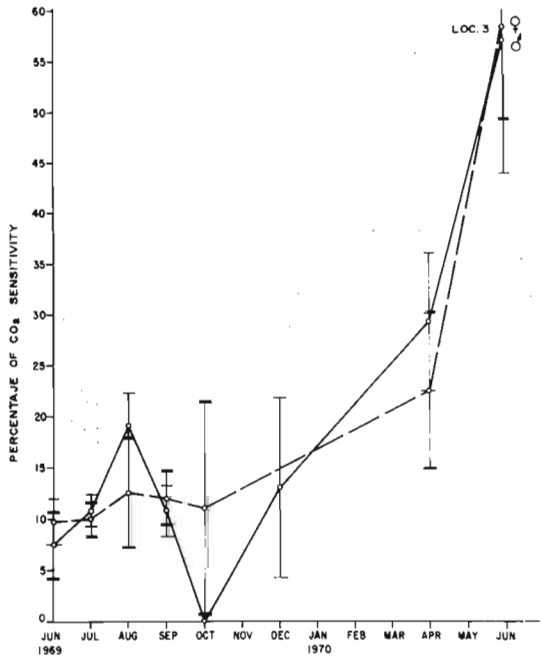



Fig. 2. Variation of $CO_2$-sensitivity percentage in location 3.

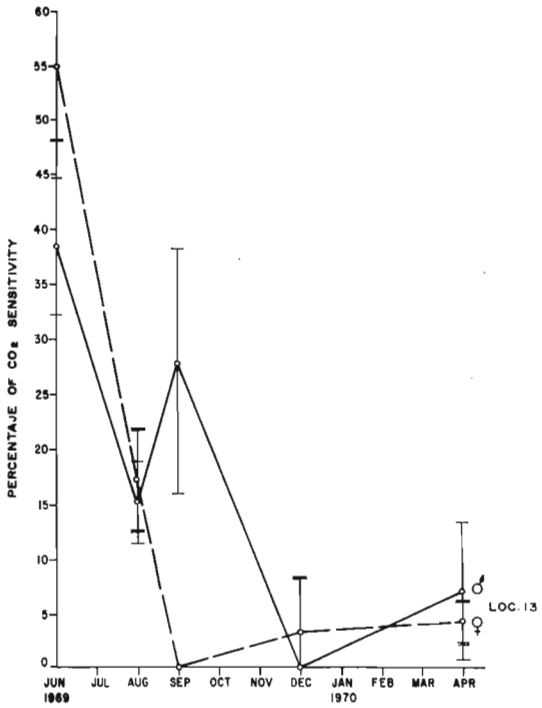



Fig. 3. Variation of $CO_2$-sensitivity percentage in location 13.

References: Bateman, A.J., 1947. Heredity 1:303-306; Brun, G., and A. Sigot, 1955. II. Ann. Inst. Pasteur 88:488-512; Burla, L., A.B. Dacunha, A.G.L. Cavalcani, TH., Dobzhansky and C. Pavan, 1940. Ecology 31:393-404; Crow, J.F., 1955. In: Brehme, K.W. (Ed) Cold Spring Harbor Symposia on Quantitative Biology XX. The Biological Laboratory. Cold Spring Harbor, L.I., New York: 54-59; Dobzhansky, TH. and S. Wright, 1943. Genetics 28(1): 304-340; Kimura, M., 1955. In: Brehme, K.W. (Ed.) Cold Spring Harbor Symposia on Quantitative Biology XX. The Biological Laboratory. Cold Spring Harbor, L.I., New York: 33-53; L'Heritier, P., 1958. Adv. Virus Res. 5:195-245; L'Heritier, P. and N. Plus, 1963. In: Harris, R.J.C. (Ed.) Biological Organization at the Cellular and Supercellular Level. Acad. Press. London and New York: 59-71; Seecof, R.L., 1964. In: Brehme, K.W. (Ed.) Cold Spring Harbor Symposia on Quantitative Biology XVII. The Biological Laboratory. Cold Spring Harbor, L.I., New York: 501-512; Timofeeff-Ressovsky, N.W. and H.A. Timofeeff-Ressovsky, 1941a. Z. Indukt.-Vererb. 79:28-34; Timofeeff-Ressovsky, N.W. and H.A. Timofeeff-Ressovsky, 1941b. Z. Indukt.-Vererb. 79: 35-43; Wallace, B., 1966a. Amer. Natur. 100 (916): 565-578; Wallace, B., 1966b. Amer. Natur. 100(916): 551-563; Wright, S., 1951. Genetics 16(1): 97-159; Wright, S., 1955. In: Brehme, W.K. (Ed.) Cold Spring Harbor Symposia on Quantitative Biology: XX. The Biological Laboratory. Cold. Spring Harbor, New York: 16-24.

Félix R., J. Guzmán and A. de Garay Arellano. Genetics and Radiobiology Program. National Commission of Nuclear Energy. Mexico City, Mexico. $CO_2$ sensitivity of Drosophilid flies from a location in the outskirts of Mexico City.

Among natural populations of Drosophilids some individuals are readily found which show a physiological anomaly, sharply outlined and easy to recognize, when brought in contact with carbon dioxide. Sensitive flies will not recover after being anaesthetized with $CO_2$ but, rather, will remain paralyzed and eventually die (L'Héritier and Teissier, 1945). In most respects the infection is typical of those caused by animal viruses (L'Héritier, 1958; Seecof, 1962), but there are several aspects of the infection which make it noteworthy.

$CO_2$-sensitive strains show a maternal effect which assures sensitivity to all the progeny, whereas non-stabilized lines tipically throw resistant as well as sensitive progeny (Goldstein, 1949). The non-stabilized hereditary transmission pattern is also displayed by flies that receive sigma virus initially by infection (L'Héritier, 1951).

From June 1969 to May 1970, a survey was made on the distribution of $CO_2$-sensitivity of D. melanogaster collected at six locations from the south-west of Mexico City. The bait used for trapping was fermented cantaloupe, and the proportion of sensitive flies was obtained by submitting all the collected samples to the treatment with pure $CO_2$ at $8^{\circ}$ during 15 minutes.

A first survey of Drosophilid species gathered in the same traps was done from October 1969, to March, 1970. Since the flies were trapped incidental to collecting samples of D. melanogaster, and since but one collecting technique was employed, the list of species is no doubt incomplete.

The distribution of the collected species; immigrans, hydei, busckii, pseudoobscura and Drosophila sp. (repleta group) shows a scarce dispersion into the trapping sites located in the urban area. It was possible to collect significant, although small, numbers of adults of the five species, only in location 1 (See: Felix, R. et al., 1971). The dominant population at this trapping site was a non-identified species of the repleta group. As location 1 is situated in a house in the outskirt of the city, the collected specimens are immigrants from the non-urban area surrounding this place, scarcely inhabited by man, and with an abundant arboreus vegetation (Cupressus lindleyi Krotsch, Casuarina equisetifolia L., Eucalyptus globulus L. and Schinus molle L.)

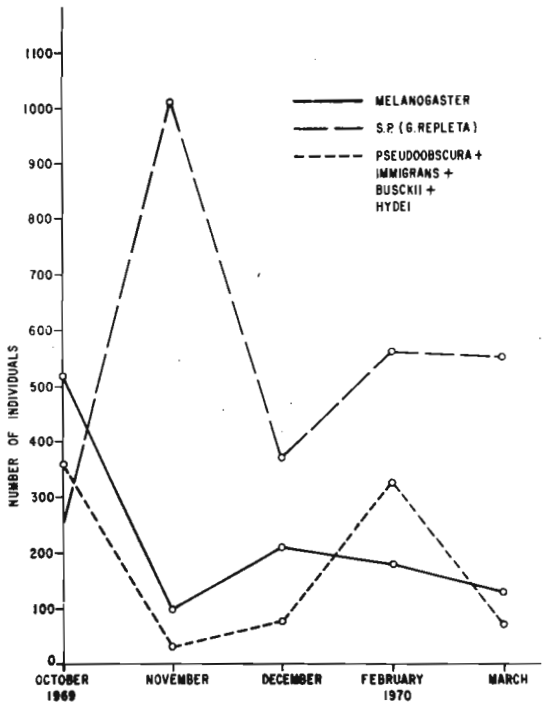



Table 1 shows the relative abundance of each species and its percentage of $CO_2$-sensitivity throughout the winter months. Drosophilids, other than D. melanogaster do not migrate into urban area, as only about 300 individuals belonging to the five species named above were collected at five trapping sites located in the densely inhabited urban area, at distances from 3.43 to 8.80 Km. apart from location 1 (Fig. 1).

Fig. 1. Relative abundance of Drosophilid species collected at location 1.

The data on the sensitivity related to sex, shows that the values for males are consistently larger than that for females (excluding D. busckii). The species which showed the largest proportion of $CO_2$-sensitivity is D. immigrans. No sensitive flies were found among 145 tested females of D. pseudoobscura, which constitutes an interesting feature of this species in a suburban area.

Table 2 shows the relative abundance, as compared to D. melanogaster, collected at the same traps, of the five species from the locations 1, 9 and 19.

Table 1. Relative abundance and percentage of $CO_2$ sensitivity of Drosophilid species collected at location 1. N, number collected; S, number of sensitive flies; %S, percentage of sensitive flies ± standard error.

| | October | | November | | December | | February | | March | | | Mean |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Species | N/S | %S | N/S | %S | N/S | %S | N/S | %S | N/S | %S | Totals | %S |
| D. immigrans | | | | | | | | | | | | |
| | 110/19 | 17.3 ±3.60 | 18/0 | 0.0 | 19/0 | 0.0 | 2/0 | 0.0 | 2/0 | 0.0 | 110 | 17.3 ±3.60 |
| | 80/7 | 8.7 ±3.15 | 3/0 | 0.0 | 12/0 | 0.0 | 1/0 | 0.0 | 2/0 | 0.0 | 80 | 8.7 ±3.15 |
| | 190/27 | 13.7 ±2.48 | | | | | | | | | 190 | 13.7 ±2.48 |
| D. hydei | | | | | | | | | | | | |
| | 36/9 | 25.0 ±7.21 | 12/2 | 16.7 | 11/0 | 0.0 | 33/0 | 0.0 | 11/1 | 9.1 | 69 | 12.50 ±2.97 |
| | 25/1 | 4.0 ±3.92 | 5/0 | 0.0 | 6/0 | 0.0 | 11/0 | 0.0 | 4/0 | 0.0 | 36 | 2.00 ±1.24 |
| | 61/0 | 16.4 ±4.73 | | | | | 44/0 | 0.0 | | | 105 | 8.20 ±1.89 |
| D. melanogaster | | | | | | | | | | | | |
| | 261/33 | 12.6 ±2.05 | 60/1 | 1.8 ±1.33 | 126/3 | 2.3 ±1.65 | 90/3 | 3.3 ±1.88 | 73/2 | 2.7 ±1.87 | 610 | 4.54 ±1.40 |
| | 255/20 | 7.8 ±0.60 | 41/2 | 4.8 ±3.33 | 82/0 | 0.0 | 93/3 | 3.2 ±1.82 | 56/2 | 3.5 ±2.46 | 527 | 3.86 ±1.79 |
| | 516/53 | 10.3 ±1.57 | 101/3 | 3.0 ±1.67 | 208/3 | 1.4 ±0.31 | 183/6 | 3.3 ±1.21 | 129/4 | 3.1 ±1.53 | 1,137 | 4.21 ±0.71 |
| Drosophila sp. (repleta group) | | | | | | | | | | | | |
| | 165/20 | 12.1 ±2.59 | 849/3 | 0.4 ±0.21 | 202/0 | 0.0 | 410/17 | 4.1 ±0.98 | 427/5 | 1.2 ±0.50 | 2,053 | 3.56 ±0.26 |
| | 88/9 | 10.2 ±3.23 | 161/1 | 0.6 ±0.61 | 174/0 | 0.0 | 152/0 | 0.0 | 125/0 | 0.0 | 700 | 2.16 ±0.54 |
| | 253/29 | 11.4 ±3.35 | 1010/4 | 0.4 ±0.20 | 376/0 | 0.0 | 562/17 | 3.0 ±0.74 | 552/5 | 0.9 ±0.40 | 2,753 | 3.14 ±0.11 |
| D. busckii | | | | | | | | | | | | |
| | 50/1 | 2.0 ±2.07 | 11/0 | 0.0 | | | | | | | 61 | 1.00 ±1.27 |
| | 30/2 | 6.7 ±2.05 | 12/0 | 0.0 | | | | | | | 42 | 3.35 ±2.75 |
| | 80/3 | 3.7 ±2.11 | 23/0 | 0.0 | | | | | | | 103 | 1.85 ±1.32 |
| D. pseudoobscura | | | | | | | | | | | | |
| | 78/2 | 2.5 ±1.76 | | | 19/0 | 0.0 | 191/2 | 1.1 ±0.71 | 69/1 | 1.4 ±1.41 | 357 | 1.25 ±0.57 |
| | 17/0 | 0.0 | | | 6/0 | 0.0 | 89/0 | 0.0 | 33/0 | 0.0 | 145 | 0.00 |
| | 95/2 | 2.1 ±1.46 | | | 25/0 | 0.0 | 280/2 | 0.7 ±0.48 | 102/1 | 1.0 ±0.93 | 502 | 0.95 ±0.42 |

Table 2. Relative abundance of species collected at locations 1, 9 and 19. N, number collected; sp./mel., species/melanogaster.

| | | October | | November | | December | | February | | March | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Species | Loc. | N | sp./mel. | N | sp./mel. | N | sp./mel. | N | sp./mel. | N | sp./mel. | Totals | sp./mel. |
| Melanogaster | 1 | 516 | 1.00 | 101 | 1.00 | 208 | 1.00 | 183 | 1.00 | 129 | 1.00 | 1,137 | 1.00 |
| " | 9 | 34 | 1.00 | 0 | - | 22 | 1.00 | 0 | - | 25 | 1.00 | 81 | 1.00 |
| " | 19 | 238 | 1.00 | 64 | 1.00 | 0 | - | 0 | - | 0 | - | 302 | 1.00 |
| Sp. (repleta) | 1 | 253 | 0.49 | 1,010 | 10.00 | 376 | 1.81 | 562 | 3.07 | 552 | 4.28 | 2,753 | 2.42 |
| " | 9 | 3 | 0.09 | 12 | - | 5 | 0.23 | 0 | - | 0 | - | 20 | 0.25 |
| " | 19 | 0 | - | 0 | - | 0 | - | 0 | - | 19 | - | 19 | 0.06 |
| Pseudoobscura | 1 | 95 | 0.18 | 0 | - | 25 | 0.12 | 280 | 1.53 | 102 | 0.79 | 502 | 0.44 |
| " | 9 | 0 | - | 0 | - | 3 | 0.14 | 0 | - | 22 | 0.88 | 25 | 0.31 |
| " | 19 | 0 | - | 0 | - | 0 | - | 0 | - | 0 | - | 0 | - |
| Immigrans | 1 | 190 | 0.37 | 21 | 0.21 | 31 | 0.15 | 3 | 0.02 | 5 | 0.04 | 250 | 0.21 |
| " | 9 | 34 | 1.00 | 13 | - | 0 | - | 0 | - | 2 | 0.08 | 49 | 0.60 |
| " | 19 | 4 | 0.02 | 0 | - | 0 | - | 0 | - | 0 | - | 4 | 0.01 |
| Hydei | 1 | 61 | 0.11 | 17 | 0.17 | 17 | 0.08 | 44 | 0.24 | 15 | 0.11 | 154 | 0.14 |
| " | 9 | 0 | - | 0 | - | 4 | 0.27 | 0 | - | 9 | 0.36 | 13 | 0.16 |
| " | 19 | 0 | - | 0 | - | 0 | - | 0 | - | 0 | - | 0 | - |
| Busckii | 1 | 80 | 15.5 | 23 | - | 0 | - | 0 | - | 0 | - | 103 | 0.09 |
| " | 9 | 44 | 1.29 | 0 | - | 0 | - | 0 | - | 0 | - | 44 | 0.54 |
| " | 19 | 0 | - | 0 | - | 0 | - | 0 | - | 0 | - | 0 | - |

Acknowledgments: The authors are most grateful to Dr. M.R. Wheeler, for the classification of the Drosophilid species collected during the present survey.

References: Félix, R., J. Guzmán and A. de Garay Arellano, 1971. DIS; Goldstein, L., 1949. Bull. biol. 83:177-188; L'Héritier, P., 1951. In: Harris, R.G. (Ed.). Cold Spring Harbor Symposia on Quantitative Biology XVI. The Biological Laboratory. Cold Spring Harbor, L.I., New York; L'Héritier, P., 1958. Adv. Virus Res. 5:195-245; L'Héritier, P. and G. Teissier, 1945. Publs. lab. Ecole Norm. Sup. Paris 1:35-74; Seecof, R.L., 1962. In: Harris, R.G. (Ed.) Cold Spring Harbor Symposia in Quantitive Biology, XXVII. The Biological Laboratory. Cold Spring Harbor, L.I., New York.

Gupta, J.P. Banaras Hindu University, Varanasi, India. Key to Indian species of subgenus Scaptodrosophila.

During last few years taxonomists and geneticists in India have reported several new and unrecorded species of Drosophila, among which seven species belong to the subgenus Scaptodrosophila so far. A taxonomic key is given here to distinguish them with an addtional note on their distribution.

1. Mesonotum and scutellum unicolorous.....2
   Mesonotum and scutellum not unicolorous.....3
2. Tarsal segments of male fore legs with many long curved upright hairs .....latifshahi Gupta and Ray-Chaudhuri
   Tarsal segments of male fore legs with no such hairs.....4
3. Mesonotum and scutellum with silvery white striations arranged longitudinally .....silvalineata Gupta and Ray-Chaudhuri
   Mesonotum and scutellum with scattered silvery white spots arranged longitudinally .....chandraprabhiana Gupta and Ray-Chaudhuri
4. Posterior parameres forming a triangular flap-like structure .....paratriangulata Gupta and Ray-Chaudhuri
   Posterior parameres not forming a triangular flap-like structure.....5
5. Heel observable and produced into a large spur-like projection .....ebonata Parshad and Duggal
   Heel observable but not produced into a spur-like projection.....6

6. Acrostichal hairs in six rows. $Or_2$ less than half of vibrissa .....bryani Malloch
Acrostichal hairs in eight rows. $Or_2$ not differentiated .....bambuphila Gupta

| Species | Source | Locality |
|---|---|---|
| D. chandraprabhiana | Bait | Chandraprabha (Chakia forest, Varanasi), Sirsi Dam (Mirzapur) |
| D. silvalineata | Bait | Chandraprabha (Chakia forest, Varanasi). |
| D. paratriangulata | Bait | Chandraprabha (Chakia forest, Varanasi); River Bank colony (Lucknow); Ayurvedic garden (B.H.U.). |
| D. latifshahi | Bait | Chandraprabha, Latifshah (Chakia forest, Varanasi); River bank colony (Lucknow). |
| D. ebonata | Bait | Srinagar, Pahalgam (Kashmir valley). |
| D. bryani | Bait and sweeping | Old Botanical garden (B.H.U.) |
| D. bambuphila | Bait and sweeping | Old Botanical garden (B.H.U.); Jatili near Padmapur (Berhampur). |

Franklin, I.R. C.S.I.R.O. Division of Animal Genetics, Epping, N.S.W. Genetic variation at the Esterase-6 locus in D. melanogaster.

Wright (1963) in describing the Esterase-6 polymorphism in D. melanogaster reported two alleles, Est-$6^S$ and Est-$6^F$. Subsequently Rodino and Martini (DIS 46:139) have reported a third allele, Est-$6^V$. In a number of samples of D. melanogaster from the Hunter Valley, N.S.W. four alleles have been observed, and a fifth seen rarely in other collections. Extension of the above notation would result in a cumbersome terminology, and I have followed Hubby and Lewontin (1966) in using the following designation -- Est-$6^{1.0}$, Est-$6^{1.1}$, Est-$6^{1.15}$, and Est-$6^{1.25}$. The first two are equivalent to Est-$6^S$ and Est-$6^F$, and Est-$6^V$ probably corresponds to Est-$6^{1.25}$. (The fifth allele alluded to above is slower than Est-$6^{1.0}$, and has been represented Est-$6^{0.95}$).

The frequencies of the four most common alleles are quite constant from site to site, and the genotypic frequencies are shown in Table 1.

Table 1. Genotypic frequencies at the Est-6 locus

| | Genotype | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 1.0/1.0 | 1.0/1.1 | 1.0/1.15 | 1.0/1.25 | 1.1/1.1 | 1.1/1.25 | 1.25/1.25 | Others | Total |
| Numbers | 376 | 234 | 7 | 45 | 52 | 7 | 2 | - | 714 |
| Frequency | .514 | .328 | .010 | .063 | .073 | .010 | .003 | - | |

The frequencies of the four alleles are 0.714, 0.242, 0.005, and 0.039.

Some additional data on the location of the Est-6 locus have been collected in test-crosses to 'rucuca'. Using a similar experiment to that described by Wright (1963), 185 flies showing recombination between hairy and thread were tested for their genotype at the Est-6 locus. In 81 cases the recombination had occurred between thread and Est-6. Wright observed 57 out of 149 tested. Pooling these data the location of the Est-6 locus is 3-35.9±0.5.

References: Wright, T.R.F. 1963 Genetics 48:787; Hubby, J.L. and Lewontin, R.C. Genetics 54:577.

Félix, R. and M.E. de la Rosa. Genetics and Radiobiology Program of the National Commission of Nuclear Energy, Mexico. Cytogenetic studies with cyclohexylamine in D. melanogaster females.

The artificial sweetener sodium cyclamate, increases chromosome breaks when added in relative high concentrations in human leukocytes in vitro (Stone et al., 1968), as well as in monolayer cultures derived from human skin and carcinoma of the larynx (Stone et al., 1969). The same compound fails to induce chromosomal damage in Haworthia variegata Haw (Majumdar and Lane, 1970). This inability is probably due to the fact that chemical agents that break animal chromosomes may not induce chromosomal aberrations in plants, and different plant genera may react in different ways to the same agent as is found in mammals (Brodie, 1965).

Legator (1968), and Legator et al. (1969) demonstrated that cyclohexylamine, a breakdown product of cyclamate, also induces chromosome breaks in vitro in rodent cells in culture, as well as in vivo, in rat spermatogonia.

The Food Additives and Contaminants Committee provides evidence on the transformation of cyclamates to cyclohexylamine in persons ingesting cyclamates for 2 to 3 days (Food Additives and Contaminants Committee, 1967).

In another line of research a cyclamate-saccharin mixture was fed to male and female rats (FDRL strain, Wistar derived). The doses received in the food varied from 0 to 2,500 mg/Kg/day. Cyclohexylamine was assayed in the urine using the procedure of Derse and Daun (1966), omitting the oxidative steps. The research was carried on, in order to determine whether the conversion of cyclamate to cyclohexylamine takes place in the gastrointestinal tract or systematically as a biotransformation product. In the latter case, the reason for the differences on metabolic handling and the possibility for its genetical control would be of particular interest, considering the similar findings in man (Oser et al., 1968).

Kojima and Ichibagase (1966) and Leahy et al. (1967) also found that cyclamate is metabolized to cyclohexylamine in dogs and man. The most difficult areas in which to determine a cause-and-effect relationship in the human population are in the assessment of carcinogenicity, mutagenicity, or teratogenicity after exposure of the subject to a specific compound. Because of the long latent period between exposure and expression of effects as well as the high background rate of damage, it is difficult to detect effects of a given agent in the population even after years of exposure. Induction of chromosome damage is one of the methods used to evaluate potencial carcinogenic, mutagenic, or teratogenic effects of the cyclamate metabolite.

The development of bladder neoplasms was reported in the Wistar strain of rats fed with cyclamate of saccharin (Price et al., 1970). There is no evidence that the use of cyclamate or saccharin has caused cancer in man, malformations in children, or any other abnormality in humans other that a rare skin hypersensitivity. However, in view of the requirements of the Delaney clause of the Food Additives Amendment, the removal of cyclamates from the classification of substances generally recognized as safe resulted in the prohibition of their use in general purpose food products.

The present report contains preliminary results on the sensitivity of oocytes of D. melanogaster using the adult feeding method. The process of oogenesis in adult females has already been described in detail (King et al., 1956), and the main features of germ-cell stage sensitivity to some mutagens are well known (Pelecanos, 1964). The chemical treatments administered by adult feeding require a longer period than the irradiation treatments, as the responsible mutagenic reaction is expectedly prolonged after the period of treatment. If newly emerged adult females are treated by adult feeding during 24 hours the stages affected are the stages immediately preceeding stage 7, stage 7 itself, and stages 7-13, which have developed during such a period.

The sensitivity of stage 14 oocytes, reached at the 3rd day of adult life can be studied with more confidence, as there is no further development of oogenesis after the treatment.

The procedure adopted for the present study was to collect newly-emerged virgin females which were aged afterwards during 4 days and mating each of them with two males in vials containing regular agar-cornmeal food-medium, making further collection of less than 24 early eggs. The post-treated group includes the isolation of newly-emerged virgin females in regular medium, aging them during 4 days in food with cyclohexylamine, and further mating and oviposition in regular medium (Table 1). In the pre and post-treated group the embryonic, larval and first 4 days of adult life prior to mating took place in a medium with cyclohexylamine (Table 2). Afterwards, the flies were transferred to the regular agar-cornmeal medium employed during all the experiment, and mated in order to collect samples of stage-14

oocytes.

Cyclohexylamine (Merck) was added to the food medium and homogenized with a stirrer (Félix, 1970) at the temperature of 40 ± 2° C. The examination of the progenies was done 13 days after oviposition.

In order to test the toxicity of cyclohexylamine, several concentrations were tested, feeding all the stages of development. The concentration of 8.60 mg/ml killed adults before 2 hours, while feeding with concentrations from 4.30 mg/ml to 6.88 mg/ml gave adult survival during 24 hours without development of the eggs layed during such a period. Cyclohexylamine at concentrations of 0.86 mg/ml did not noticeably affect the life-cycle of adult and larvae.

Table 1. Progenies obtained from adult feeding of females with cyclohexylamine.

| cyc mg/ml | Control | 0.08 | 0.86 |
|---|---|---|---|
| ♂ (m.p.c.) | 10.40 | 11.17 | 10.54 |
| ♀ (m.p.c.) | 6.40 | 7.00 | 7.42 |
| ♂/♀ (s.r.) | 1.63 | 1.60 | 1.42 |
| ♂ (total | 260 | 201 | 274 |
| ♀ (total) | 160 | 226 | 193 |
| (n.c.) | 25 | 18 | 26 |

cyc mg/ml, cyclohexylamine, milligram/milliter; (m.p.c.) mean per culture; (s.r.), sex ratio; (n.c.), number of cultures.

An improved method for detecting non-disjunction and chromosome X loss was applied, that gives particularly reliable evidence concerning the origin of the exceptional recovered progeny. Females were taken from one stock with $sc^8Y$ chromosome, to avoid the existence of any secondary exceptions from XXY mothers ($y^2$ $w^a/sc^8Y$; e/e). The tester stock which provides males has an attached $Y^SX{\cdot}Y^L$ chromosome with the markers yellow and Bar (In(1)EN, $Y^S$ B $y{\cdot}Y^L$/ $\frac{y^2}{su\text{-}w^a\ bb}$/0). When virgin females are isolated, the marker $sc^8$ in the Y chromosome, which contains the normal allele of y identifies XXY females which show gray phenotype instead of the yellow color showed by normal XX females. The marker ebony insures the virginity of such females, as the genotype of the females from the cross $y^2$ $w^a/y^2$ $w^a$;e/e x In(1)EN, $Y^S$ B $y{\cdot}Y^L$; +/+, is heterozygous for the ebony marker.

Table 2. Progenies obtained from larval and adult feeding of females with cyclohexylamine.

| cyc mg/ml | Control | 0.08 | 0.86 |
|---|---|---|---|
| ♂ (m.p.c.) | 10.40 | 7.57 | 7.81 |
| ♀ (m.p.c.) | 6.40 | 7.57 | 6.81 |
| ♂/♀ (s.r.) | 1.63 | 1.33 | 1.15 |
| ♂ (total) | 260 | 174 | 125 |
| ♀ (total) | 160 | 131 | 109 |
| (n.c.) | 25 | 23 | 16 |

cyc mg/ml, cyclohexylamine, milligram/milliter; (m.p.c.), mean per culture; (s.r.), sex ratio; (n.c.), number of cultures.

The fertilization of an XX egg with an XY spermatozoon would produce meta-females with low viability which were excluded from the following analysis, whereas the fertilization of eggs of the same non-disjunctional chromosomal constitution with a non X or Y chromosome bearing spermatozoon, would produce matroclinous yellow, white apricot females of the same genotype as their mothers. These females are easily identified from their normal Bar eyed sisters. The no X egg when fertilized with an XY bearing sperm would become an XY patroclinous male, which can be identified by the Bar eyes in its phenotype.

Virgin females of the genotype $y^2$ $w^a/y^2$ $w^a$; e/e were aged during 4 days. In each vial an aged female was mated with 2 attached $Y^S$ X B $y{\cdot}Y^L$; +/+ males, and eliminated after the collection of stage 14 oocytes. All the cultures were kept at 25 ± 1° C throughout the experiment.

P $y^2$ $w^a/y^2$ $w^a$;e/e ♀♀ x In(1)EN,$Y^S$ By·$Y^L$;+/+ ♂♂

$F_1$ regular: $y^2$ $w^a$/In(1)EN,$Y^S$ B $y{\cdot}Y^L$;e/+($\frac{B\ y}{y^2}$) ♀♀

$y^2$ $w^a$;e/+ ($\frac{y^2}{}$ $\frac{w^a}{}$) ♂♂

exceptional: $y^2\ w^a/y^2\ w^a;e/+$ $(\underline{y^2\ w^a})$ ♀♀

$In(1)EN,Y^S\ B\ y\cdot Y^L;e/+$ $(\underline{B\ y})$ ♂♂

Data on the progenies obtained in the treated and control groups are included in Tables 1 and 2. No exceptional progenies were found either among 894 treated flies, from the adult feeding group, nor among 539 treated flies from the larval and adult feeding group.

Among 1,433 stage-14 oocytes treated with the maximum concentrations permissible for the female adult, no exceptions from non-disjunction and X-chromosome loss were recovered. However, the equivalence between the effects of the dosages employed in this experiment, and those from previous findings named above, is evidently difficult. Such factors as absorption, protein binding and excretion are to be considered to make pertinent comparisons with mammalian or human intake cyclamates and its transformation to cyclohexylamine.

It seems from this experiment that Drosophila shows a low sensitivity to the ingestion of cyclohexylamine when the genetic events mentioned before are recorded.

References: Brodie, B.B., 1965. Toxicology and the Biomedical Sciences, Science 148: 1547-1554; Derse, D.H. and R.J. Daun, 1966. J. Asoc. Offic. Agr. Chemist 49: 1090; Felix, R., 1970. High speed stirrer and mixer for Drosophila food medium. DIS 45:178. Food Additives and Contaminants Committee Second report on Cyclamates, 1967. Min. of Agric., Fisheries and Food; Kojima, S. and H. Ichibagase, 1966. Chem. Pharm. Bull. Japan 14: 971. King, R.C., J.B. Darrow and N.W. Kay, 1956. Oogenesis in adult D. melanogaster. Growth 20: 121-157; Leahy, J.S., M. Wakefield, T. Taylor, 1967. Food Cosmet. Toxicol 5: 447; Legator, M., 1968. In: Medical World News 15: 25; Legator, S., K.A. Palmer, S. Green, K.W. Petersen, 1969. Science 165: 1139; Majumdar, S.K. and D.J. Lane, 1970. Effects of Sodium Cyclamate on Haworthia Callus Cultured in Vitro. Journ. Heredity 61(5): 193-195; Oser, L.B., S. Carson, E.E. Vogin, 1968. Conversion of Cyclamate to Cyclohexylamine in Rats. Nature 220: 178-179; Pelecanos, M. and T. Alderson, 1964. The Mutagenic Activity of Diethyl Sulphate in D. melanogaster. III. The Sensitivity of the Immature (Larval) and Adult Ovary. Mutation Res. I: 302-309; Price, J.M., C.G. Biava, B.L. Oser, E.E. Vogin, J. Steinfeld, H.L. Ley, 1970. Bladder tumors in Rats Fed Cyclohexylamine or High Doses of a Mixture of Cyclamate and Saccharin. Science 167: 1131-1132; Stone, D., E.Lamson, Y.S. Chang, K. Pickering, 1968. In: Ann. Meeting Tissue Culture Ass. Puerto Rico (Abstr.): 60; Stone, D., E. Lamson, Y.S. Chang, K.W. Pickering, 1969. Science 164: 568.

Sreerama Reddy, G. and N.B. Krishnamurthy. University of Mysore, Manasagangotri, Mysore-6, India. Preliminary survey of Drosophilids in Nilgiris and Kodaikanal ranges.

Maiden trips were made to Nilgiris and Kodaikanal ranges to explore the Drosophilids. Nilgiris is about 100 miles to the south of Mysore and Kodaikanal is about 300 miles also to the south of Mysore. Both these localities are hill stations characterised by the combination of temperate and tropical forests. The sholas are evergreen with moist and humid atmosphere in most part of the year. Shifting cultivation forms the common biotic factor in both the localities. The annual rain fall ranges from 1524 to 2540 mm. The highest peak of Nilgiris is 2580 meters and that of Kodaikanal is 1900 meters.

Collections were made in the beginning of December 1970. In the Nilgiris, flies were trapped at 11 different altitudes ranging from 840 to 2580 meters, whereas in Kodaikanal the flies were trapped at six altitudes ranging from 1000 to 1900 meters. A total of 1470 flies were collected from both the localities, of which 1009 flies come from Nilgiris and 461 from Kodaikanal. The details of the collection record are depicted in tables 1 and 2. The various species collected from both the localities are D. melanogaster, D. ananassae, D. melarkotliana, D. takahashi, and D. immigrans. The species like D. repleta, D. kikkawai, D. mysorensis, D. hoozani like species and D. busckii are found in Nilgiris and absent in Kodaikanal range. In addition, two new species of Drosophila which will be described elsewhere were also found in the traps. In Kodaikanal range one individual belonging to the Genus Leucophenga was found in an orchard near Valegiri at an altitude of 1000 meters. It is quite remarkable to note that D. immigrans is wide spread in its distribution in almost all places scanned. This shows that D. immigrans thrives well in a moist and humid climate. Further the most interesting feature of our collection study is the lack of Drosophila flies

in the traps except one individual male of Drosophila hoozani like fly at a high altitude of 2580 meters in Doddabetta (Nilgiris).

Table 1. Distribution of different species of Drosophilids in Nilgiri Range

| | Wild Domestic | Altitude in Metres | D. melanogaster | D. ananassae | D. melarkotliana | D. repleta | D. takahashi | D. kikkawai | D. mysorensis | D. immigrans | D. busckii | D. species like hoozani | Prob. new species (near truncata)A3 | Prob. new species A2 | Total |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Thippakadu | Wild | 840 | - | - | 5 | - | 35 | - | - | - | - | - | - | - | 40 |
| Gudalur | Domestic | 900 | 42 | 331 | - | 32 | - | - | - | - | - | - | - | - | 405 |
| Gudalur | Wild | 990 | - | - | 14 | - | 3 | 5 | 2 | 20 | - | - | - | - | 44 |
| Merupalyam | Wild | 1200 | - | 25 | - | - | 8 | - | - | 45 | - | - | 8 | - | 86 |
| A virgin forest on the Ooty Road | Wild | 1310 | - | - | - | - | - | - | 31 | 31 | - | - | - | - | 62 |
| Coonur | Domestic | 1800 | 25 | - | - | - | 5 | 9 | - | 15 | - | - | - | 10 | 64 |
| Naduvattam | Domestic | 1840 | 3 | 2 | 2 | 10 | 2 | - | - | 65 | 3 | - | - | - | 87 |
| Naduvattam | Wild | 1850 | - | - | - | - | - | - | 10 | 10 | - | - | - | - | 20 |
| T.R. Bazar | Domestic | 1970 | 5 | - | - | 90 | - | - | - | 10 | - | - | - | - | 105 |
| Ooty city | Domestic | 00 | 6 | - | - | - | - | 33 | - | 56 | - | - | - | - | 95 |
| Doddabetta | Wild | 580 | - | - | - | - | - | - | - | - | - | 1 | - | - | 01 |
| | | | 81 | 358 | 21 | 132 | 53 | 47 | 43 | 252 | 3 | 1 | 8 | 10 | 1009 |

Table 2. Distribution of Different Species of Drosophilids in Kodaikanal Range

| | Wild or Domestic | Altitude in Metres | D. melanogaster | D. ananassae | D. melarkotliana | D. takahashi | D. immigrans | Leucophenga (Trichiaspiphenga) invicta (Walker) | Total |
|---|---|---|---|---|---|---|---|---|---|
| Valegeri | Orchard | 1000 | - | - | 4 | 2 | 65 | 1 | 72 |
| Oothu | Domestic | 1100 | - | 165 | 9 | - | 0 | - | 194 |
| Oothu | Wild | 1400 | - | - | 3 | 2 | 40 | - | 45 |
| Perumalmalai | Domestic | 1580 | 38 | 30 | - | - | 10 | - | 78 |
| Kodaikanal | Wild | 1750 | - | - | 9 | 5 | 18 | - | 32 |
| Kodaikanal Town | Domestic | 1900 | - | - | - | - | 40 | - | 40 |
| | | | 38 | 195 | 25 | 9 | 193 | 1 | 461 |

Acknowledgements: We are highly grateful to Dr. M.R. Rajasekarasetty, Professor and Head of the Department of Zoology, Manasagangotri, University of Mysore, Mysore for his help and encouragement. We are indebted to Dr. T. Okada, Professor of Biology, Tokyo Metropolitan University, Department of Biology, Setagaya-Ku, Tokyo, Japan for identifying the species. One of us (G. Sreerama Reddy) is thankful to the University of Mysore and University grants commission for the financial support.

Ringo, J.M. University of California, Davis, California. The effects of anesthetization upon survival and behavior of D. grimshawi.

To study some elements of behavior of D. grimshawi Oldenberg, it is necessary to mark and mutilate individuals, which in turn requires that the flies be lightly anesthetized. In order to find an anesthetization procedure which interferes the least with subsequent behavior, the effects of three agents ($CO_2$, cold, and ether) were evaluated.

To assess the effects of treatment upon survival, one control group (n=30) was maintained and each agent was applied to five other groups of flies (N=20 in each group) for different lengths of time (1/2, 1, 2, 4, and 8 mins.) Flies were chosen at random from a population of PK9 D. grimshawi adults, aged 1 to 22 days. $CO_2$ was administered by suspending flies in a plastic tube over dry ice in a one lb. coffee can. The tube was fitted through a hole in a cardboard top, and the bottom of the tube was covered by a piece of bolting silk. This apparatus was modified from Seecof (1963). The temperature at the bottom of the tube was approximately 4°C. Anesthetization with cold was attained by placing flies in an aluminum cigar tube immersed in ice; the temperature was about 0°C. Ether was used in an ordianry small plastic etherizer. About two ml of ether was placed on the gauze at the bottom of the etherizer, and a few drops were added between treatment groups. The temperature was approximately 20°C. After treatment, each group was placed in a half pint bottle containing fresh food and maintained at 20°C±1°. Dead flies were removed and counted at 24, 48, 72, and 96 hours after treatment. The results are summarized in the following table and graphs:

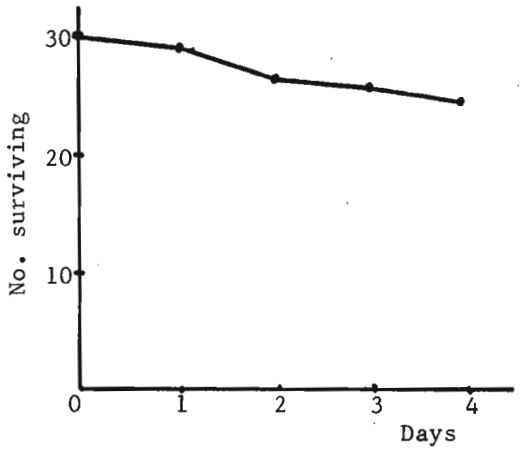



Figure 1.

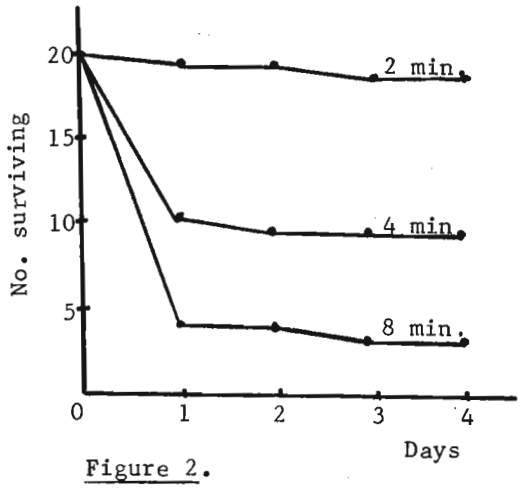



Figure 2.

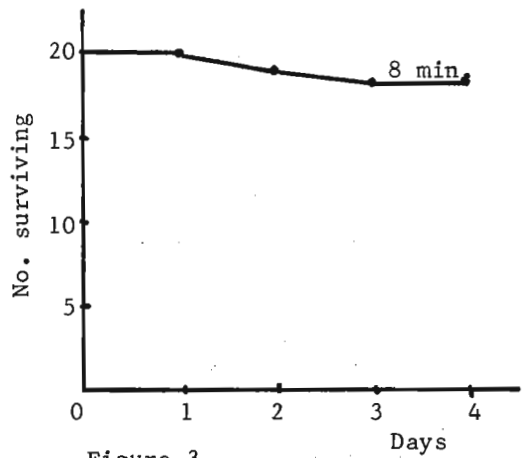



Figure 3.

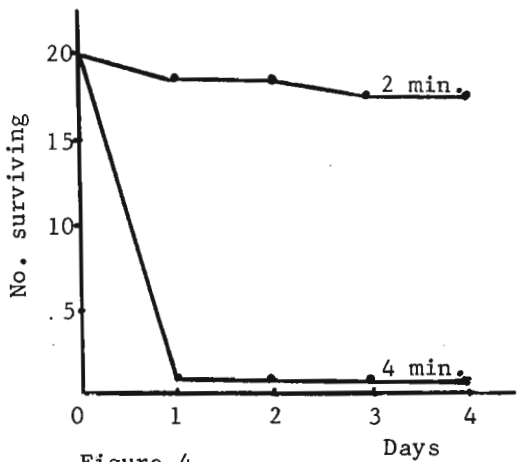



Figure 4.

Survival curves for six treated groups and control group. Fig. 1, controls; fig. 2, $CO_2$ treatment for 2, 4, and 8 mins.; fig. 3, cold treatment for 8 min.; fig. 4, ether treatment for 2 and 4 mins.

Table 1

| | Min. with $CO_2$ | | | | | Min. with cold | | | | | Min. with ether | | | | | Controls |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| No. surviving | 1/2 | 1 | 2 | 4 | 8 | 1/2 | 1 | 2 | 4 | 8 | 1/2 | 1 | 2 | 4 | 8 | -- |
| after 24 hours | 19 | 19 | 19 | 10 | 4 | 20 | 18 | 19 | 20 | 20 | 19 | 20 | 18 | 1 | 0 | 29 |
| " 48 " | 19 | 19 | 19 | 9 | 4 | 19 | 14 | 19 | 18 | 19 | 18 | 19 | 18 | 1 | 0 | 26 |
| " 72 " | 18 | 18 | 18 | 9 | 3 | 17 | 14 | 18 | 18 | 18 | 18 | 18 | 17 | 1 | 0 | 25 |
| " 96 " | 18 | 18 | 18 | 9 | 3 | 17 | 14 | 18 | 18 | 18 | 18 | 18 | 17 | 1 | 0 | 24 |

We accept the hypothesis that the proportion of survivors among the controls and all flies treated for 30 sec. were equal ($\chi^2$=1.43, df=2, p>.20)

A second experiment sought to determine differences in behavior attributable to these three methods of anesthetization. The phenotype of greatest interest is jousting, a type of behavior found only in males of this species. Subjects were drawn at random from a population of adult PK9 males aged 19 to 25 days. N=30 for each treatment group. Ss were anesthetized for 30 sec., their wings were marked with nail polish containing non-toxic dyes; they were isolated in individual half pint bottles containing fresh food and were maintained at 20°C±1°. Allowing at least two hours for recovery, Ss were observed in batches (N=10) in plexiglass cells (2x5x9 cm) with moist sponge at one end. Their interactions were observed for 20 minutes and recorded; the exact time spent jousting was recorded for each subject using an Esterline Angus 10-channel event recorder. The observations were repeated four more times for each S.

There were marked behavioral differences between treatments. Aggression and courting were very much reduced in cold-treated Ss, and somewhat reduced in $CO_2$-treated Ss relative to etherized Ss. The quantitative results for jousting show a similar pattern:

Table 2

| Treatment | Total of all scores | No. of Ss |
|---|---|---|
| $CO_2$ | 834.3 | 28 |
| cold | 932.9 | 25 |
| ether | 1842.1 | 30 |

The data can be analyzed in two ways. One can simply record whether or not a subject jousted during a given observation period, or one can consider the relative amount of jousting for each test period. An ordinary analysis of variance is impossible, since the scores have a J-shaped distribution. Out of 415 observations (7 Ss died) or scores, 271 were zero. Using 271/415 = .653 as the expected proportion of zero scores among treatments and testing $H_o$:$\theta_1=\theta_2=\theta_3$ against the alternative that the proportions are not equal, we reject $H_o$ ($\chi^2$=10.37, df=2, and p<.01). The large number of zero scores in all groups of Ss indicates that a simple dichotomous measure has as much biological significance as the amount of time spent jousting. The simplest non-parametric test using the scores is the Friedman two-way analysis of variance by ranks (Siegel 1956). The Friedman test requires equal sample sizes, but 7 Ss died during the experiment and could not be replaced so we averaged the scores for each batch. We reject the hypothesis that treatments do not differ in their effects ($\chi_r^2$=6.50, df=2, p<.05).

The results of these experiments show that light etherization is a better method of anesthetization for behavioral studies in D. grimshawi than the use of either $CO_2$ or low temperature.

References: Seecof, R.L. 1963 DIS 37:145; Siegel, S. 1956 Nonparametric Statistics for the Behavioral Sciences, McGraw-Hill, Inc., New York.

Hunt, D.M. University College London, England. A haemolymph protein anomaly associated with the lethal-giant-larvae mutant in Drosophila melanogaster.

Faulhaber (1959) demonstrated a reduction in the haemolymph protein content of larvae homozygous for the lgl mutant. However, the paper electrophoresis technique employed by Faulhaber allowed the clear separation of only two protein fractions. With the introduction of acrylamide gel as a supporting medium for electrophoresis, it is now possible to

identify a large number of protein fractions in larval haemolymph. A re-examination of the situation in lgl therefore would seem appropriate.

Two alleles lgl and $lgl^B$ have been studied. Both mutants were maintained as balanced lethals over the SM5 chromosome. Haemolymph samples from non-lethal larvae were collected at about 5 days post-hatching when the larvae leave the food medium prior to pupation. Development in lethal larvae is delayed and haemolymph samples were taken therefore from at least 6 days post-hatching. The technique of acrylamide gel disc electrophoresis was used. The procedure follows Davis (1964) except that the spacer and sample gels were omitted and 50µl of sample applied directly to the top of each gel. Gels were stained in 0.5% amido black in 7% acetic acid.

No differences could be detected in the haemolymph proteins from lgl/lgl and lgl/SM5 third instar larvae, and $lgl^B$/SM5 larvae also gave a normal protein pattern. However, the electrophoretic separation of haemolymph samples from $lgl^B/lgl^B$ larvae revealed clear differences in protein content; fraction 7 was entirely missing and the amount of stainable material in fractions 12 and 15 considerably elevated (Fig. 1).

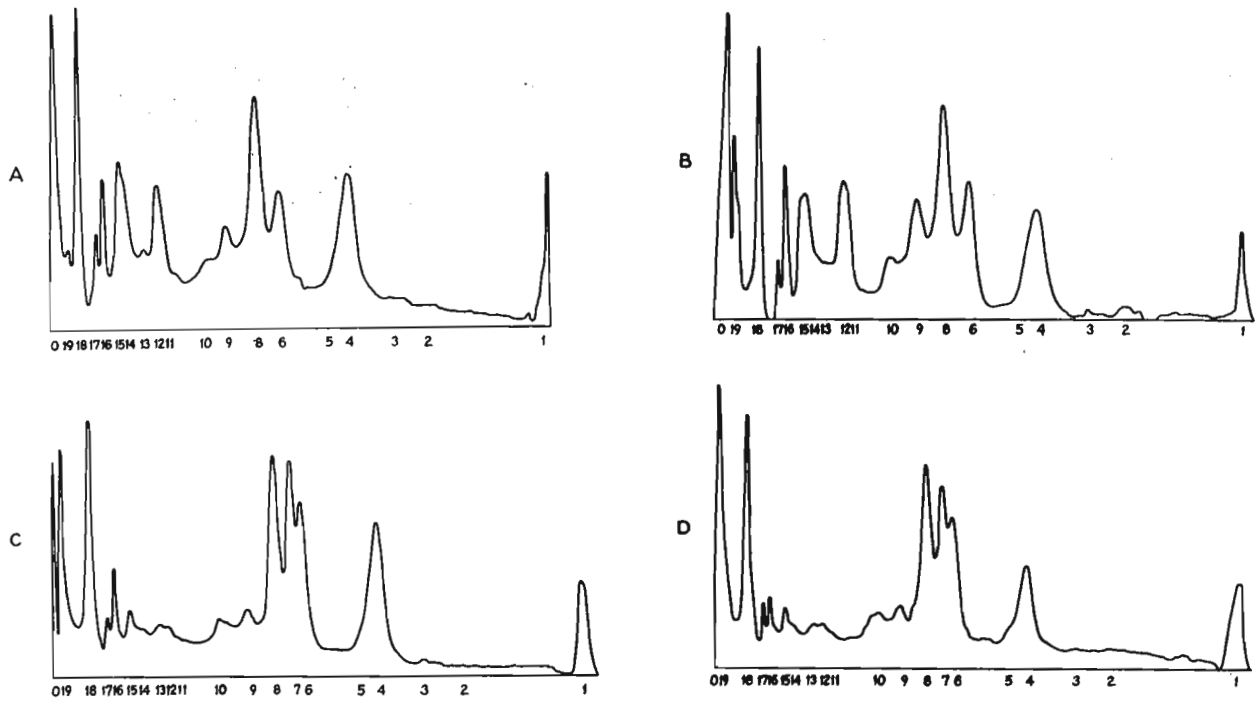



FIGURE 1. Densitometer tracings of electropherograms obtained from haemolymph samples of third instar $lgl^B/lgl^B$ larvae (A,B) and $lgl^B$/SM5 larvae (C,D). The protein fractions are numbered from the running front to the origin (0).

Fractions 12 and 15 show considerable quantitative variation between strains so the inheritance of the elevated quantities in $lgl^B$ homozygotes was not examined further. The possibility that the absence of fraction 7 in lethal larvae depends on another gene locus on the $lgl^B$ second chromosome was tested by outcrossing the $lgl^B$/SM5 strain to the Edinburgh wild type. Non-SM5 $F_1$ progeny was mated to expose the $lgl^B$ chromosome to recombination and haemolymph samples were taken from the resulting third instar larvae. In a total of 104 $lgl^B/lgl^B$ larvae examined, no recombinants were obtained.

References: Davis, B.J., 1964, Ann. N.Y. Acad. Sci. 121: 404; Faulhaber, I., 1959, Z. Induktive Abstammungs-Vererbungslehre 90: 299.

Nirmala Sajjan, S., and N.B. Krishnamurthy,
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Karyotype of Drosophila nasuta.

D. nasuta subgroup of the immigrans group created by Wilson et al (1969) includes 8 morphologically similar species. Males of this subgroup have silvery markings on the frons, in all but one species. However, whitish to silvery sheen over the entire frons is present only in D. albomicans, D. kohkoa, D. kepulauana and D. nasuta Lamb (1914). The species described here is characterized by silvery sheen over the entire frons but differs cytologically from that of D. albomicans, D. kohkoa and D. kepulauana. Though Ray Chaudhuri and Jha (1969) have given an account on the cytology of D. nasuta, it is not known to which species proper it belongs under nasuta subgroup. The karyotype of D. nasuta sensu strictu is yet unknown. Hence the karyotype is reported here.

The flies collected from Soundatti (Mysore state) are big and yellowish in color with silvery frons. There is brown longitudinal streak on pleura reaching back to the wing base in both males and females. Other morphological characters are similar with that of D. nasuta reported by Okada (1964). The metaphase karyotype of the larval neuroblast cells (Fig. 1) consists of a pair of rods which represents X chromosome in females, one of which is replaced by V-shaped Y chromosome in males, a pair of V's (chromosome 2), a pair of double length rods (chromosome 3) and a pair of dots (chromosome 4). No additional heterochromatin is found in the dot.

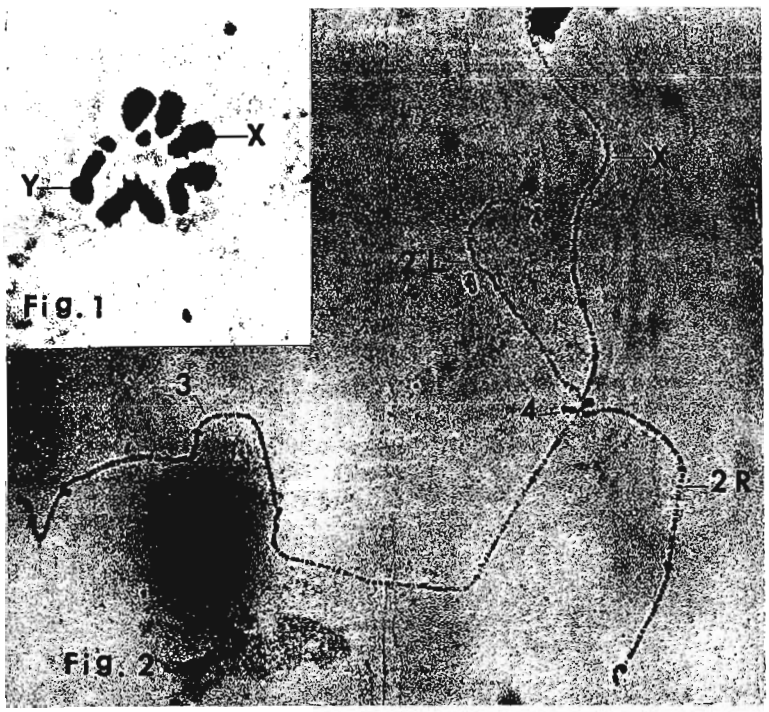



Fig. 1. The Metaphase Karyotype of male larval neuroblast cell.

Fig. 2. Salivary gland chromosomes

The salivary gland chromosomes show four long arms and one small arm as shown in the fig. 2. Centric heterochromatin is practically absent except for a little between 2L and 2R. Like other species of the subgroup, here also a loop, which is not an inversion is frequently observed in the basal region of 2R. The longest arm represents the double length rod of the metaphase karyotype and the arm next to third chromosome in length is the X chromosome and the remaining two long arms are the left and right arms of the metacentric second chromosome. The small arm represents the dot chromosome of the somatic metaphase.

The species under study is allied to D. albomicans, D. kepulauana and D. kohkoa in having entire frons with silvery sheen. This is also true with the original species, D. nasuta according to published notes for which cytology is not known. Based on the cytological analysis, the species here described differs from D. albomicans in that D. albomicans

has 2n=6, whereas here we have 2n=8. It also departs from D. kepulauana in having V-shaped Y chromosome and basic type of dots while in D. kepulauana Y is rod shaped and the dot chromosomes with added heterochromatin are slightly thicker and longer. The other member of the same series with entire silvery frons -- D. kohkoa, is characterized by the pinched constriction in the third chromosome which is always accompanied by the dot. This species also has a small amount of added heterochromatin to the dot which gives it a comma-shaped appearence (Wilson et al, 1969). This has not been observed in the present species. The karyotype described by Ray Chaudhuri and Jha (1969) consists of 6 pairs of chromosomes in metaphase configuration and 6 arms (5 long and one short arm) in salivary gland nuclei. Our findings are different from this.

Recounting the similarities and differences that are exhibited by the members of the nasuta subgroup, the species herein described must be either D. nasuta sensu strictu or a new species of the nasuta subgroup for which confirmation is needed. Further this species is highly polymorphic in having duplications and deficiencies and a multitude of inversions which will be presented elsewhere.

Acknowledgements: The authors are very grateful to Dr. M.R. Rajasekarasetty, Professor and Head of the Department of Zoology, University of Mysore, Manasagangotri, Mysore for his advice and encouragement. We are thankful to Mr. Ramakrishna Raju for preparing Photomicrographs. This work is supported by the department of Atomic Energy, Government of India.

References: Ray Chaudhuri, S.P. and A.P. Jha. 1969, The Nucleus, Vol. XII(1): 9-13; Wilson, F.D., M.R. Wheeler, Margaret Harget and Michael Kambysellis. 1969, Cytogenetic relations in the D. nasuta subgroup of the immigrans group of species.

Sanjeeva Rao, M. and S. U. Devi. Osmania University, Hyderabad-7, AP., India. Induction of mutations in D. melanogaster with radioisotypes - $^{90}Sr$ and $^{131}I$.

Even though much work was done on the induction of mutations in Drosophila by ionizing radiations and chemicals, the possible mutagenic effects of radioisotopes have received little attention. Blumel (1950) reported that phosphorus-32 induces mutations in Drosophila while Rubin (1950) observed mutagenicity in microorganisms. $Sr^{90}$ and $I^{131}$ are more powerful radioisotopes than phosphorus-32 and to assess their genetic damage in Drosophila the following experiments were carried out.

Two concentrations of each isotope were tried. The isotope was mixed in food medium. Flies were allowed to lay eggs on this medium and the offspring were allowed to grow on the medium containing the isotope. The treated males were crossed individually with 3 virgin females of y $sc^{S1}$ In-49 $sc^{8}$;bw;st for three days only to assess the genetic damage in spermatozoa alone. The $F_1$ females were mated individually with y $sc^{S1}$ In-49 $sc^{8}$ males while the males were mated with bw;st females to score for sex linked recessive lethals and translocations, respective in the $F_2$ generation. The results are presented in Table 1.

Table 1

| Treatment | Sex linked recessive lethals | | | | Translocations | | | |
|---|---|---|---|---|---|---|---|---|
| | T | l | % | Chi-square value | T | l | % | Chi-square value |
| 1. Control | 505 | 1 | 0.2 | - | 712 | - | - | - |
| 2. $Sr^{90}$ 0.2µcc in 100cc of food | 329 | 8 | 2.12 | 9.3 | 439 | 3 | 0.68 | 4.94 |
| 3. $Sr^{90}$ 1.0µcc in 100cc of food | 268 | 5 | 1.86 | 6.33 | 247 | 3 | 1.21 | 8.74 |
| 4. $I^{131}$ 1.00µcc in 100cc of food | 436 | 8 | 1.83 | 6.64 | - | - | - | - |
| 5. $I^{131}$ 2.00µcc in 100cc of food | 363 | 5 | 1.40 | 4.28 | 347 | 2 | 0.6 | 4.2 |

T = Total number of X chromosomes or $F_1$ sons scored; l = Lethals recorded; t = translocations recorded

These preliminary studies indicate that $^{90}Sr$ and $^{131}I$ cause mutations in D. melanogaster similar to phosphorus - 32.

Kastritsis, C.D.[1] and J. Grossfield[2]. [1]University of Texas Southwestern Medical School, Dallas, Texas. [2]Purdue University, Lafayette, Indiana. Balbiani rings in D. auraria.

Since different strains of D. auraria differ with respect to their ability to mate in darkness, and since this trait is at least under partial genetic control (Grossfield, 1970), an investigation was undertaken to explore possible cytological correlations.

Different strains were found to differ by a number of inversions and, in addition, two strains were found to exhibit two Balbiani rings (Fig. 1) in one of the chromosomes of the

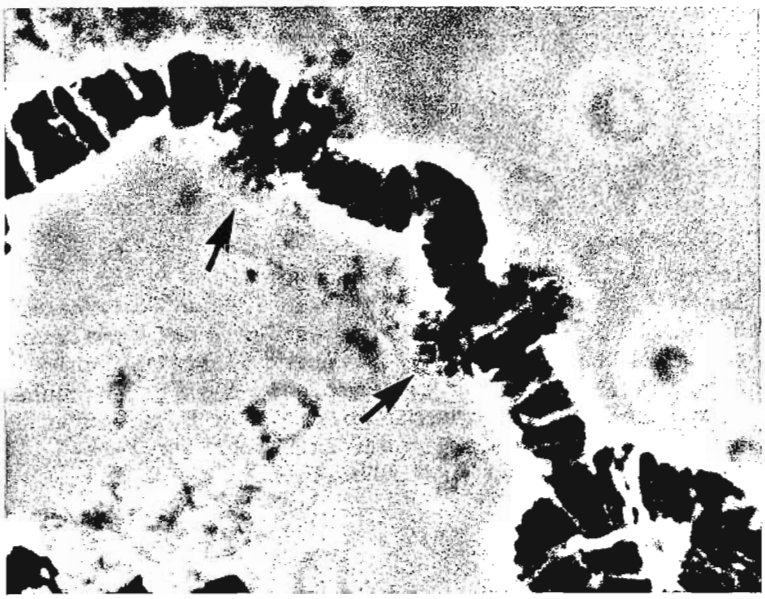


Fig. 1. Phase contrast photomicrograph of D. auraria polytene chromosome. Arrows point at two Balbiani rings.

salivary glands cells. Due to the fact that Balbiani rings have not been described in Drosophila before, we feel that our observation warrants this note. Our preliminary data indicate that these may be stage-specific structures. Research is now under way to further investigate the implications of the phenomenon.

Reference: Grossfield, J., 1970. Genetics 65:s27.

Würgler, F.E., R. Büchi and P. Maier. Swiss Federal Institute of Technology, Zürich, Switzerland. Relative viability of different types of Drosophila melanogaster males without a free Y chromosome.

If R(1)2,y B / $B^S$ Y $y^+$ males are X-rayed and mated to nonirradiated females partial as well as complete loss of sex chromosomes is indicated by non-Bar males. The reports of Graf and Würgler (DIS-46, 73-74, 1971) and Würgler and Kälin (DIS-46, 79-80, 1971) show that the rates of chromosome losses recorded depend on the type of females used in the test crosses. The data obtained for X-irradiation of ring-X males with 2000 R in nitrogen are summarized in the following table ("Oster" = Inscy;dp bw;st $p^p$ and "XY" = $y^2$ su($w^a$)$w^a$ KS.KL $y^+$ (Parker 110-8)):

| females | spontaneous rate of loss | X-ray experiment | corrected for spontaneous loss | relative rate |
|---|---|---|---|---|
| Oster | 0.54 % | 2.33 % | 1.8 % | 1 |
| y sn | 0.71 | 4.37 | 3.7 | 2.1 |
| XY | 2.2 | 8.7 | 6.7 | 3.7 |

In the three tests males of different genotype are indicative for a sex chromosome loss. The difference found between stocks might therefore simply reflect the relative viability of the non-Bar males. To test this possibility the relative viability of y sn /0 and XY/0 males compared to Oster/0 males was determined. For this purpose hybrid females y sn /Oster and XY/Oster were mated to XY/0 males and the progeny classified according to the genotype. This is a more rigorous test than the one described by Graf and Würgler because the different types of males to be compared develop under identical conditions within the same vial. The data obtained are given in the following table. For comparison, results from some other comparable test crosses are also included: (All hybrid females are heterozygous for dp bw st $p^p$.)

| females ($X^*$/Oster) | total progeny | X*/0 males | Oster/0 males | male ratio |
|---|---|---|---|---|
| y sn /Oster | 6117 | 1945 | 1627 | 1.20 |
| XY / Oster | 5871 | 1790 | 1374 | 1.30 |
| +/Oster ; t/vg | 6561 | 2194 | 1522 | 1.44 |
| Oregon-R/Oster | 5175 | 1680 | 1136 | 1.47 |
| Hikone-R/Oster | 10043 | 3090 | 1836 | 1.68 |
| Berlin wild/Oster | 3370 | 1081 | 628 | 1.72 |

For the y sn stock we find a relative viability of the y sn/0 males compared to Oster /0 males of 1.2. The corresponding ratio for the induced chromosome losses in the X-ray experiments is 2.1. A similar result is found for the XY stock : 1.30 versus 3.7. This analysis shows that, although the various types of males show a slightly different viability, this difference is not sufficient to explain the variation of the chromosome loss rates encountered in the X-ray experiments. It is postulated that after insemination of the nonirradiated egg some factors which are under the control of the maternal genome influence the X-ray lesions induced in mature sperms. Work supported by Schweizerischer Nationalfonds zur Förderung der wissenschaftlichen Forschung.

Ondřej M. Prague, Czechoslovakia. The induction of large chromosomal fragments by ethylnitrosourea and radiation.

Ethylnitrosourea is one of the most effective mutagens in producing recessive lethals. It is of interest to know the relative effectiveness of ethylnitrosourea (ENH) in producing large chromosomal aberrations. The relationship of large chromosomal fragments to lethals in the X chromosome after ENH treatment was compared with that after X-radiation. ENH was applied by injections; each fly received, on the average, 0.2 µl of solution. X-radiation was applied by means of Siemens apparatus with those parameters: 22 mA, 200 kV, OK - 17.5, filter 0.5 mm $C_u$, dose - rate 394.7 R/min., overall dose 1500 r. The concentration of ENH was near the upper limit of applicable concentrations. The radiation dose was of medium magnitude, as regards the induction of sterility. The results show, that while ENH induces extremely high frequency of recessive lethals it is very weak chromosome breaker. The ratio of the frequencies of large fragments to recessive lethals after ENH treatment was in our experimental conditions of about two orders of magnitude lower than that after irradiation.

The frequencies of large fragments and recessive lethals in the X chromosome induced by ENH and x-rays.

| Treatment | Fragments | | Rec. lethals | | Fragments/ lethals |
|---|---|---|---|---|---|
| | No. of $F_1$ females | % of fragments | No. of X chromosomes | % of rec. lethals | |
| ENH 10 mM | 4,770 | 0.02 | 550 | 41.5 | 0.0005 |
| X-rays 1500 R | 18,577 | 0.27 | 715 | 5.6 | 0.048 |
| Control | 19,570 | 0.005 | | | |

Jacob, M. and S.P. Ray-Chaudhuri, Banaras Hindu University, Varanasi, India. Protective effect of glutathione (reduced) against X-ray induced sex-linked recessive lethals in D. melanogaster.

The thiol tripeptide, glutathione, was shown to be protective against radiation-induced lethality in mice (Chapman et al. 1950). This chemical yielded significant protection against chromosomal aberrations also, in Tradescantia (Mikaelsen 1952) and grasshopper (Chaudhuri 1965). But Mittler (1964) reported the failure of glutathione as well as a few other well-known chemicals to protect Drosophila chromosomes against X-ray induced sex-linked lethals, dominant lethals, translocations and deletions.

A number of experiments were conducted to test the protective effect of reduced glutathione against sex-linked recessive lethals using Oster's stock. One-day old males ($X^{c2}yB$)

Frequency of sex-linked recessive lethals after 2000 r and pretreatment with glutathione(reduced)=GSH

| Experiment No. | Treatment | No. of sex linked lethals | No. of chromosomes tested | % of lethals | $\chi^2$ | Probability | Degree of protection |
|---|---|---|---|---|---|---|---|
| 1 | GSH + 2000r | 41 | 770 | 5.3 | 4.1 | $0.05>P>0.025$ | 35.3 |
| | Saline+2000r | 50 | 604 | 8.2 | | | |
| 2 | GSH + 2000r | 48 | 923 | 5.2 | 2.6 | $0.25>P>0.10$ | 24.6 |
| | Saline+2000r | 76 | 1096 | 6.9 | | | |
| 3 | GSH + 2000r | 51 | 932 | 5.4 | 7.1 | $0.01>P>0.005$ | 36.6 |
| | Saline+2000r | 147 | 1722 | 8.5 | | | |
| 4 | GSH + 2000r | 134 | 2729 | 4.9 | 5.3 | $0.02>P>0.01$ | 28.9 |
| | Saline+2000r | 72 | 1037 | 6.9 | | | |
| Total | GSH + 2000r | 274 | 5354 | 5.1 | 24.9 | $P>0.005$ | 33.7 |
| | Saline+2000r | 345 | 4456 | 7.7 | | | |

were pretreated with the chemical dissolved in saline to a concentration of 10 mM and each fly received 0.6 µl of the solution. The controls were treated with saline and both the lots received a dose of 2000 r X-ray after 30 minutes of the treatment. Crosses were made with these males and the virgins of the Oster ♀ stock. Nearly twenty pair-matings were made from the $F_1$ progeny of each treated male and the $F_2$ offspring were examined for the absence of Bar males, indicating the presence of a lethal induced in the paternal X-chromosome.

The consistent results of the four repeated experiments are presented in the table. Significant protection was observed in all the experiments, only one being slightly below the border line. The pooled data of all the experiments show a highly significant protection, $P(\chi^2)$ 0.005, the degree of protection being 33.7.

References: Chapman, W.H., C.R. Sipe, D.C. Eltzholtz, E.P. Cronkite and F.W. Chambers 1950, Radiology 55: 865; Chaudhuri, J.P., 1965, Ph.D. Thesis, Banaras Hindu University; Mikaelsen, K., 1952, Science 116 (3007): 172-174; Mittler, S., 1964, Int. J. Rad. Biol. 6 (5): 405-413.

Lee, T.J. Chungang University, Seoul, Korea. Frequency of races in males of D. auraria in natural populations.

D. auraria is a polymorphic species in Korea populations. This species was divided into three races, A, B and C, mainly by forms of genitalia. In natural populations of Korea, the distribution area of races A and C is much wider than race B. In general, race A is the most domestic of the three, abundant around areas of human habitation, while race B inhabits rather cool, and mountainous or relatively northern regions. The habitat of race C is, in general, wider in environmental and higher altitude than that of race A. However, it is sometimes found that the two or three races live sympathetically.

Among the flies collected in twelve localities, a few flies of D. auraria showed a

hybrid character in male phallic organs. The shapes in the phallic organs found in these males seems to be the same as those found in experimental hybrids obtained in the laboratory. However, no natural AB hybrid has yet been detected.

| Races | | A | B | C | AC | BC | Total |
|---|---|---|---|---|---|---|---|
| Is. Quelpart | No. | 11 | | 58 | 2 | | 71 |
| | % | 15.4 | | 81.6 | 2.8 | | |
| Mt. Chiri | No. | 41 | | 35 | 6 | | 82 |
| | % | 50.0 | | 42.6 | 7.3 | | |
| Mt. Kaya | No. | 33 | 13 | 41 | 4 | 2 | 93 |
| | % | 35.4 | 13.9 | 44. | 4.3 | 2.2 | |
| Muju | No. | 75 | | 13 | 2 | | 90 |
| | % | 83.3 | | 14.4 | 2.2 | | |
| Mt. Palkong | No. | 15 | | 48 | 7 | | 70 |
| | % | 21.4 | | 68.5 | 10.0 | | |
| Mt. Kyeryong | No. | 58 | | 25 | 5 | | 88 |
| | % | 65.9 | | 28.4 | 5.6 | | |
| Kongju | No. | 75 | | 7 | 3 | | 85 |
| | % | 88.2 | | 8.2 | 3.5 | | |
| Mt. Sokli | No. | 75 | 20 | 26 | 3 | 2 | 126 |
| | % | 59.5 | 15.9 | 20.6 | 2.3 | 1.6 | |
| Daekwanryong | No. | 19 | 30 | 53 | 4 | 2 | 108 |
| | % | 17.5 | 27.7 | 49.0 | 3.7 | 1.8 | |
| Kwangneung | No. | 40 | 32 | 22 | 3 | 1 | 98 |
| | % | 40.8 | 32.6 | 22.2 | 3.1 | 1.0 | |
| Mt. Soyo | No. | 17 | | 22 | 7 | | 46 |
| | % | 36.9 | | 47.8 | 15. | | |
| Mt. Sulak | No. | 20 | 39 | 25 | 2 | 2 | 88 |
| | % | 22.7 | 44.3 c | 28.4 | 2.7 | 2.7 | |

The sexual isolating mechanisms among the three races were analyzed. The mean coefficient of interracial sexual isolation in the three classes are arranged in descending order, A ⟷ B (0.845) > B ⟷ C (0.600) > A ⟷ C (0.426) at 25°C, and A ⟷ B (0.788) > B ⟷ C (0.417) > A ⟷ C (0.165) at 19°C.

In crosses between A and B, a highly significant sexual isolation was demonstrated at both temperatures of 19°C and 25°C.

It is conjectured that races A and B, in natural populations, have been completely precluded genetically from one another by many isolating mechanisms.

References: Kurokawa, H. 1960. Japan J. Genet., 35:161-166; Kurokawa, H. 1967. Annot. Zool. Japan., 40:154-160; Lee, T.J. 1970. Chungang Univ. Theses Collection, 15: 239-258.

## PERSONAL AND LABORATORY NEWS

D. Sperlich is now at Tübingen, Germany, from Vienna, Austria. New address: Institut für biologie, Lehrstuhl für Genetik, Aus der Morgenstelle 1, D-7400 Tübingen, Germany.

After September 1, 1971 the address of Th. Dobzhansky, F.J. Ayala and Mrs. Olga Pavlovsky will be: Department of Genetics, University of California, Davis, California 95616.

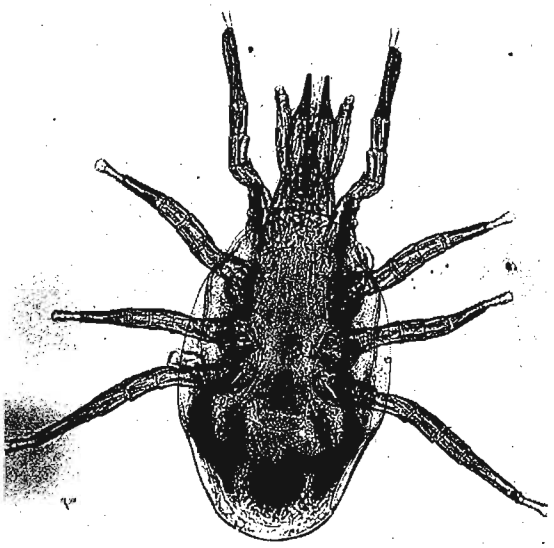
J.K. Choo is now at the Physiological Department, National Institute of Genetics, Misima, Japan (from Chungang University, Seoul, Korea).

A. Shearn is now Assistant Professor in the Department of Biology, Johns Hopkins University, Baltimore, Maryland 21218 (from Yale University, New Haven, Conn.).

P.Dennis Smith is now Assistant Professor in the Department of Biology, Emory University, Atlanta, Georgia 30322 (from Storrs, Conn.)

Félix, R., J. Guzmán, M.E. de la Rosa and O. Olvera. Control of mites in Drosophila cultures.

Histiostoma sp. is the most serious pest found in Drosophila cultures, as the control of infestation with this predatory mite has proved to be more difficult than the well known contaminations with either molds or bacteria. Mites are introduced into laboratories when collections of wild flies are being made, or when Drosophila cultures received from other laboratories are not carefully examined before new cultures are started from such flies.



Histiostoma sp.

Histiostoma sp. has a hypopus stage which attaches itself to Drosophila, as well as to other insects. It differs from other less dangerous mites mainly by its absence of long hairs. Besides, the predatory mite has a squatting body build in contrast with the long and thinner non-parasitic mites. As female adults produce both male and female progeny by parthenogenesis, a laboratory might become infected from a single introduced mite in the hypopus stage.

The newly eclosed smallest nymphs thrive on the culture medium. After a week or so, they metamorphose into the migratory (hypopus) state. These hypopi, that develop in large numbers in old, infested cultures, are extremely active. They crawl up out of the culture medium and, as they penetrate tiny crevices, they may infest other cultures, unless very tightly stoppered. The migratory nymphs attach to any insect they may come into contact with, sucking the mouth parts into the insect. After ten days of attachment, they leave the host and grow to the adult stage on the surface of the medium, where they reproduce.

A heavy infestation which extended to stock cultures occurred last year at this laboratory. The mites were introduced with samples of wild flies regularly collected at several trapping sites in Mexico City. All the infested cultures were submitted to the treatment detailed below, in order to eliminate all the mites from the cultures. Contaminated bottles and instruments were heated in a furnace before washing. The instruments used to manipulate flies, as well as the surface of the microscope, the outer surface of bottles, and upper surface of tables were continuously washed with a solution of benzyl-benzoate (20%) in 96° ethanol.

To start new cultures, flies were examined under the microscope, to use only adults that were apparently free from mites. As it is difficult to avoid contamination of the new medium, the adults were allowed to lay eggs on it, only during a period of 24 hours. When a few of the small hypopus nymphs from contaminated flies, or from other cultures were found in the new medium, it was necessary to cover its surface with a solution of benzyl-benzoate (20%) in 96° ethanol. This treatment kills the nymphs, without producing any noticeable effect in Drosophila larvae, which develop to the adult stage without hinderance from depredatory mites. Newly emerged flies were transferred each day to new cultures, to avoid the attachment of mites which survived after the treatment.

In heavily infested cultures all of the flies died, and there was found to be a crowding of mite nymphs among Drosophila larvae. In such a case, it was necessary to apply another treatment, thoroughly washing the larvae by immersion in a solution of benzyl-benzoate (20%) in ethanol. After 2-4 minutes in the benzly-benzoate solution larvae were washed with Ringer solution and transferred to fresh vials. Following the above steps, the pest was effectively controlled after three weeks.

Grossfield, J. and J. Smith. Purdue University, Lafayette, Indiana.
Video taping Drosophila behavior.

In the course of working out some details of the behavior of species that require light in order to mate, it was necessary to ascertain whether or not the beasts made contact with one another in darkness (They do). The system we used has general applicability for the analysis of Drosophila behavior. A TV camera with its lens removed (A in Fig. 1) is mounted vertically on a trinocular dissecting scope. This allows the microscope adjustment to focus the camera. A 10X eyepiece is located in the phototube supporting the camera. A lower power would give a wider field of view. The problems of glare from wings and thorax and heavy shadows can be compensated for by diffusing incident light, placing a set of polarizers in the light path, balancing light with aluminum foil reflectors, and using a deep pile underlay (velvet) on the bottom of the lucite observation chamber. A 1/4 wave plate

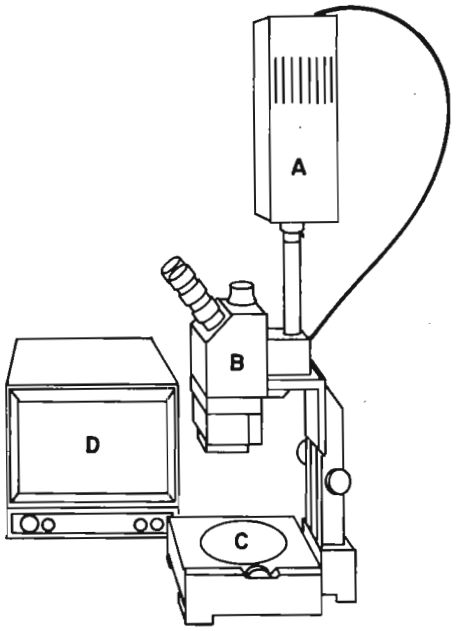



Figure 1. A. TV Camera; B. Dissecting scope with trinocular head; C. Observation Chamber, flat surface; D. TV monitor.

can also be used to cut glare. Any remaining glare can be compensated for by turning down the automatic gain control on both the camera and the monitor. A light coat of vaseline on the inside vertical surfaces of the observation chamber is reasonably effective in keeping the flies from assuming poorly photogenic positions on the corners or walls of the chamber.

For work in the dark a flashlight with a red filter (650 nm cut off, no UV transmittance) is a sufficient light source since the vidicon tube in the TV camera is sensitive to infrared light. IR Image Converter equipment can be used to work with wavelengths further towards that region of the spectrum (RCA laboratories, David Sarnoff Research Center, Princeton, N.J.).

A videotape recorder can be interposed in the system. This yields the capability to stop action at any point in a behavioral sequence and measure distances (angles of body parts, etc.) on the face of the TV monitor during playback. The videotape records can, of course, be stored to form a library of behavioral activities. In the long run this method is less expensive than using and processing 16mm film.

If you have sufficient funds to think about color TV, we'd be glad to hear where you got them.

Zalokar, M. Centre de Génétique Moléculaire, CNRS 91, Gif sur Yvette (France).
Fixation of Drosophila eggs without pricking.

Because of the impermeability of the vitelline membrane, the usual cytological fixatives can not penetrate the Drosophila egg and the egg has to be pricked to facilitate their entry. Only Carnoy fixative can be used directly and then only if its content of chloroform is higher than in recommended formulas, but this fixation shrinks eggs very badly.

Lipid solvents can penetrate the vitelline membrane, and if they contain a fixative in solution, they can carry it across the membrane. Any fixative which is soluble both in the solvent and in water will diffuse into the ooplasm and partition itself between its aqueous phase and the solvent according to the phase rule. If we want to fix an egg with 50% acetic acid, we should shake the solvent with the acid of this concentration. The solvent will take up the acid at the proper concentration so that the acid entering an egg submerged in the solution will reach 50%.

If we use a solvent which does not disrupt the egg lipids too drastically, we can achieve

fixation which is equivalent to fixation by the corresponding aqueous fixative. It was found that heptane or octane did not injure the cytoplasm unduly, while penetrating well through the vitelline membrane. An egg remains alive if submerged in these solvents for 10 minutes or more. The eggs become fixed in heptane loaded with acetic acid, picric acid, acrolein or glutaraldehyde, in less than one minute and can remain in the fixative for several minutes before beginning to shrink.

In order to facilitate the penetration of post-fixatives, colorants or dehydrating liquids, the vitelline membrane should be removed after initial fixation. To do this, the egg is transferred into the aqueous phase of the fixative and the membrane torn away with sharp needles. Surface tension helps to remove the membrane and the egg falls into the liquid. This operation can be performed best in 30% acetic acid, but after some practice, one can do it also in other fixatives.

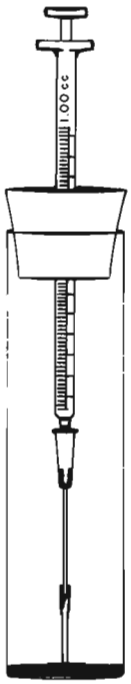
Fixation in heptane containing acrolein or glutaraldehyde is quite adequate for electron microscopy. Cell inclusions and organelles are well fixed, the ergastoplasm has its normal appearance and mitochondria have well perserved cristae. The following procedure is used:

1. Dechorionate eggs.
2. Fix in heptane which has been shaken with a 10% solution of acrolein or 25% solution of glutaraldehyde, for 1 to 2 minutes.
3. Remove the vitelline membrane in a buffered glutaraldehyde solution (conventional electron microscopy fixative).
4. Fix in the same solution for 1 hour.
5. Wash with buffered physiological solution.
6. Post fix with osmic acid 2 to 24 hours.
7. Further processing for embedding like any other tissue.

This fixation may be useful also in other cases where lipophilic membranes prevent the penetration of the usual fixatives, e.g. to fix Drosophila larvae and adults.

Félix, R. National Commission of Nuclear Energy, Mexico City, Mexico. A system for feeding liquids to adult flies.

The following method may be used as an alternative to injection of solutions to Drosophila flies, especially when several treatments with liquids should be tested in adults at separate time intervals. This system is particularly effective as the solution is administered during a period of time that may be lengthened to several days. It proved effectual for feeding cyclamates and cyclohexylamine to Drosophila melanogaster.



The liquid is gradually injected by means of a thin hypodermic syringe that goes through a hole of a rubber plug occluding the 2.8 x 9.0 cm vial, into a double layer of filter paper. The piece of polietilene tubing (Intramedic, Clay Adams, Inc.) adapted to the needle of the syringe, touches the filter paper, assuring a continuous delivery of the solution, when the embolus is pushed in.

The quality of filter paper cut to fit the bottom of the vial is important because it must be sufficiently absorbent to remain moist, without retaining an excessive amount of solution, which would drown the flies. Whatman 3 filter paper was used for such a purpose.

The syringe may be removed without the removal of the rubber plug, thus avoiding the escape, as well as the squashing of the flies, that occurs if the plug is removed and replaced. The amount of solution contained in the syringe (1.00 cc B.D. Yale turbeculin, Becton Dickinson) is enough to feed flies during several days. An additional pasteur pipette made at the laboratory with thin glass tube may be adapted through another hole, assuring the proper aeration of the vial, if the system is to be used during a period of several days without the removal of the flies.

Erk, F.C., H.V. Samis, M.B. Baird and H.R. Massie. Masonic Medical Research Laboratory, Utica, New York. A method for the establishment and maintenance of an aging colony of Drosophila.

The routine maintenance of an aging colony is highly desirable for studies involving senescence in Drosophila. A major difficulty in maintaining such a colony is the lack of coincidence between the generation time of D. melanogaster and the normal work week. In this note the details are given of a method for the initiation and maintenance of such a colony, as developed in this laboratory. The method will be described in terms of codes we arbitrarily use. Details of the environmental conditions for the maintenance of the colony are given elsewhere[1].

The original stock of D. melanogaster (Oregon-R) was obtained from the Division of Biological Sciences, State University of New York at Stony Brook. These flies, designated $_0P$ (parental), were allowed to lay eggs over a single 24-hour period; these eggs were collected on Thursday (Th). Cultures resulting from these eggs were labelled $A_1P$ (A group, parental). Eggs were again collected on Friday (F), and these cultures were designated $A_0A$ (A group, Aging Colony). During the following week, eggs from $_0P$ were again collected on Th, but labelled $B_1P$. Eggs from $_0P$ collected on F were labelled $_0B_0A$ (B group, Aging Colony). By Monday (M) of the third week, $_0A_1P$ flies had eclosed and were transferred to fresh medium. $A_0A$ flies eclosed on Tuesday (T) and entered the aging colony. $_0P$ eggs were collected on Th and lebelled $C_1P$, while eggs collected on Friday were labelled $C_0^0A$ (C group, Aging Colony). $_0P$ flies were then discarded.

During week 4, $B_1P$ flies were collected on M and placed on fresh medium, and $B_0A$ flies entered the aging colony on T. Eggs from $A_1P$ flies were collected on Th and designated $A_2P$, while $A_1P$ eggs collected on F were designated $A_1A$. The $A_1P$ flies were then mixed with $A_0A$ flies which were already in the aging colony; these groups of flies had the same parents. This process is repeated during week 5 and subsequent weeks. During week 5, for example, $C_1P$ flies were placed on fresh medium on M, $C_0A$ flies were placed in the aging colony on T. Eggs from $B_1P$ were collected on Th and designated $B_2P$, while eggs collected on F were labelled $B_1A$.

It becomes apparent (with pencil, paper, and time) that an aging colony of Drosophila may be maintained conveniently through standardized manipulation of these 3 groups of flies, all of which were derived from common parental stock. The continued input to the aging colony (e.g., $A_1P$ and $A_0A$) may be adjusted to experimental needs. Our usual input is approximately 10,000 flies (mixed males and females) per week, but is being expanded to 30,000 flies per week. This method produces large numbers of flies which are one week apart in age. In addition, the parental age of all flies in the aging colony is held constant at 11-12 days.

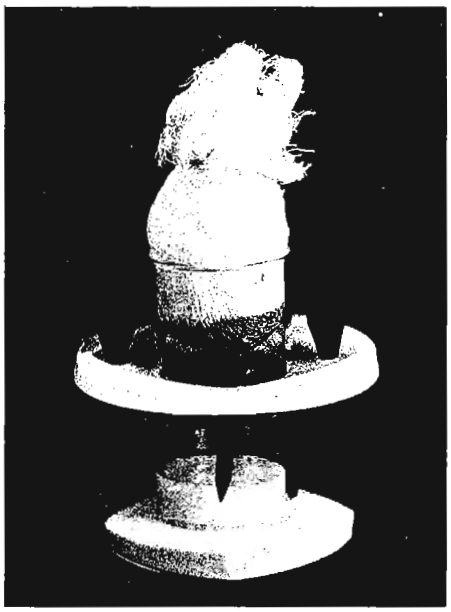
Reference: 1. Samis, H.V., Jr., Erk, F.C. and Baird, M.B. 1971. Exper. Gerontol. 6: 9-18.

Merriam, J.R., University of California, Los Angeles, California. A low cost disposable bottle for Drosophila culture.

Propak-California Corp., 211 N. Willow Ave. City of Industry, Ca. 91746 (phone 213-968-6447) sells 1/2 pint cylindrical milk bottles 5.3 cm in diameter, made of translucent plastic, which we use for all our cultures. The bottles are transparent enough so that the food condition and the pupae on the bottle walls can be easily seen. Although we originally bought them because of their low cost (currently $1.70 per 100 bottles) other advantages over the usual glass bottles are also important: they weigh much less and take up less space. A shipping carton with 400 bottles weighs just 15 3/4 lbs. This enables us to use low cost baskets "homemade" from welded wire fending. We plug the bottles with dispo plugs 28 x 35 mm obtained from Scientific Products. Snap lids did not work. We find it economical to clean and reuse the bottles, although other labs might want to use them for large one-shot projects or classes, or as a low cost reserve. Schools that want to set up fly keeping on a small scale or for a limited time will find these bottles especially attractive. If anyone would like to see a bottle we will be glad to send an empty on request within the U.S.

Hedrick, P.W. University of Kansas, Lawrence, Kansas. A culture which allows sand pupation.

Many species of Drosophila prefer to pupate in sand rather than on paper or the sides of a culture bottle. Several types of cultures have been used for these Drosophila but they usually entail transferring the larvae to a second container. The device I am using now for study of niche separation permits larvae to have the option of pupating either in sand or on the side of a chimney. Furthermore, this container permits downward migration of larvae into the sand simulating the behavior of Drosophila larvae in fallen fruit.



The container used is a clear plastic, eight ounce refrigerator container made by Deka Plastics, Inc. (see photograph). In the bottom of the container is placed approximately one eighth cup of sterilized white sand. In order to prevent dehydration of the media, the sand is saturated with water. The media is poured into a 3 inch high, 1-1/2 inch diameter pyrex chimney which has been placed on aluminum foil. After the media has set, the foil is peeled away and the chimney slipped though a hole in the container lid made earlier by a hot pyrex chimney. Larvae or adult flies are placed in the chimney and it is stoppered by a cotton-cheesecloth plug.

One must use an aspirator in order to remove flies from the container. To remove those inside the container, an aspirator tube is placed inside a plug. Flies which emerge outside the chimney, that is pupate in the sand, are aspirated through several holes which have been drilled in the container lid. These are stoppered by golf tees when not is use. Even with saturated sand, I have encountered some shrinkage of the media. This is dependent on the media used, the humidity, and the amount of larvae working. When adult flies are used, I suggest that they be allowed to lay on the media for 24-48 hours before placing the chimney in the container. I am indebted to J.S.F. Barker for suggesting many of the ideas in the design of this container.

Tomkins, J.K. and T. Billington. Monash University, Clayton, Vic., Australia. Analysis of D. melanogaster RNA by acrylamide gel electrophoresis of single fly homogenates.

The acrylamide gel electrophoresis procedure of Loening (1967) as modified by Becker et al. (1971) has been adapted for the analysis of RNA from homogenates of single individuals of D. melanogaster developmental stages. Third instar, pupa or adult individuals have been used successfully in this study. The method permits the resolution of ribosomal and transfer RNA as distinct sharp bands.

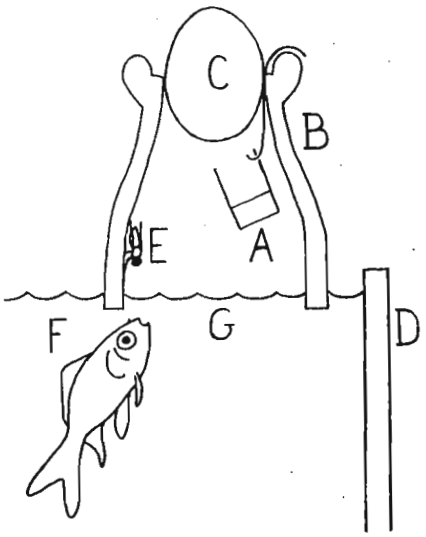
An individual of the developmental stage to be studied is briefly washed in distilled water before transfer to a small all-glass homogenizer. Homogenization is carried out at room temperature in 0.1 ml Loening's electrophoresis buffer containing 1% (w/v) sodium dodecyl sulphate (SDS) and 10% (w/v) sucrose. The homogenate is then stood at room temperature for 1 1/2 hours.

Acrylamide gels are prepared by the method of Loening. The gels are pre-run at 5 mA/gel for 1 hour at 4° in electrophoresis buffer containing 0.1% (w/v) SDS. This pre-run is in the direction the sample electrophoresis is to take place. The buffer is then renewed and a further pre-run, in the opposite direction, of 1 hour is carried out.

The homogenate is applied to the gels and electrophoresis at 5 mA/gel in fresh 0.1% SDS buffer is carried out at 4°. After electrophoresis the gels are removed from the tubes, fixed, stained and destained according to the method of Solymosy et al. (1970).

References: Loening, U.E., 1967, Biochem. J. 102: 251-257; Becker, H., C.P. Stanners, and J.E. Kudlow, 1971, J. Cell. Physiol. 77: 43-50; Solymosy, F., G. Lazar and G. Bagi, 1970, Anal. Biochem. 38: 40-45.

Wong, P.T. and W.E. Trout III. City of Hope National Medical Center, Duarte, California. The psychic fly fish feeder: A reply to Ogden Nash.

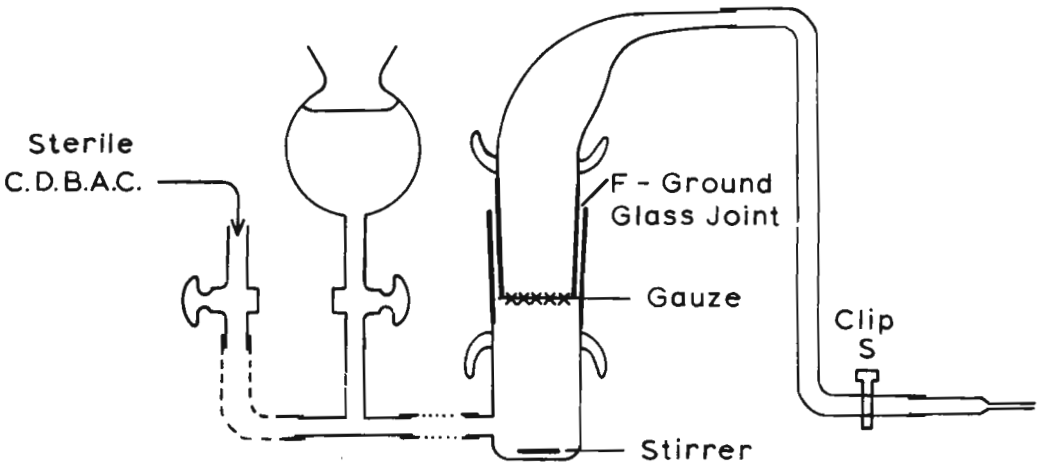



"The Lord in His wisdom made the fly
And then forgot to tell us why."
The Fly, by Ogden Nash

A culture of the neurological mutant $Hk^1$ (who falls over when he sees something moving), is placed in container (A), in bottomless bottle (B), plugged with cotton (C), which is suspended at the surface of the water in aquarium (D). When a fly emerges (E) and sees fish (F) moving below him, he loses control of himself and falls into the water (G) thereby feeding fish (F).

Sparrow, J.C. University of Sussex, Brighton, England. Eggwashing apparatus.

This apparatus replaces the reversing pump described by Sang (1956) in our Drosophila egg sterilization procedure for axenic culture. The eggs are collected by the method of Sang (1956), dechorionated with hypochlorite and suspended in 1.0% saline. This stops the dechorionated eggs from aggregating. Any larvae are removed by hand using a binocular microscope. The eggs are then placed in the apparatus through the thistle funnel and enter the egg chamber. They are washed in this chamber by a sterile 0.1% C.D.B.A.C. (cetyldimethylbenzylammonium chloride) solution which runs continuously through the apparatus. The flow rate is controlled by the screw clip (S) at a rate of 90 drops/minute




Egg Chamber

from the standard pasteur pipette. The eggs are kept in suspension and from sticking to the gauze by means of a small magnetic stirrer, revolving at about 74 revs./minute. The figures for flow rate and stirring are those which give optimum egg-hatching rates with minimum culture infection. The eggs undergo 15 minutes of this treatment and then the egg chamber is removed from the apparatus. The egg chamber must be washed through with sterile water before plating so as to remove any C.D.B.A.C. Prolonged exposure to this detergent was found to kill eggs. Some strains appear more sensitive than others to the length of time spent in C.D.B.A.C. The egg chamber is then dismantled at (F) in a sterile hood and the eggs plated on agar using the procedure of Sang (1956).

Reference: Sang, J.H. (1956). J. Exp. Biol. 33:45-72.

Lewis, E.B. and L. Craymer. California Institute of Technology, Pasadena, California. Quinacrine fluorescence of Drosophila chromosomes.

We describe below a modification we have developed for Drosophila of the quinacrine-fluorescent staining methods developed by Caspersson and his colleagues (Expt. Cell Res. 58: 141-151, 1969) for plant and, later, human chromosomes. Our findings are in general agreement with those of Vosa (Chromosoma 31: 446-451, 1970) who reports selective staining with this dye of the long arm of the Y chromosome and of the fourth chromosome in Drosophila melanogaster. In addition we have observed a bright fluorescing band in region 81F (3R) of the salivary gland chromosomes. Also in larval ganglion metaphases a weakly fluorescent spot is visible in the basal heterochromatic region of X, 2L, 2R and 3L, while 3R has two such spots. The Y has at least three strongly fluorescing spots in Y-long and at least one in Y-short ($Y^{closed}$ has five spots visible); the fourth chromosomes appear at metaphase as two very bright fluorescent bodies. Adult muscle or brain tissues show in resting nuclei a large, usually single, fluorescent body, which may represent a chromocentral fusion of the fluorescent spots found in the basal part of each chromosome arm. The XY male has a somewhat brighter body, evidently due to fusion of the Y chromosome as well. It may prove possible therefore to "sex" somatic resting nuclei of adult tissues.

The resting nuclei of imaginal disc cells also have a single large fluorescing body in XX or XO tissues but tend to have two such bodies, of approximately equal fluorescent intensity, in XY or XXY resting nuclei. We interpret this to mean that in these rapidly dividing tissues the Y often does not fuse with the chromocenter. We have also extended this observation by studying males carrying an extra Y-long arm attached to X in addition to a normal Y. Such males often show three fluorescing bodies in the resting nuclei of their imaginal wing discs instead of two, suggesting that extra Y's do not tend to fuse with one another in imaginal disc tissue.

A. Procedure for staining Drosophila salivary gland chromosomes or resting nuclei of many larval or adult (except brain) tissues.

1. Dissect larvae in 45% acetic acid. Place tissue in a small drop of 45% acetic acid on a siliconed coverslip. Lower a slide which has been "subbed"* over the drop; after it touches the drop, invert the slide; blot to remove excess mounting fluid; tap the coverslip sharply over the tissue area with a blunt instrument to disperse cells; cover with absorbent tissue and squash with strong pressure.

2. Immerse slide in liquid nitrogen until bubbling stops (or freeze on a block of dry ice).

3. Pry off coverslip with a razor blade. Dip slide in 95% alcohol for about a minute and then into absolute alcohol for a minute. Remove and dry by waving in the air.

4. Flood area over tissue with a few drops of an 0.5% to 2% solution of quinacrine hydrochloride in 45% acetic acid and stain for one or two minutes. (Batches of Gurr's "atebrin" or Sigma's quinacrine have proved satisfactory.)

5. Drain off staining solution and quickly dip slide into a jar of 95% ethanol followed by one or two transfers through absolute ethanol. The total time in the alcohols should be 20 seconds or less at 25°C to avoid excessive destaining. Insufficient rinsing may result in excessive background fluorescence in the final preparation.

6. Remove slide from the absolute ethanol and quickly dry by waving the slide vigorously in air. Heating at this stage tends to destain the preparation.

7. To mount, place a drop of sucrose solution (0.5 to 1.0 molar in double distilled water) in the center of a coverslip. Invert the slide over this coverslip until it touches

the drop. Quickly reinvert and blot firmly to remove excess mounting fluid. Seal with clear nail polish.

B. Procedure for staining adult brain tissue.

1. The same procedure as that described for the larvae is used except that the percentage of acetic acid in both the dissecting fluid and the quinacrine staining solution is reduced from 45% to 10%.

C. Procedure for staining larval ganglia for metaphase chromosome studies (modified from DIS 34: 118-119).

1. Dissect larvae in a solution of 1.0% Na Citrate in distilled water. Place the dorsal ganglia in a drop of this solution for 10 minutes on a slide. Warm the slide on a hot plate at 40°C for one minute (this hastens separation of sister chromatids). Pass the ganglia into a pre-fixative composed of equal parts of 45% acetic acid and 95% ethanol and leave for 30 seconds. Then remove tissue and place in a drop of 45% acetic acid on a siliconed coverslip. Continue with procedure described in part A, par. 1 above.

*3 gm gelatin. 600 ml distilled water. Heat to dissolve gelatin. Cool. Add chrom. alum - $KCr(SO_4) \cdot 12 H_2$) - 300 mg. Dip slides, drain and allow to dry in dust-free container.

## TEACHING NOTE

Potter, J.H. University of Maryland, College Park, Maryland. A demonstration of compensation for an inherited biochemcial defect in D. melanogaster.

A simple demonstration of compensation for an inherited biochemical defect can be carried out by beginning students using D. melanogaster. In essence, students supply kynurenine to larvae of vermillion mutants which cannot convert tryptophan to kynurenine, one of the steps in the synthesis of omnochrome pigments. Since students frequently do not distinguish vermillion from wild type flies, they use the white-eyed, double mutant, vermillion brown. Vermillion brown larvae fed kynurenine develop brown eyes. To emphasize the specificity of the block, students also feed kynurenine to the double mutant, cinnabar brown. Cinnabar brown mutants develop white eyes whether or not they receive kynurenine.

Experimental procedure: Students set up two cultures each of vermillion brown and cinnabar brown mutants in 80 x 25 mm. shell vials containing 5 ml of Carolina Instant Drosophila Medium. As soon as larvae appear the parents are removed and the medium in one vial of each genotype is injected with 0.2 ml of a kynurenine-antibiotic solution. The medium in the other two vials is injected with 0.2 ml of plain antibiotic solution. The injections are made with a 2 1/2 ml syringe without a needle inserted in a hole made in the medium with an applicator stick. Injections are repeated every two days until pupae appear. The adults are scored in the usual way. The kynurenine treated, vermillion brown, flies are mated after scoring and their progeny scored for eye color to demonstrate that the genotype has not been changed by the kynurenine treatment.

The kynurenine antibiotic solution is similar to that used by Parsons and Green (1959) for culturing eye discs: 0.05% streptomycin, 0.033% penicillin and 1.00% D.L. kynurenine can be obtained from Sigma Chemical Co., St. Louis, Missouri, at $14.00/gram.

References: Parsons, P.A. and M.M. Green, 1959, Proc. Nat. Acad. Sci., Wash. 45: 993.

## MATERIALS REQUESTED OR AVAILABLE

H.R. Feijen, University of Malawi, Genetics Section, P.O. Box 5200, Limbe, Malawi, would be grateful to obtain reprints on speciation in Drosophila and reprints on systematics of Drosophila.

R.C. King, Dept. of Biology, Northwestern University, Evanston, Illinois, is undertaking the editing of a Handbook of Genetics for the Van Nostrand-Reinhold Company, and intends to include a cytogenetic map of Drosophila melanogaster. In his monograph on Ovarian Development in Drosophila melanogaster, he published a fold-out which included the cytological localization of about 71 genes or gene clusters. This used the data from Lindsley and Grell which by now are four years old. He would therefore appreciate hearing from Drosophila workers who have new data (published or unpublished) that would enable him to update the comparative maps. Also any suggestions as to other material that should be covered will be greatly appreciated.

Discontinuance of Drosophila Stock Service: After 31 years, the Drosophila service at Cold Spring Harbor has been discontinued. Copies of the "Drosophila Guide" will continue to be available from: Office of Information, Carnegie Institution of Washington, 1530 P Street, Northwest, Washington, D.C. 20005. Flies can be purchased from several sources, including the following: Carolina Biological Supply Company, Burlington, North Carolina, 27215 and Gladstone, Oregon 97027; Ward's Natural Science Establishment, Inc., Post Office Box 1712 Rochester, New York 14603.

The Seton Hall University stock list has been discontinued.

Announcing publication of "Ovarian Development in Drosophila melanogaster," by R.C. King, Department of Biological Sciences, Northwestern University, Evanston, Illinois, June 1970, 227 pp, $16.50, Academic Press, New York, N.Y.

The Egyptian Society of Genetics is putting out a new Journal entitled "Egyptian Journal of Genetics and Cytology". Submission of publications from the different fields of genetics and cytology is encouraged. It is hoped that this new Journal will be of great help to researchers in genetics all over the world. The Journal will be in two numbers and about 300 pp. a year, starting Vol. 1 No. 1, January 1972. The subscription rates are $10 for institutions and $5 for individuals. Subscriptions are to be ordered through the Editorial Office (Prof. A.O. Tantawy, Dept. of Genetics, Faculty of Agriculture, Alexandria University, Alexandria, Egypt). All checks should be made payable to the Egyptian Society of Genetics.

The Genetics and Biology of Drosophila. Announcement of a proposed three volume work, intended to cover comprehensively the genetics and biology of Drosophila.

The field of Drosophila research suffers from the absence of any modern comprehensive account of the biology and genetics of the organism. No complete account of Drosophila genetics has been published since 1925, although individual topics have often been well reviewed in recent years. In addition the current accounts of Drosophila biology (Demerec) and evolution (Patterson and Stone) are, although irreplacable, either out of date of fail to cover the many fields of research which have opened up dramatically in the last 20 years. The size and breadth of the current Drosophila literature is a severe handicap to students starting research in Drosophila, to workers switching to Drosophila from other fields and to Drosophila workers themselves with interests in aspects of Drosophila research other than their immediate research interest.

The current project, to publish in three volumes an account of the genetics and biology of Drosophila, is an attempt to fulfill the requirements of the research biologist. Each volume will consist of a number of chapters by invited authors. The standing brief to each author is that he makes an attempt to cover comprehensively his particular topic with emphasis on its historical context and in particular the techniques used and its place in modern Drosophila research. Although emphasis will obviously be on melanogaster, especially in the volume on genetics, information from other species, and where necessary from other insects, will also be included. It would, for example, be quite illogical to consider certain aspects of the developmental biology of Drosophila without drawing on the literature concerned with similar problems in other species of flies and other insect groups.

The three volumes, which will be published consecutively at yearly intervals, will be as follows: Vol. I. Genetics; Vol. II Biology and Development; Vol. III Evolution, Taxonomy and Ecology. Volume I is being edited by Michael Ashburner and Ed Novitski and is planned for publication in 1973.

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Fujii, H.M. Japan, Fukuoka
Fujii, S. Japan, Kobe
Fujimoto, M. Japan, Tokyo
Fukatami, A. Japan, Sakado-Machi
Fulch, D.G. San Diego, California
Fullilove, S.L. Austin, Texas
Gabay, S.J. Urbana, Illinois
Gale, J.S. England, Birmingham
Galia, M.S. Brazil, Pôrto Alegre
Gall, J.G. New Haven, Connecticut
Galmuzzi, G. Italy, Naples
Ganguly, R. India, Calcutta
Garcia-Bellido, A. Spain, Madrid
Garcia, P. Spain, Barcelona
Garen, A. New Haven, Connecticut
Garrido, P. Spain, Madrid
Gavin, J. Canada, Calgary
Gay, H. Ann Arbor, Michigan
Gay, P. France, Gif-sur-Yvette
Gaylord, C.A. Washington, D.C.
Greer, B.W. Galesburg, Illinois
Gehring, W. New Haven, Connecticut
Gelbart, W.M. Pasadena, California
Geltosky, J.E. Pasadena, California
Gerdy, J.R. Carbondale, Illinois
Gerresheim, F. Germany, München
Gersh, E.S. Pasadena, California
Gethmann, R.C. San Diego, California
Gibson, J.B. England, Cambridge
Gill, R.W. Riverside, California
Glass, B. Stony Brook, New York
Glätzer, K.H. Germany, Düsseldorf
Gloor, H. Switzerland, Geneva
Gnes, A. Italy, Padova
Gochberg, C. Madison, Wisconsin
Godbole, N.N. India, Poona
Gold, E.E. Urbana, Illinois
Gold, J.R. Davis, California
Gonzales, F.W. Upton, New York
González, C. Spain, Barcelona
González, R. Spain, Barcelona
Gorodenski, S.A. Raleigh, North Carolina
Gorospe, Mª J. Spain, Madrid
Gossi, S.J. Pullman, Washington
Gottlieb, F.J. Pittsburgh, Pennsylvania
Götz, K.G. Germany, Tübingen
Gouveia, G. England, Oxford
Grabicki, E. New Haven, Connecticut
Grace, D. Netherlands, Leiden
Graf, U. Switzerland, Zürich
Graham, R.S. Chapel Hill, North Carolina
Granobles, L.A. Colombia, Bogotá
Grant, B.S. Williamsburg, Virginia
Gray, K.F. Willimsburg, Virginia
Graziano, M.H. Charlottesville, Virginia
Green, M.M. Davis, California
Greer, G. Australia, Bundoora
Greisen, K.S. Pasadena, California
Grell, E.H. Oak Ridge, Tennessee
Grell, R.F. Oak Ridge, Tennessee
Grimes, W.P. Dallas, Texas
Groh, G. Germany, Berlin
Groot-van Stralen, C. Th. de Netherlands, Leiden
Grossfield, J. Lafayette, Indiana
Grüneberg, H. England, London
Gruwez, G. Belgium, Heverlee
Guedes, M.A. Brazil, Pôrto Alegre
Guest, W.C. Fayetteville, Arkansas
Haapala, O. Finland, Turku
Hablas, A.A. U.A.R., Alexandria
Hackman, W. Finland, Helsinki
Hadorn, E. Switzerland, Zürich
Haendle, J. Germany München
Hale, G. New York, New York
Halfer, C. Milan, Italy
Hall, J. Seattle Washington
Hall, L. Canada, Vancouver
Halm, J. Houston, Texas
Hama, H. Japan, Chiba
Hamdalla, H. U.A.R. Assuit
Hammerschmidt, H. Germany, München
Hammond, K. Australia, Sydney
Hanks, G.D. Gary, Indiana
Hannah-Alava, A. Finland, Turku
Hanratty, W.P. Pittsburgh, Pennsylvania
Hanson, T.E. Pasadena, California
Hanstein, B. Duarte, California
Hara, Miss Japan, Fukuoka
Hardwick, J.S. Chapel Hill, North Carolina
Hardy, D.E. Honolulu, Hawaii
Hardy, R.W. San Diego, California
Harrison, B. England, Sheffield
Harrison, B.J. England, Norwich
Harrod, M.J.E. Dallas, Texas
Hartley, P. Durham, North Carolina
Hartmann-Goldstein, I.J. England, Sheffield
Harvey, K. Ithaca, New York
Hashem, Y.D. U.A.R., Alexandria
Hashim, M. U.A.R. Assuit
Hauri, H.-P. Switzerland, Zürich
Havermans, E.M.J. Netherlands, Utrecht
Hawk, J.A. Lafayette, Indiana
Hawkins, E. Stony Brook, New York
Hay, D.A. England, Birmingham
Hayashi, K. Japan, Osaka
Hazevoet, I. Netherlands, Leiden
Hebert, P. England, Cambridge
Heisenberg, M. Germany, Tübingen
Hengstenberg, B. Germany, Tübingen
Henstenberg, R. Germany, Tübingen
Hennig, I. Germany, Tübingen
Hennig, W. Germany, Tübingen
Hensen, A.E. Netherlands, Leiden
Hess, O. Germany, Düsseldorf
Hewitt, N.E. Washington, D.C.
Hihara, F. Japan, Tokyo
Hihara, Y.K. Japan, Tokyo
Hill, C.L. Madison, Wisconsin

Hillman, R. Philadelphia, Pennsylvania
Hinton, C.W. Wooster, Ohio
Hippard, J. Los Angeles, California
Hiraizumi, Y. Austin, Texas
Hirose, Y. Japan, Kobe
Hiroyoshi, T. Japan, Osaka
Hodges, C. Raleigh, North Carolina
Hodgetts, R.B. Canada, Edmonton
Hoenigsberg, H.F.B. Colombia, Bogotá
Hoff, D. Madison, Wisconsin
Hofman, J.D.D. Netherlands, Haren
Holden, J. Canada, Vancouver
Hollander, W.F. Ames, Iowa
Hollie, E. Austin, Texas
Hollingsworth, M.J. England, London
Hollingworth, M. Australia, Brisbane
Hollis, R.J. Williamsburg, Virginia
Holzworth, K.W. Pitsburgh, Pennsylvania
Honda, Y. Japan, Nagasaki
Hooper, G.B. Poughkeepsie, New York
Hoste, C. Belgium, Heverlee
Hotta, Y. Pasadena, California
Hoyland, M. England, Sheffield
Hoyt, J.P. Ann Arbor, Michigan
Huang, S.-L. Austin, Texas
Huang, S.-M. Austin, Texas
Hubby, J.L. Chicago, Illinois
Hubert, L.M. Brazil, Pôrto Alegre
Hudson, A.S. Chapel Hill, North Carolina
Hudson, G. England, Cambridge
Hunt, D.M. England, London
Hürlimann, R. Switzerland, Zürich
Huth, A.C. Germany, Tübingen
Ibrahim, H. U.A.R., Assuit
Ibrahim, S. U.A.R., Alexandria
Ikeda, H. Japan, Tokyo
Ikeda, K. Duarte, California
Illmensee, K. Germany, München
Imaizumi, Y. Japan, Chiba
Inove, A. Japan, Nagasaki
Ito, S. Japan, Tokyo
Ivic, G. Yugoslavia, Belgrade
Iyama, S. Japan, Misima
Jack, K. Garden City, New York
Jacobs, M.E. Goshen, Indiana
Jacobs, P.A. San Diego, California
Jacobson, A.G. Austin, Texas
James, J.W. Australia, Sydney
Janning, W. Germany, Münsten
Jefferson, M. Boulder, Colorado
Jeliasvčić, B. Ugoslavia, Belgrade
Jen, K.-d. DeKalb, Illinois
Jha, A.P. India, Bhagalpur
Jinks, J.L. England, Birmingham
Johnsen, R.C. Garden City, New York
Johnson, F.M. Raleigh, North Carolina
Johnson, G. Philadelphia, Pennsylvania
Johnson, J.H. Chicago, Illinois
Johnson, L. Madison, Wisconsin
Johnson, T. Northridge, California
Johnson, W.E. Honolulu, Hawaii
Johnston, C.J. Pasadena, California
Johnston, F. Storrs, Connecticut
Jones, D.A. England, Birmingham
Jones, G.H. England, Birmingham
Jones, J.S. Chicago, Illinois
Jones-Mortimer, M.C. England, Birmingham
Jonker, F.H. Netherlands, Haren
Jousset, F.X. France, St. Christol les Alès
Judd, B.H. Austin, Texas
Julien, J. Chicago, Illinois
Jungen, H. Switzerland, Zürich
Kachur, F. Gary, Indiana
Kaji, S. Japan, Kobe
Kalicki, H. Garden City, New York
Kalisch, W.-E. Davis, California
Kalisz, A. Brazil, Pôrto Alegre
Kamra, O.P. Canada, Halifax
Kanehisa, T. Japan, Kobe
Kaneshiro, K.Y. Honolulu, Hawaii
Kang, M.-J. Korea, Seoul
Kang, S.-H. Notre Dame, Indiana
Kang, (Song), S.-J. Korea, Seoul
Kang. Y.S. Korea, Seoul
Kankel, D.R. Pasadena, California
Kaplan, M.L. New York, New York
Kaplan, W.E. Duarte, California
Karlik, A. Austria, Vienna
Kastritsis, C.D. Dallas, Texas
Katt, S. Atlanta, Georgia
Kaufman, T.C. Canada, Vancouver
Kaufmann, B.P. Ann Arbor, Michigan
Kawabe, M. Japan, Kobe
Kawaharada, M. Japan, Nagasaki
Kawanishi, M. Japan, Misima
Kearney, M. England, Sheffield
Kearsey, M.J. England, Birmingham
Keith, M.J. Houston, Texas
Kekić, V. Yugoslavia, Belgrade
Kellen, C.M. Iowa City, Iowa
Keller, E.C. Morgantown, West Virginia
Keller, H.E. Morgantown, West Virginia
Kelly, J.F. Canton, New York
Kelly, M. Australia, Brisbane
Kercher, M.D. Lexington, Kentucky
Kernaghan, R.P. Stony Brook, New York
Kersten, H.J.M.G. Netherlands, Utrecht
Kessenich, M. Madison, Wisconsin
Kessler, S. Madison, Wisconsin
Khishin, A. U.A.R., Assuit
Kieft, P. Netherlands, Leiden
Kikkawa, H. Japan, Osaka
Kim, K.-J. Korea, Seoul
Kim, K.W. Korea, Kwangju
Kimura, M. Japan, Misima
King, R.C. Evanston, Illinois
Kiriasis, W.C. Upton, New York
Kirschbaum, W.F. Argentina, Buenos Aires
Kitagawa, O. Japan, Tokyo
Kitazume, Y. Japan, Kobe
Kitos, R. Syracuse, New York
Kleisch, U. Germany, Berlin

Klug, M. Germany, Freiburg
Knaap, A.G.A.C. Netherlands, Leiden
Kobel, H.R. Switzerland, Geneva
Koepfer, R. New York, New York
Köhler, B. Germany, Tübingen
Kojima, K.-I. Austin, Texas
Kolodzieg, G. Germany, Freiburg
Komori, M. Japan, Nagasaki
Konopka, R.J. Pasadena, California
Kooistra, J. Netherlands, Haren
Koref-Santibanez S. New York, New York
Korge, G. Germany, München
Korinek, E. Canada, Vancouver
Kosuda, K. Japan, Tokyo
Kothari, R.M. India, Poona
Kovarik, A. Austin, Texas
Kramer, P.G. Netherlands, Leiden
Kratz, F.L. Brazil, Pôrto Alegre
Krause, E. South Orange, New Jersey
Kreisman, D.J. Pullman, Washington
Kress, H. Germany, München
Kreteski, D. Atlanta, Georgia
Krimbas, C. Greece, Athens
Krivshenko, J.D. Rochester, New York
Krunić, M. Yugoslavia, Belgrade
Kubli, E. Switzerland, Zürich
Kucherlapati, R.S. Urbana, Illinois
Kuchino, C. Japan, Misima
Kunze-Mühl, E. Austria, Vienna
Kuroda, Y. Japan, Misima
Kurokawa, H. Japan, Tokyo
Label, E. France, Gif-sur-Yvette
Lachaise, D. France, Gif-sur-Yvette
Lai, Y.-H. Pasadena, California
Laird, C. Seattle, Washington
Lakovaara, S. Finland, Helsinki
Lamb, M.J. England, London
Landner, L. Sweden, Stockholm
Lang, E.L. Carbana, Illinois
Langley, C. Madison, Wisconsin
Langlois, B. France, Gif-sur-Yvette
LaPushin, R. Houston, Texas
Lasa, L. Spain, Madrid
Lasseter, A. Austin, Texas
Latter, B.D.H. Australia, Sydney
Laughnan, J.R. Urbana, Illinois
Laurent, J. France, Gif-sur-Yvette
Lawrence, M.J. England, Birmingham
Lebherz, H. Switzerland, Zürich
Lechien, J. Belgium, Namur
Lee, B.W. Korea, Chungang
Lee, C.C. Korea, Seoul
Lee, C.S. Korea, Chungang
Lee, J.-Y. Pasadena, California
Lee, K.-S. Korea, Seoul
Lee, L.Y. Lafayette, Indiana
Lee, T.J. Korea, Chungang
Lees, G. Duarte, California
Lefevre, G. Northridge, California
Leffel. L.E. Madison, Wisconsin
Legay, J.M. France, Lyon
Leightwood, J. Australia, Canberra
Lemeunier, F. France, Gif-sur-Yvette
Leonard, J. Purchase, New York
Leto, G. DeKalb, Illinois
Levine, H. Storrs, Connecticut
Levins, R. Chicago, Illinois
Lewandowski, Y. Buffalo, New York
Lewellyn, S. Australia, Canberra
Lewis, E.B. Pasadena, California
Lewis, J. Pasadena, California
Lewontin, R.C. Chicago, Illinois
Lezzi, M. Switzerland, Zürich
L'Helias, C. France, Gif-sur-Yvette
L'Héritier, Ph. France, Clermont-Ferrand
Libion, M. Belgium, Namur
Lidington, K. Lafayette, Indiana
Lieb, E. Germany, München
Lifschytz, E. San Diego, California
Limbird, D. Washington, D.C.
Lindsley, D.L. San Diego, California
Linney, R. England, Birmingham
Lints, C. Belgium, Heverlee
Lints, F.A. Belgium, Heverlee
Lipps, K. Davis, California
Liszczynskyj, J. Utica, New York
Llewellyn, M.A. Bellingham, Washington
Lloyd, B. Australia, Adelaide
LoCascio, H. Buffalo, New York
Lokki, J. Finland, Helsinki
Lommerse, M.A.H. Netherlands, Leiden
Long, G. Chapel Hill, North Carolina
Long, T.C. Chapel Hill, North Carolina
Loos, M.J. Netherlands, Leiden
Louis, M. Grance, Gif-sur-Yvette
Loukas, M. Greece, Athens
Lowy, P.H. Pasadena, California
Lu, C.C. Houston, Texas
Lu, M.-H. Austin, Texas
Lucas, K. New Haven, Connecticut
Lucchesi, J.C. Chapel Hill, North Carolina
Luce, W.M. Urbana, Illinois
Ludwig, M.R. Brazil, Pôrto Alegre
Lüers, H. Germany, Germany
Lumme, J. Finland, Helsinki
Luna, E. San Marcos, Texas
Lundelius, J. Austin, Texas
Lüning, K.G. Sweden, Stockholm
Lütolf, H.-U. Switzerland, Zürich
Lyttle, T.W. Madison, Wisconsin
Macaulay, S. Canada, Vancouver
MacBean, I.T. Australia, Bundoora
Machado, D.M. Brazil, Pôrto Alegre
Machida, I. Japan, Chiba
Machová, H. Czechoslovakia, Brno
Macht, V.C. Notre Dame, Indiana
MacPhail, S. Corvallis, Oregon
Madhaven, K. Switzerland, Zürich
Maeda, M. Japan, Misima
Maeda, Y. Japan, Kobe
Magalhães, L.E. de Brazil, São Paulo
Magnusson, J. Sweden, Stockholm

Maier, P. Switzerland, Zürich
Maier, V. Switzerland, Zürich
Mainx, F. Austria, Vienna
Maitra, S.N. India, Calcutta
Majoral, J. Spain, Barcelona
Mange, A. Seattle, Washington
Manna, P.K. India, Calcutta
Marien, D. New York, New York
Marinković, D. Yugoslavia
Markowitz, E.H. Madison, Wisconsin
Maroni, G. Madison, Wisconsin
Marques, E.K. Brazil, Pôrto Algere
Marree, C.M. Netherlands, Utrecht
Marsh, D. Tuscaloosa, Alabama
Martensen, D. Galesburg, Illinois
Marstokk, A. Norway, Oslo
Martin, A.O. Cleveland, Ohio
Martínez, J.M. Spain, Barcelona
Martinez, M.N. Brazil, Pôrto Alegre
Martinsen, D. Davis, California
Maruyama, T. Japan, Misima
Marynick, S.P. Dallas, Texas
Masry, A.M. U.A.R., Alexandria
Massie, H.R. Utica, New York
Masterson, J.E. Ames, Iowa
Masuda, H. Japan, Misima
Mather, W.B. Australia, Brisbane
Matheson, A.C. Australia, Bundoora
Matsubara, T. Japan, Nagasaki
Mayoh, H. Canada, Vancouver
Mazar Barnett, B. Argentina, Buenos Aires
McAdams, L.W. Raleigh, North Carolina
McCabe, K. Australia, Brisbane
McCaman, R. Duarte, California
McCarron, M. Storrs, Connecticut
McCormack, M.K. South Orange, New Jersey
McCrady, E. Greensboro, North Carolina
McCaman, M.W. Duarte, California
McDonald, R.P. England, London
McDonnell, J. Garden City, New York
McFarland, J.L. Riverside, California
McKenzie, G. Scotland, Aberdeen
McKenzie, J.A. Australia, Bundoora
McLean, J.L. Chapel Hill, North Carolina
McMahon, M. Utica, New York
McMurtrey, M. Houston, Texas
McNeil, H.M. Dallas, Texas
McNeil, R.M. Raleigh, North Carolina
McQue, R. Flagstaff, Arizona
Meeles, E. Netherlands, Haren
Meer, B. Germany, Tübingen
Megaheid, M.A. U.A.R., Assuit
Megna, F. Italy, Naples
Melon, I. Italy, Naples
Meltzer, P.S. Pasadena, California
Memhauser, I. New York, New York
Ménsua, J.L. Spain, Barcelona
Mercader, J. Mexico, Mexico City
Mercio, A.L. Brazil, Pôrto Alegre
Merrell, D.J. Minneapolis, Minnesota
Merriam, J.R. Los Angeles, California
Merritt, R. Rochester, New York
Messerschmid, V. Germany, München
Metcalfe, J.A. England, Heslington
Mettler, L.E. Raleigh, North Carolina
Meyer, G.F. Germany, Tübingen
Meyer, H.U. Madison, Wisconsin
Micheli, A. Italy, Rome
Miklos, G.L. San Diego, California
Mikušová, J. Netherlands, Utrecht
Milkman, R. Iowa City, Iowa
Miller, D.D. Lincoln, Nebraska
Miller, D.H. Australia, Sydney
Miller, S. Seattle, Washington
Millington-Ward, A.M. Netherlands, Leiden
Milton, M.K. Australia, Sydney
Minato, K. Japan, Misima
Mindek, Géza Switzerland, Zürich
Mishra, M. India, Bhagalpur
Mitchell, H.K. Pasadena, California
Mitchell, J.P. Tuscaloosa, Alabama
Mitra, N. India, Calcutta
Mittler, S. DeKalb, Illinois
Mizobuchi, K. Japan, Chiba
Mizuguchi, Y. Brazil, São Paulo
Moffitt, S. England, Oxford
Mohler, J. Iowa City, Iowa
Mohler, J.D. Iowa City, Iowa
Mohr, M.A. Houston, Texas
Moisand, R.E. Buffalo, New York
Mollet, P. Switzerland, Zürich
Monclús, M. Spain, Barcelona
Monjeló, L.A. dos S. Brazil, Pôrto Alegre
Montalenti, G. Italy, Rome
Montana, D. Syracuse, New York
Montelius, I. Sweden, Stockholm
Montgomery, S.M. Honolulu, Hawaii
Morales, N.B. Brazil, Pôrto Alegre
Moran, C. Corvallis, Oregon
Morata, G. Spain, Madrid
Moree, R. Pullman, Washington
Moreno, N.I. Colombia, Bogotá
Mori, S. Japan, Nagasaki
Moriwaki, D. Japan, Misima
Morrow, D. Canada, Calgary
Mortensen, M. Denmark, Copenhagen
Morton, M.D. Charlottesville, Virginia
Moskwinski, T. Notre Dame, Indiana
Mosna, G. Italy, Milan
Mossige, J. Norway, Oslo
Moth, J.J. Australia, Sydney
Moura, V.L.P. de Brazil, Pôrto Alegre
Mourad, A.M. U.A.R., Alexandria
Mueller, D. Iowa City, Iowa
Mukai, T. Raleigh, North Carolina
Mukherjee, A.S. India, Calcutta
Mukherjee, T.K. India, Calcutta
Mukherjee, U. Austria, Vienna
Mulley, J.C. Australia, Sydney
Munoz, E. Netherlands, Leiden
Muñoz, E.R. Argentina, Buenos Aires
Murakami, A. Japan, Misima

Murata, M. Japan, Chiba
Murch, A. Australia, Adelaide
Murnik, M.R. Illinois, Macomb
Muzyka, G. Galesburg, Illinois
Myszewski, M.E. Des Moines, Iowa
Nagib, F.M. U.A.R., Alexandria
Naguib, F.N.M. Canada, Edmonton
Nahas, P. Notre Dame, Indiana
Nakai, S. Japan, Osaka
Nakai, S. Japan, Chiba
Nakashima-Tanaka, E. Japan, Sakai
Narise, S. Japan, Sakado
Narise, T. Japan, Sadado-Machi
Nash, D. Canada, Edmonton
Nash, W.G. Washington, D.C.
Natori, S. New Haven, Connecticut
Nawa, S. Japan, Misima
Nayudu, P.L. Australia, Clayton
Neeley, J.C. Portland, Oregon
Neto, C.C. Brazil, Pôrto Alegre
Nettleton, R.W. Lincoln, Nebraska
Nevin, E. Gary, Indiana
Newton, J.A. Chapel Hill, North Carolina
Nicoletti, B. Italy, Rome
Miet, J.P. van der Netherlands, Leiden
Nigon, V. France, Lyon
Nill, A. Austin, Texas
Nilsson, B. Sweden, Stockholm
Nishiura, J.T. Seattle, Washington
Nix, C.F. Oak Ridge, Tennessee
Noble, W.R. Raleigh, North Carolina
Nogués, R. Spain, Barcelona
Norby, S. Denmark, Copenhagen
Norton, S. Charlottesville, Virginia
Nöthel, H. Germany, Berlin
Nöthiger, R. Switzerland, Zürich
Novitski, E. Eugene, Oregon
O'Donald, P. England, Cambridge
Oftedal, P. Norway, Oslo
Ogaki, M. Japan, Sakai
Ogita, Z. Japan, Osaka
Oguma, Y. Japan, Tokyo
Ohanessian, A. France, Gif-sur-Yvette
Ohba, S. Japan, Tokyo
Ohki, K. Japan, Nagasaki
Ohnishi, O. Madison, Wisconsin
Ohta, T. Japan, Misima
Oishi, K. Japan, Misima
Ojeda, A.A. Colombia, Bogotá
Okada, T. Japan, Tokyo
Oksala, T.A. Finland, Turku
Okubo, K. Japan, Nagasaki
Olive, M. Houston, Texas
Oliver, D. Iowa City, Iowa
Oliver, D. Austin, Texas
Olivieri, G. Italy, Rome
Olthoff, H.M. Netherlands, Haren
Olvera, O. Mexico, Mexico City
Ondřej, M. Czechoslovakia, Prague
Ortiz, E. Spain, Madrid
Oshima, C. Japan, Misima
Outwater, T.W. Chapel Hill, North Carolina
Ouweneel, W.J. Netherlands, Utrecht
Paez, D. Colombia, Bogotá
Paik, S.K. Korea, Seoul
Paik, Y.K. Honolulu, Hawaii
Paika, I.J. Lincoln, Nebraska
Pak, W.L. Lafayette, Indiana
Pakonen, C.Z. Pullman, Washington
Pal, T. India, Calcutta
Pankow, W. Switzerland, Zürich
Parisi, G. Italy, Naples
Park, E.H. Korea, Seoul
Park, M.S. Korea, Kwangju
Parker, D.R. Riverside, California
Parker, M. Houston, Texas
Parry, D. Seattle, Washington
Parshad, R. India, Chandigarh
Parsons, P.A. Australia, Bundoora
Parzen, S.D. Madison, Wisconsin
Pasteur, G.D. Dallas, Texas
Pasteur, N. Dallas, Texas
Patricolo, M.R. Italy, Naples
Patrouli, H. Greece, Athens
Patty, R.A. Lincoln, Nebraska
Pavlich, L. Flagstaff, Arizona
Pavlovsky, O. Davis, California
Paxman, G.J. England, Lancaster
Paz, C. Argentina, Buenos Aires
Peacock, W.J. Australia, Canberra
Peeding, N. Durham, North Carolina
Peers, E. New York, New York
Pelecanos, M. Greece, Patras
Perdrix-Gillot, S. France, Lyon
Perekovic, V. Canada, Edmonton
Pereyra, E. Argentina, Buenos Aires
Perkins, J.M. England, Birmingham
Perreault, W.J. Ann Arbor, Michigan
Peterson, K. Northridge, California
Petković, D. Yugoslavia, Belgrade
Pex, A.M. Netherlands, Leiden
Picard, G. France, Clermont-Ferrand
Piekielniak, M. Utica, New York
Pierre, A.M. France, Gif-sur-Yvette
Pinsker, W. Austria, Vienna
Pipkin, S.B. Washington, D.C.
Phelps, C.G. Chapel Hill, North Carolina
Phillips, G. Austin, Texas
Phillips, J. Austin, Texas
Plagens, U. Madison, Wisconsin
Plaut, W.S. Madison, Wisconsin
Plus, N. France, St. Christol les Alès
Poirier, M. Durham, North Carolina
Polan, M.L. New Haven, Connecticut
Pomato, N.J. Notre Dame, Indiana
Poodry, C. Canada, Vancouver
Pospisil, Z. Eugene, Oregon
Postlethwait, J.H. Eugene, Oregon
Porter, H.N. Austin, Texas
Portin, P. Finland, Turku
Posch, N.A. Pasadena, California
Poulson, D.F. New Haven, Connecticut

Powell, J.R. New York, New York
Powers, L.M. Washington, D.C.
Prakash, S. Rochester, New York
Prendergast, L. San Diego, California
Prevosti, A. Spain, Barcelona
Prins, F.W. Netherlands, Haren
Printz, P. France, Gif-sur-Yvette
Procunier, D. Canada, Calgary
Pronk, P. Netherlands, Haren
Prout, T. Riverside, California
Prudhommeau, C. Netherlands, Leiden
Pulliam, R. Chicago, Illinois
Puro, J. Finland, Turku
Pyati, J. Boulder, Colorado
Querubin, M.A. Brazil, São Paulo
Räber, E. Switzerland, Zürich
Rahman, R. India, Calcutta
Rahman, S.M.Z. India, Bhagalpur
Raibley, D.W. Carbondale, Illinois
Raichaudhuri, A. India, Calcutta
Raikow, R. Honolulu, Hawaii
Rajaraman, R. Canada, Halifax
Ramel, C. Sweden, Stockholm
Ramila, D. Brazil, Pôrto Alegre
Rankin, S. Bellingham, Washington
Rathie, K.A. Australia, Sydney
Ratty, F.J. San Diego, California
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