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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 $ac^a w$
9 amx/FM3, y^{31d} sc^8 dm B l
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 $B_3 Bx^r$ car/y f:=
* B_i 42
* $B^i B^i$ 45
* BB_{36b} 43
* BB^{36b} 44
14 Bg B/$In(1)AM_2^6$
15 bi ct^6 g^2
16 bo v
17 br
18 $br_3 w^e$ ec rb t^4/FM1, y^{31d} sc^8 w^a lz^s B
* br 664
19 Bx_2
20 Bx_3
21 Bx_J
22 Bx^r
* Bx_{r49k} 13
* Bx^{r49k} 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_K . . . . . 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^1/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_3/y f:=
42 f B^3/y f:=
43 f BB_{36b}/y f:=
44 f BB^{36b}/y f:=
45 f $B^i B^i$/y f:=
46 $f_3 fu$/y f:=
* f_5 167
* f_{36a} 157
47 f^{B15}
* f_{B27} 774
* f^{B27} 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_2w^{a}
58 Hk
* Hw^{49c} . . . . . 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}
70 lz^{50e}/y f:=
* lz^{BS} 766
71 lz^{D} lz^{46} lz^{g} lz^{s} ras^{4} v/y f:=
* lz^{g} 667
* lz^{K} . . . . . 10, 180, etc.
* lz^{s} . . . . . 18, 27, etc.
* lz^{y4} 120, 766
72 m_2
* m^{D} 667,733
73 $m^{}$/FM3, y^{31d} sc^{8} dm B l
74 M(1)n/FM6, y^{31d} sc^{8} dm B
75 $M(1)o^{Sp}$ f/FM6, y^{31d} sc^{8} dm B
76 $M(1)o^{Sp}$/FM6, y^{31d} sc^{8} dm B
* M(1)Sp . . . . . (see $M(1)o^{Sp}$)
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^{3}/In(1)ClB
* 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^{9}/y f:=
88 r^{39k} f B/In(1)AM
89 ras_2dy
90 ras^{3}
91 ras^{4} m
* ras_v 70
* ras 845
92 rb
93 rb cx
94 rg
95 rst^{2}/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_3 cv_2 ct^{6} v_2 g^{2} f/FM3, y^{31d} sc^{8} dm B l
105 sc pn g^{2} Bx ... (g^{2} reverted)
(sc reverted)
106 sc z ec ct^{6}
107 sc z swb^{62b} (Ives)
108 sc_2z 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
* sc^{10-1} . . . . . (see ac^{3})
114 sc^{19}/y Hw
* sc^{D2} 838
* sc^{J1} 182
* sc^{J4} 62
* sc^{S1} 769
* sc^{S2} 178
* sc^{260-14} 837
* sc^{260-15} 806
* sc^{260-22} 847
* $sc^{}$ 807

* sc^{z} 846
115 scp t
116 sd$_{5}$ ^{58d14}/y f:=
117 Sh^{2}
118 shf^{2}
* sl$_{1}$ . . . . . (in C1B, C1B^{36d})
* sl^{2} 723
* sn$_{2}$ 713
* sn^{2} 161
119 sn^{3}
120 sn^{3} $_{4}$ lz^{y4} v/y f:=
121 sn$_{34e}$
122 sn^{36a}
123 sn^{36a}/y f:=
* 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} v$_{a}$ (bw)
127 su(s)^{2} w^{a} cv t
128 su(s)^{3} cv v f/FMA3, y^{2} (bw)
129 su(s)^{S} v/FMA3, y^{2} (bw)
* su(w^{a})$_{S2}$ . . (= su-w^{a})$_{S}$ . . 700, 708, etc.
* su^{S}-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^{2} v f
138 t^{3}
t$_{4}$
* t$_{5}$ . . ^{12}. 18
139 t^{t} v r
* tuh-1 . . $_{3}$ ^{31d}(= tu-h) . . 673
140 tw/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
* ty 54
* tyl . . $_{2}$ (= ty-1) ^{4} . . . 779, 780
141 un$_{4}$ Bx^{2}/In(1)AM, ptg
142 un^{4}
143 v
144 v f Bx^{r49k} car/y f:=
145 v$_{2}$f su(f)
146 v^{2} ^{36f}fw
147 v^{Of}
* v 781
148 vb
* vb^{2} 135
149 vs
150 w
151 w m f
152 w sn^{3} $_{11E4}$ m
* w^{17G2} 199
* w 108
153 w^{a}
154 w$_{a2}$
155 w$_{a3}$
156 w$_{a4}$
157 w$_{bf}$ f^{5}
158 w$_{bf2}$
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$_{t}$ fw
170 w$_{u}$
171 w
172 wy$_{2}$
* wy^{2} 192
173 y
174 y ac$_{K}$v
175 y ct^{6} (bw)
176 y pn
177 y pn w cm ct^{6} sn^{3} oc ras^{2} v dy 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^{S1} ras^{2} v dy g^{2} f os^{o}
car sw/In(1)sc^{S1}, In(1)d1-49, y v B
179 y sc
180 y sc$_{5}$ lz^{g} v f/y f:=
181 y sc^{D2}
182 y sc
183 y v f mal^{bz}
184 y w Co/y f:=
185 y$_{2}$w spl
186 y^{2}
187 y^{2} cho^{2}
188 y^{2}cv v f
189 y$_{2}$ sc w^{a} ec
190 y$_{2}$ w$_{a}$
191 y$_{2}$ w^{2}w
192 y^{2} wy^{2} g^{2} (g^{2} partly reverted)

193 y^{2S}
194 y^{2S} fw^{34e}
195 y^{3d}/y^{3P} f:=
* y^{4} 812
* y^{31d} 814
* y^{34c} . . . . . 9, 18, etc.
196 y^{59b}
* y^{td} 709
197 y^{v2}
198 y^{11E4}
199 z w

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^{lvI} 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^{blt} 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^{lvI} 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-1)
217 Alu
218 an/SM5, al^{2} Cy lt^{v} sp^{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^{blt}
224 arch ch1/SM5, al^{2} Cy lt^{v} sp^{2}
225 ast^{2} ho cl
* ast^{3} 815
226 ast^{4} dp cl
* ast^{X} 300
227 Ata 868
228 b
229 b tyr-1
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^{blt} 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^{blt})
247 bri
* bs^{2} 380
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} Cy lt^{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 1(@)crc/SM5, al 2 Cy 1t 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 1t v sp 2
276 d b/SM5, al 2 Cy 1t v sp 2
277 da/SM1, al 2 Cy sp 2
278 dil 2 hv bw sp/SM5, al 2 Cy 1t v sp 2
279 dk e c
280 dp
281 dp 2 cn bw
* dp D . . . . . (see dp lv2)
* dp lv 876
282 dp lv2 b/SM5, al 2 Cy 1t v sp 2
* dp lvI . . . . . 292, 293, etc.
* dp Nov 204
* dp o . . . . . (see dp ovN)
283 dp o2
284 dp olvR /SM5, al 2 Cy 1t v sp 2
285 dp ovN
286 dp Rf
* dp Th . . . . . (see dp olvR)
* dp tx . . . . . (see dp lvI)
* dp v . . . . . (see dp lv)
* dp v2 690
287 dp v1
* dp vM . . . . . (see dp vM)
288 dp vM /SM5, al 2 Cy 1t v sp 2
289 ds dp
290 ds rv ft dp v2 l(2)M b pr/SM5, al 2 Cy 1t v sp 2
291 ds S 2 G b pr/In(2L+2R)Cy, al 2 Cy 1t 3 L 4 sp
292 ds w /In(2L)Cy L t R , Su(S) dp lv2 pr 33k
* ds 38k . . . . . 328, 353, etc.
293 ds /In(2L)Cy, Cy dp lv2 b pr
294 dsr
295 dw-24F cl/SM5, al 2 Cy 1t v sp 2
296 dw-24F l(2)cg, cg/SM5, al 2 Cy 1t 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 1t v sp 2
302 fj wt/SM5, al 2 Cy 1t v sp 2
303 fr/In(2L+2R)Cy, Cy dp lv2
304 fr 2 wt/SM5, al 2 Cy 1t v sp 2
305 Frd/In(2L+2R)Cy, Cy sp 2
* fs 2.1 . . . . . (see fs(2)El)
306 fs(2)B Alu 1t/SM5, al 2 Cy 1t v sp 2
* fs(2)El 249
307 ft
* G rv 291
308 G rv /SM5, al 2 Cy 1t v sp 2
* Go 232, 233
* gp 234
* gt-4 416
* Hia 439, 440
309 hk
310 hk 2 pr
* hk 205
311 ho
312 hv/SM5, al 2 Cy 1t v sp 2
313 Hx/ see also 215
314 hy/SM5, al 2 Cy 1t v sp 2
315 hy a px sp/SM1, al 2 Cy sp 2
* ix 2 374
* ix 2 204
316 j
317 J/In(2L)NS
318 J 34e
319 kn
320 L 2
321 L 4
322 L 5
323 L G
324 L r
325 L si
326 L
* l(2)301 367
327 l(2)39 a 3 px slt sp/SM5, al 2 Cy 1t v sp 2 V1 33k
328 l(2)a bs , In(2L)t/bw , ds
* l(2)ax 210
329 l(2)ay b c sp 2 /SM5, al 2 Cy 1t v sp 2
* l(2)Bld 236
* l(2)bw 262
* l(2)C 399
* l(2)cg 296
* l(2)crc 270
330 l(2)gl a px or/SM5, al 2 Cy 1t v sp 2
331 l(2)H L 2 /SM5, al 2 Cy 1t v sp 2
* l(2)M 290
332 l(2)mat/SM5, al 2 Cy 1t v sp 2
333 l(2)me/SM1, al 2 Cy sp 2
* l(2)mr 2 738
* l(2)R 411

* 1(2)Su(H) 301, 426
* ll_2 363
334 ll^2
335 lm/In(2L+2R)Cy, Cy S^2 dp^{lv2} E(S)
336 lt/T(Y;2)A
337 lt std/SM2, al^2 Cy lt^v sp^2
338 lt_3 stw^3
* lt^3 . . . . . 291, 864, 888
* lt^v . . . . . 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, $In(2R)bw^v$
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 $M(2)S2^3$/SM5, al^2 Cy lt^v sp^2
* M(2)S3 . . . . . (see $M(2)S2^{S5}_3$)
* M(2)S5 . . . . . (see $M(2)H_{S6}$)
* M(2)S6 . . . . . (see $M(2)m_3$)
348 M(2)S7/SM5, al^2 Cy lt^v sp^2
* M(2)S9 . . . . . (see $M(2)S2^9_S$)
* 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^{45a} 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 1(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}_{V32g})
* Pm^{V32g} . . . . . (see bw^{V1})
370 po_2 vg
371 po^2
372 pr
373 pr cn/T(Y;2)C
374 pr cn ix/SM5, al^2 Cy lt^v sp^2
375 pr^{bw}
376 pu_{Gr}
* Pu^{Gr} 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^R 335, 771
397 S_X/bw^{V1}, ds^{33k}
* S^X 300
398 sca
399 sca 1(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, $l(2)R_v$ sp^2
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-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_M Cy lt^v sp^2
440 vg_U Hia/T(2;3)S^M In(2L+2R)Cy, Cy
441 vg /In(2L)t, 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_G 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(3L+3R)P, Dfd ca
486 Dl_{12}/In(3L+3R)P, Dfd ca
487 Dl_{13}/In(3R)C, Sb e l(3)e
488 Dl_{14}/In(3R)Cyd, Cyd
* Dl^{B} 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
* $e(dp)^{v}$ 690
494 e_{4}^{wo} ro
495 e^{s}
496 e^{s}
497 e^{s} ca^{nd}/TM6, ss $bx^{34e}\ Ubx^{P15}$ e
498 eg_{2}/In(3LR)Cx
499 eg^{2}/In(3LR)Cx
* er 684, 693
500 eyg
* fl 541
501 fz
502 gl_{2}
503 $gl_{3}^{2}\ e^{4}$
504 gl^{60j}
505 gl
506 Gl Sb/LVM
* gm 559, 623
507 gro
508 gs
509 h_{2}
510 h^{2}
511 H/In(3R)P
512 $H_{2}Pr$/In(3R)C, e
513 H_{3}^{Xa}/T(2;3)ap
514 H_{57c}/In(3R)C, Sb e l(3)e
* H 620
* Hm 878
* Hn_{r} 879
* Hn^{r} 518
515 Hn^{r3} 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 $l(3)tr\ Sb/In(3L)P,\ In(3R)P18,\ Me\ Ubx\ e^{4}$
525 $l(3)tr\ Ubx/TM1,\ Me\ ri\ sbd^{1}$
* 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)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_{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, 1(3)a
567 ro$_{2}$ra ca/T(2;3)Me
568 rs
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^{Xa}
581 Sb Ubx/T(2;3)ap^{130}
582 Sb^{63b}/In(3LR)Ubx^{130}, Ubx^{130} e^{s}
583 Sb^{Spi}_{V}/In(3LR)Cx
* Sb 885, 886
* sbd_{2} 603
584 sbd^{2}_{105}
* sbd^{1} 757
* sbd^{1} 456, 525
585 se
586 se h
587 se rt^{2} th/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 1(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} 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 $su(Hw)^{2}_{2}$ 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 844
615 Tri/In(3LR)DcxF
616 tt wo
617 Tu (= Tubby)
* tu-36e . . (= tu^{36e}) . . 447
* tuh-3 673
618 tx
619 Ubx^{4}_{61d}/In(3L+3R)P, Dfd ca
620 Ubx_{101}/H^{57c}
* Ubx_{130} 579
* Ubx . . . . . 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 1(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^{R}
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^{2}
645 ey^{4}
* ey^{D} 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$_{2}$/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^{Cat}/ci^{D}
659 spa^{pol}
660 spa^{p65}
661 sv^{35a}
662 sv^{de}/ey^{D}
663 sv^{n}

Multichromosomal Stocks

664 br^{3} dxst;ed Su(dx)^{2}(1;2)
665 dxst;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 y ac w^{ch} fa/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}/FMA3, y^{2};Dp(2;3)P/TM6, ss^{-} bx^{34e} Ubx^{P15} e(1;3)
676 y;mwh(1;3)
677 y^{2} su(Cbx) v/FMA3, y^{2};Cbx/T(2;3)ap^{Xa} bx^{34e}(1;3)
678 y^{2} e(bx)^{2} w^{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};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)
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 712
700 y^{2} 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}/gv1 sv^{n}
707 C(4)P2. ci ey^{R}/gv1 sv^{n}

Attached-XY

708 v f B, $\overline{XY}/y^{2}$ su(w^{a}) w^{a} bb
709 y^{59b} 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^{R}(1)d1-49, Y^{S} y·Y^{L}/ y X·Y;bw;e;ci ey

Triploid

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

Extra-Y

713 In(1)w^{m4L} $N^{264-84R}$, y sn/FM3, y^{31d} sc^{8} dm B 1/Y ♀;dm sn ♂ (DIS 28: 137)
714 y v f mal/mal^{6} Y ♀;In(1)d1-49, B^{M1}, Df (1)mal^{6}, y v sn^{+X2}/mal^{+} Y ♂
715 y v f mal/y^{+} mal^{+} Y ♀;1(T2-4a)/y^{+} mal^{+} Y ♂
* Y^{-bb} 786
716 In(X^{c2})w^{vc}/In(1)d1-49, y w lz^{s} ♀;In(1)d d1-49, y w lz^{s}/sc^{8}·Y ♂
717 X^{c1}, y/y f:=/y^{+} Y
718 X^{c2}, cv v f/C1B, 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)260-1	Df(1)260-1/FM4, y^{31d} sc^{8} dm B
722	Df(1)B^{263-20}	Df(1)B^{263-20}/In(1)sc, In(1)AM, sc^{7} 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^{268-42}	 738
725	Df(1)ct^{1}	Df(1)ct^{1}, y/FM4, y^{31d} sc^{8} dm B
726	Df(1)g^{259-4c}	Df(1)g^{259-4c}, f B/In(1)AM
727	Df(1)m	Df(1)m/FM4, y^{31d} sc^{8} dm B
728	Df(1)mal	Df(1)mal/In(1)d1-49, lz^{s}
729	Df(1)N^{8}	Df(1)N^{8}/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
730	Df(1)N^{264-39}	Df(1)N^{264-39} w^{ch}/FM4, y^{31d} sc^{8} dm B
731	Df(1)$N^{264-105}$	Df(1)$N^{264-105}$/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
*	Df(1)rst^{2}	 95
*	Df(1)sc^{4L} sc^{8R}	 795
*	Df(1)sc^{8}	 769
732	Df(1)syr^{258-11}	Df(1)syr^{258-11}, Dp(1;f)101, sp1/y f:=
733	Df(1)w^{258-42}	Df(1)w^{258-42}, y/In(1)d1-49, y Hw m^{2} g^{4}
734	Df(1)w^{258-45}	Df(1)w^{258-45}, y/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
735	Df(1)w^{258-48}	Df(1)w^{258-48}, y/FM4, y^{5} sc^{8} dm^{vco} B
736	Df(1)w	Df(1)w, y sc sp1;Dp(1;3)w ;y f:=

Deficiencies-Y

737 Df(Y)Y^{bb-} — Df(Y)Y^{bb-}, y^2 eq w^e bb^1/w^e bb^1;Y^{st}

738 Df(Y)Y^{st} — and w^e bb^1;Y^+;In(2L+2R)NS, px sp/l(2)mr^2

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^{VDe2L} Cy^R — Df(2R)bw^{VDe2L}, sp^2/T(2;3)ap^{Xa}_R

750 Df(2R)bw_2 — Df(2R)bw_2, In(2R)Cy^R/Gla

751 Df(2R)Px^2_B — Df(2R)Px^2_B, bw sp/SM1, al^2 Cy_2 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_C/SM5, al^2 Cy lt^v sp^2

* Df(2R)vg — . . . . . (= 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^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, Df(0+ac)·w^a sc^8·

760 Dp(1;f)107 — Dp(1;f)107;In(1)sc^8, Df(0+ac)·w^a sc^8·

761 Dp(1;f)118 — Dp(1;f)118;In(1)sc^8, Df(0+ac)·w^a sc^8·

762 Dp(1;f)135 — Dp(1;f)135, y^2;In(1)sc^8, Df(0+ac)·w^a sc^8·

763 Dp(1;f)R — Dp(1;f)R/y dor^1/y dor

* Dp(1;f)X^{c2}_9 — . . . . . (see Dp(1;f)R)

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

765 Dp(1;1)112 — Dp(1;1)112, y_f (homozygous stock)

766 Dp(1;1)lz — Dp(1;1)lz, lz^{50e} 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$^{J4}_{vco}$ — Dp(1;3)sc^{J4}/Df(1)sc^{8}, w^{a}
* Dp(1;3)w — 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$^{M1}_{M2}$ — In(1)B$^{M1}_{M2}$, v B$^{M1}_{B15}$ (tan-like); . . see also . . 784, 785, etc.
774 In(1)B^{M2} — In(1)B^{M2}(rv) f (reinv.; mosaic)
775 In(1)B^{M2} — In(1)B$^{M2}_{B27}$, f B^{M2}/ClB
776 In(1)B^{M2} — In(1)B^{M2} v B^{M2}
* In(1)bb — 723, 724
777 In(1)ClB^{36d} — In(1)Cl, sc t^{2} v sl B (= 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, tyl
780 In(1)dl-49 — In(1)dl-49, tyl bb^{1}/y v f car
781 In(1)dl-49 — In(1)dl-49, v^{Of} f
782 In(1)dl-49 — In(1)dl-49, y fa^{n}
* In(1)dl-49 — In(1)dl-49, y Hw m^{2} g^{4} 733, 798
783 In(1)dl-49 — In(1)dl-49, y Su(Hw) Hw m^{2} g^{4}/y w f;(nub/+)
784 Ins(1)dl-49, B^{M1} — In(1)dl-49, In(1)B^{M1}, l(1)J1 sc^{J1} oc ptg B^{M1}/In(1)sc$^{S1L}_{S1}$ sc$^{8R}_{8}$, y sc^{6} sc^{3} pn w ec rb cm ct^{2} sn^{2} ras^{2} g^{2} f os^{s} os^{o} car l/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
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 l (= 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) 128, 129
791 In(1)N$^{264-84}_{3}$ — 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^{4} — 809, 813
794 In(1)sc^{4} — In(1)sc^{4}, y sc
795 In(1)sc$^{4L}_{7}$ sc$^{8R}_{7}$ — In(1)sc$^{4L}_{7}$ sc$^{8R}_{7}$, 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(1)dl-49, y Hw$_8$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} — In(1)sc^{8}, sc^{8}

802 In(1)sc^{8} — In(1)sc^{8}, sc^{8} cv y f/y f:=

803 In(1)sc^{8} — In(1)sc^{8}, y sc^{8} w^{a}

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

* In(1)sc^{9} — 700, 784

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

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

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

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

* Ins(1)sc^{S1}, dl-49 — In(1)sc^{S1}, In(1)dl-49, y v B 178

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

809 Ins(1)sc^{S1L}, S, sc^{8R} — In(1)sc^{S1L}, In(1)S, In(1)sc^{8R}, sc^{S1} sc^{8} w^{a} B (= Muller-5)

* In(1)sc^{S1R} — 813

810 In(1)w^{m4} — In(1)w^{m4} (bb?)

811 In(1)w^{m4} — In(1)w^{m4}, y cv m f/y f:=

812 In(1)y^{3P} — In(1)y^{3P}, y B (B reverted)

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

814 In(1)y^{4} — In(1)y^{4}, y

2L Inversions

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

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

* In(2L)Cy t^{R} — In(2L)Cy 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^{V1}, ds

* 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} sp^{2} or^{45a} 241

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

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

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

* In(2L+2R)Cy, Cy E(S) . . . . 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) 771

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

* Ins(2L+2R)Cy, bw^{V34k} — In(2L+2R)Cy, Cy In(2R)bw^{V34k} 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^2	738
*	Ins(2L)t, (2R)Cy	In(2L)t, Roi In(2R)Cy, bw sp^2 or (= Roi)	441

2LR Inversions

821	$In(2LR)lO2_{V1}$	In(2LR)lO2, ds^W sp^2/SM1, al^2 Cy sp^2	
*	$In(2LR)bw^{V32g}$	In(2LR)bw^{V1}, ds^{33k}	328, 353, etc.
*	In(2LR)dp	. . . . .	352, 739
*	In(2LR)Gla	. . . . . (= Gla)	(see T(2;3)dp)
*	$In(2LR)Pm_2$	. . . . .	232
*	$In(2LR)Pm^2$	. . . . .	(see In(2LR)bw^{V1}_{V32g})
*	$In(2LR)Rev_B$	. . . . . (= Rev)	(see In(2LR)bw^{V32g})
*	In(2LR)Rvd	. . . . .	823
*	In(2LR)SM1	In(2LR)SM1, al^2_2 Cy sp^2_v . . . . (= SM1)	(see In(2LR)Rev^B)
*	In(2LR)SM5	In(2LR)SM5, al^2 Cy lt^{sp^2} . . . . (= SM5)	212, 216, etc.
*	In(2LR)U	. . . . . (= U)	206 210, etc.
			258

2R Inversions

*	$In(2R)bw^{V34k}_{VDe1}$	. . . . .	342
822	$In(2R)bw^{VDe2}$	In(2R)bw^{VDe1}_{VDe2}, b/b lt 1 cn mi sp	
823	In(2R)bw	In(2R)/In(2LR)Rev 1	
*	$In(2R)Cy_K$	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 e^4	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}	 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)$P18^{Fla}$	. . . . . (= In(3R)Pasadena 18) 524
830	In(3R)P^{Fla}_{W}	In(3R)P^{Fla}_{W} (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^{257-15}, Bld/ClB (carries In(2R)Cy)
834	T(1;2)f	T(1;2)f/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^{19}	T(1;2)sc^{19}/y f:=;fs(2)B sc^{19i} 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 l
849 T(1;3)sta T(1;3)sta/y f:=
850 T(1;3)v$_{vco}$ T(1;3)v$_{vco}$, v/FM6, y^{31d} sc^{8} dm B
851 T(1;3)w^{36d} T(1;3)w^{36d}, v f/C1B

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$_{m258-18}$(1;3)sc^{J4} T(1;4)w$_{m258-18}$;T(1;3)sc$^{J4}_{D}$(C1B)
857 T(1;4)w$^{m258-21}$ T(1;4)w$^{m258-21}$, 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$^{m258-21}$, y w^{a}/FM4, y^{31d} sc^{8} dm B
* T(1;4)w . . . . . (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^{3}
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$_{Xa}$ 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$_{Xa}$/1(3)XaR
868 T(2;3)Ata T(2;3)Ata, Ata/In(2R)Mo^{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$^{2}_{v}$
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 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} . . . . ^{D} . . ^{D} . . . . ^{2} 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^{4} . . . . ^{4} . . ^{4} . . . . . (see T(2;3)Pu^{Gr})

880 T(2;3)Pu^{4} T(2;3)Pu^{4}, Pu^{4}/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)$Dp\text{-}S_{L}$ T(2;3)Dp-S, ho/In(2L+2R)Cy, Cy E(S) (hom. viable)

884 T(2;3)S_{L}^{M} T(2;3)S^{M}/In(2L+2R)Cy, Cy E(S)

* T(2;3)S^{V} . . . . ^{V} . . ^{V} . . . . $^{+}$ . . $^{+}$ . . . . ^{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 T(2;3)Sb^{2}, Sb, 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 T(3)bxd^{100}_{107} T(3)bxd^{100}_{107}, ri/T(2;3)Me

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

903 T(3)Vno T(3)Vno/H^{2}

WASHINGTON, D.C.: THE CATHOLIC UNIVERSITY OF AMERICA
Department of Biology

Chromosome 1 lz^{59}/M-5
lz^{631}/M-5

SWARTHMORE, PENNSYLVANIA: SWARTHMORE COLLEGE
Department of Biology

Chromosome 2 (EMS-induced dumpy mutants; see Jenkins, J.B., New Mutants, DIS 45, 1970)

$dp^{olv^{em4}}$
$dp^{olv^{em5}}$
$dp^{olv^{em6}}$
$dp^{olv^{em7}}$
$dp^{olv^{em8}}$
$dp^{olv^{em9}}$
$dp^{olv^{em10}}$
$dp^{olv^{em11}}$
$dp^{ol^{em1}}$
$dp^{ol^{em8}}$
$dp^{ol^{em9}}$
$dp^{lv^{em1}}$
$dp^{lv^{em2}}$
$dp^{lv^{em4}}$
$dp^{lv^{em5}}$
$dp^{ov^{em2}}$
$dp^{o^{em2}}$
$dp^{o^{em9}}$
$dp^{o^{em10}}$
$dp^{o^{em11}}$
$dp^{v^{em3}}$
$dp^{o^{em2}}$ $dp^{olv^{sp1}}$ /$dp^{o^{em2}}$

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}_{8L} sc^{S1R}_{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^{S1L}_{8L} sc^{8R}_{S1R} + d1-49, y^{31d} sc^{S1} sc^8 w^a B/y 1(1)$J1^{259}$ w m f/y^+Y
INX-4 In(1)sc^{S1L}_{8L} sc^{8R}_{S1R} + 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)d1-49, car sn^{X2} y

Y Derivatives

Y-1 y^+Y/In(1)d1-49, y sc^{S1} B v f/C(1)RM, y f

NEWARK, DELAWARE: UNIVERSITY OF DELAWARE
Department of Biological Sciences

Wild Stocks

Newark-2
Oregon-R
Swedish-c

Chromosome 1

B
f B^{49c} Hw^{e}/Fm1, y^{31d} sc^{8} w^{a} l z^{s} B
sc^{SI} cv v f
sc^{8} B I S w^{a} sc^{8} ("Basc")
w

w m f
w^{e} sn/Cl B
y f
y f: x y B

Chromosome 2

b
b vg
bw
cn bw
dp
vg_{np}
vg

Chromosome 3

D/Gl
e
ry
se
st

Multichromosomal

en^{S}-X;S/Cy
v;bw
bw;st
vg;e

FUKUOKA, JAPAN: KYUSHU UNIVERSITY
Faculty of Agriculture, Department of Biology

Wild Stocks

Crimea
Florida-G
Lausanne
Oregon-R4 (highly in red)
Hikone
Sevelen (highly inbred)
Wageningen

Chromosome 1

B^{3}
w
y w m f

y sc w^{a} ec/FM4, y^{31d} sc^{8} dmB (triploid)
y sc^{8}.Y/M-5
X^{c}, y/y f:=/y Y

Chromosome 2

bw
cn bw

Chromosome 3

se

Multichromosomal

w^{a} v/FM3, y^{2};tra/In(3LR)Ubx^{130}
$XY^{L}.Y^{S}$, y^{2} su-w^{a} w^{a} $Y^{L}.Y^{S}$/y/Y
XY^{L}, y cv v f car.Y^{L}/yY"
$XY^{L}.Y^{L}$, y^{2} su-w^{a} w^{a} $Y^{S}.Y^{L}$ y^{+}/y v bb/O
sc^{8}.Y/yv/y^{2}

Extrachromosomal

Oregon-N sex ratio
(with nebulosa SR spirochete)
Oregon-W sex ratio
(with willistoni SR spirochete)
Hikone-W sex ratio
(with willistoni SR spirochete)

MYSORE, INDIA: UNIVERSITY OF MYSORE
Department of Zoology

Wild Stocks

1. Oregon-K (wild)

Chromosome 1

2. w
3. y

4. y f
5. Muller-5
6. Y^{+}/yBS

Chromosome 2

7. bw
8. dp b cn bw
9. Cy Bl L_{2}

Chromosome 3

10. st

Multichromosomal

11. bw st
12. O_{1} bw st

UTRECHT, NETHERLANDS: HUBRECHT LABORATORY

Wild Stocks

1 Oregon K
2 Sevelen

Chromosome 1

3 cv f^{3N} & y f:=
4 ras dy
5 rb
6 rb cv v f & y f:=
7 sc ct v wy f car & y:=
8 sc^{SI} B In(1)S w^{a} sc^{8}
9 w cv sn
10 y w m B

Chromosome 2

11 Bl L/Cy
12 L
13 S/In(2L+2R)Cy, Cy E(S) (homozygous for K-pn)

Chromosome 3

14 Dfd/In(3LR)Cx
15 Dfd^{r-L}
16 Dfd^{r-L}-opht
17 Dfd^{r-L} ld-opht
18 e
19 ld
20 ld-opht
21 mwh e
22 mwh e ld-opht
23 se ss k e^{s} ro

Chromosome 4

24 Ce^{2}/spa^{Cat}
25 ey-opht

Multichromosomal

26 Cy/Pm;Cx, D/In(3R)Sb
27 w;ld-opht
28 w^{a};ld-opht

Deficiencies

29 $Df(1)N^{8}$/dl-49, y Hw m^{2} g^{4}

PADOVA, ITALY: UNIVERSITA DI PADOVA
Istituto di Biologia Animale 1^{A} Cattedra di Zoologia

Wild Stocks

1 Varese

Chromosome 1

2 sc ec ct v gt f
3 v
4 w_{a}
5 w_{bl}
6 w^{e}
7 y w

Chromosome 2

8 b cn vg
9 cn
10 dp cl b
11 net

Chromosome 3

12 ru b ss p^{p} st e^{s}
13 se

Inversion on 2

14 Cy sp/Pm

TEHRAN, IRAN: UNIVERSITY OF TEHRAN
Faculty of Sciences, Department of Biology

Wild Stocks

Java
Oregon-R
Tehran

Chromosome 1

B
Basc
ct
v

w_{a}
w^{wB}
w_{h}^{mm}
w^{3} sp^{6} m^{2}
y ct^{6} ras^{2} f
y sc

Chromosome 2 & 3

vg;e
vg;se

Multimutant

al dp^{2} b pr px^{v} sp/SM_{5}, al Cy^{2} lt sp^{v} s 130 s
SM_{1}, al Cy sp/In(2LR) ds w^{1} sp;Sb e/Ubx e 102
Gl Sb/LVM
Bl L^{2}/al Cy^{2} lt sp^{v}
Cy/Pm;D/Sb
Cy/Pm;H/Sb

Chromosome 2

b vg
bw

vg
cn-iso2
cn-Tehran
dp^{2}
L^{2}

Chromosome 3

e^{11}
se

Chromosome 4

spa^{pol}

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

Wild Stocks

1 Oregon K/inbred
2 Hikone R/inbred
3 Suchumi/inbred
4 Moravec/inbred
5 Obora/inbred
6 Krnov/inbred
7 Brno/inbred
8 Moskva/inbred
9 Novosibirsk/inbred
10 Litava/inbred
11 Tišnov/inbred

Chromosome 1

12 y
13 w
14 v
15 Muller-5
16 ClB/w

Chromosome 2

17 dp
18 cn
19 bw
20 cn vg
21 b cn vg
22 dp b cn bw
23 Cy/BlL

Chromosome 3

24 se
25 e
26 se e
27 rucuca

Chromosome 4

28 pol

Multichromosomal

29 w^a e
30 w e
31 b se
32 vg se
33 bw e pol

Attached - X

34 y v f

Special Stocks Thirty 2nd chromosomes extracted by 10th from three different natural populations transformed on the similar genetics background.

ARMIDALE, N.S.W., AUSTRALIA: UNIVERSITY OF NEW ENGLAND
Department of Agricultural Biology

Chromosome 1

B
f
w_a
w^a
y Hw_a
gt w^a
ClB
Muller 5

sc cv v_3 f
y ac sn^3 v

Chromosome 2

b
bs
dp
vg

Chromosome 3

e
se h
wo ro

Chromosome 4

bt
ci ey

Multichromosomal

Cy sc
e al

Attached-X

f/B $su\text{-}s^2$ v pr

SYDNEY, AUSTRALIA: UNIVERSITY OF SYDNEY
Department of Animal Husbandry

Wild Stocks

6 strains from N.S.W. and Victoria

Chromosome 1

In_3rst^3
sn^3
w_{bl}
w

y
yw
sc ec_{31d} cv ct^6 vg^2 f/FM3,
y sc^8 dm_{31d} B^1
cx^{tg} oc/FM1, y sc^8 w^a lz^S B

Chromosome 2

b j
net
sca

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}

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

Chromosome 3

19 st

Multichromosomal

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

Inversions

22 Muller-5

NAMUR, BELGIUM: FACULTÉS UNIVERSITAIRES N.D. DE LA PAIX
Medical School, Laboratory of Genetics

e, vg, bl, Su^2Hw, W, B
inbred strains: Nettlebred Oregon and Canton

Gif-sur-Yvette, France: C.N.R.S.
Centre de génétique Moléculaire

Wild Stocks

Oregon R

Chromosome 1

B
ct
cv
f_{53d}
g
m
pn
v
w_a
w_{aE}
w_e
w
y
z
rb g
$v\ m_e^2$
$w\ g^2$
y ct
y f

y w_a
y z_{11G3}
z w_{ssE4}
z w
w m f
y ct f_2
y m g
y v f
y z ct
y m_a v f
y w_a ct f
y w_a cv v f

Chromosome 2

bw
cn
dp
L^4
vg
cn b
cn bw
dp b
vg bw
b vg bw

Chromosome 3

Ble
cd
e
p
se
st
Wr
ca K-pn
cd e
p e
sc e
st se

Chromosome 4

ey^2

Multichromosomal

v;bw
y v;bw
v;se
w;se
w;st
w^a;e
bw;e
b;se
bw;st
y;bw;st

Chromosome Abberations

In(1)dl-49, f
Muller-5
ClB/w lz
y f:= z w^a
y f:= y z w^{aE}
y f:= tyl
y f:= XC^2 t
y f:= Muller-5 bb^{84} y
y v f:= w sn;Y bb$^-$
y v f:= Muller-5;Y bb$^-$
Muller-5 bb^{84}/In AM y;Y B^S
Muller-5 bb^{83}/In AM y;Y B^S
y v bb^8;sc Y B^S
Cy/Tft
Cy/Bl L
Dcx F/Dfd

FREIBURG, GERMANY: ZENTRALLABORATORIUM FÜR MUTAGENITÄTSPRÜFUNG DER DEUTSCHEN FORSCHUNGSGEMEINSCHAFT

1) Berlin wild K^{S1}_{a} ^{8}
2) sc InS B w sc

MELANOGASTER - NEW MUTANTS

Report of George Lefevre, Jr.

dor^{66g}: deep orange, Lefevre 66g. EMS-induced allele of dor. Is sufficiently fertile that homozygous stock can be maintained. Fertility of dor^{66g} females not improved by crossing to + males. Cytologically normal.

rst^{68i19}: roughest, Lefevre 68i19. 1-2.2. EMS-induced allele. Eyes slightly rough. Most flies exhibit loss of one or more vertical bristles and thoracic hairs are sparse. Female fertility poor, but not sterile. Salivaries normal. Crossover studies locate rst^{68i19} about halfway between w and spl. Combination of rst^{68i19}/rst^{68i25} (see below) virtually +.

rst^{68i25}: roughest, Lefevre 68i25. 1-2.2. EMS-induced allele. Eyes slightly and variably rough; some flies show loss of one or more vertical bristles. Females essentially sterile. Salivary chromosomes appear normal; but band 3C5-6 may be slightly thinner than in +. Crossing over between w and spl may be slightly reduced; rst^{68i25} located about halfway between w and spl.

Df(1)w^{67k30}: Deficiency (1) white, Baker 67k30. X-ray-induced short deficiency from 3C2-3C6, inclusive. Effectively male lethal, but can be covered by Dp w^{Vco}. Dies in combination with w^{m4L}-rst^{3R} (Df 3C2-3), but not with Df rst^{2}. Does not interact phenotypically nor does it show recombination with spl.

In(1)y^{65f4}: Inversion(1) yellow, Lefevre 65f4. X-ray-induced short inversion, left break separating 1B1-2 from 1B3-4 and right break being just to the left of 1C1. A weak y^{2}-like phenotype is associated with the rearrangement: bristles dark, body slightly darker than y^{2}, but easily classifiable. Fertile in both sexes.

T(1:3)w^{67k27}: Insertional translocation of X(3A5-3E8) into 3R at 86E17, Lefevre 67k27. X-ray-induced in +. The X-chromosome is further broken at 8E5, and the section from 8E5 to 3F1 is inverted. The duplication component covers w and N deficiencies up to ec without exhibiting variegation.

T(1;3)y^{67k5}: Translocation(1;3) yellow, Lefevre 67k5. Tip exchange between X, broken just to the left of or through 1B1-2, and 3R broken at 98C. Male viable and fertile. A y^{1} phenotype is associated with the translocation, but the X breakpoint clearly does not involve 1A5-6, which is generally thought to contain the y locus.

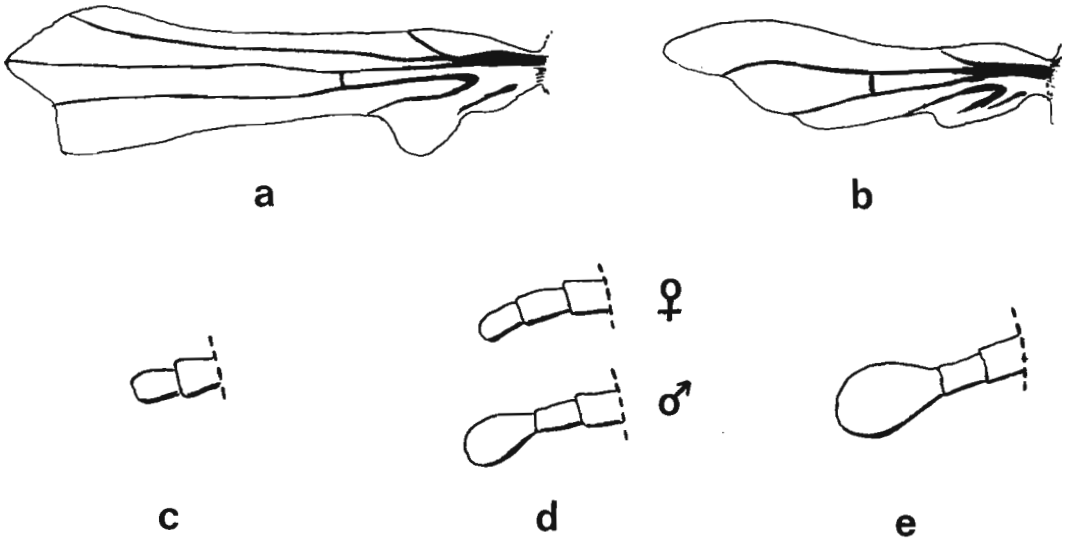
Report of James J. Colaianne and A.E. Bell

snl: sonless 1-56.1±. Spontaneous in wild-type population. A homozygous, snl/snl, female produces less than 10% male offspring regardless of the genotype of the sire. Otherwise snl/snl females are phenotypically indistinguishable from wild type. Heterozygous, snl/+, females and hemizygous, snl/Y, males are both viable and fertile and give normal progeny sex-ratios. The expression of the gene is dependent on an interaction of maternal snl/snl cytoplasm with the genotype of male offspring. The lethal action of snl occurs during the embryonic and early larval stages.

Report of J. David, M.P. Javellot and J. Touzet.

$su^{(vg)}$: suppressor (partial) of vestigial : 3-98 Spontaneous in a vestigial strain. Phenotype highly dependent on temperature and sex and expressed only in vg/vg flies.

At usual temperature (25°) the vestigial phenotype is only partially suppressed. Homozygote males ($su^{(vg)}/su^{(vg)}$; vg/vg) have intermediate wings (fig.a) as in the vestigial allele strap; in females of the same genotype, wings are shorter (fig.b). Phenotypes are best recognized, in both sexes, by the halteres, intermediate between vestigial (fig.c) and wild (fig.e), with a piece of the third segment always present (fig.d).



Phenotype of homozygotes ($su^{(vg)}$ $su^{(vg)}$;vg/vg) at 25°
a and b: wings of male (a) and female (b)
c to e: halteres of vestigial (c); vestigial suppressed (d); wild type (e).

At high temperature (28° or more) the phenotype may be entirely normal (size of wings and halteres, position of post-scutellar bristles). At low temperature (under 20°) the phenotype is very close to that of vestigial.

Viability is lowered by the suppressor. At 25°, developmental mortality is above 30%, against only 10% in +/+; vg/vg. Development is impossible under 13° and above 30°, while, in +/+; vg/vg or in wild type flies, it is still possible at 11° and 32°. The dominance of the wild suppressor allele is not complete. Heterozygotes ($su^{(vg)}$/+;vg/vg) are of vestigial phenotype at 25° but their wings are markedly increased at 28°. Viability of the heterozygotes is good.

Report of G. Periquet.

ag (2;3): Atrophie gonadique. Found in a natural population on the French Mediterranean coast. Polygenic. Extreme reduction (unilaterally or bilaterally) of ovaries and testes, correlated with abnormal abdominal metamerisation. Absence of oocytes and spermatozoids in the atrophic gonad. Germinal discs also reduced in third instar larvae. Penetrance and expressivity variable and correlated, dependent on genetic and environmental factors (for this point see also Research Notes). Chromosomes 2 and 3 are concerned with this polygenic system. Viability good. Fertility reduced in unilaterally atrophic gonad (at 25°), and nul in bilaterally atrophic gonad.

Report of T.C. Kaufman

lz^{ntg}: lozenge nitrosoguanidine. 1-27.7 Induced in Ore-R male. Eye reduced and extremely rough. Color normal and distributed evenly over entire eye. Tarsal claws apparently normal. Female sterile.

lz^{s-ntg}: lozenge spectacled nitrosoguanidine. 1-27.7 Induced in Ore-R male. Phenotype same as lz^{s}. Female sterile.

N^{ntg}: Notch nitrosoguanidine. 1-3.0 Induced in Ore-R male. Phenotype like Notch. Salivary chromosomes normal.

N^{69}: Notch 69. 1-3.0 Nitrosoguanidine induced in Ore-R male. Phenotype like Notch Salivary chromosomes normal.

N^{J}: Notch of Judd. 1-3.0 Nitrosoguanidine induced in Ore-R male. Phenotype like Notch. Salivary chromosomes normal.

N^{T}: Notch of Texas. 1-3.0 Nitrosoguanidine induced in Ore-R male. Phenotype like Notch. Salivary chromosomes normal.

N^{Co69}: Notch Confluens 69. 1-3.0 Nitrosoguanidine induced in Ore-R male. Phenotype of heterozygous females similar to N^{Co}. $N^{Co69}/w^{+}Y$ males show same phenotype as heterozygous females. Hemizygous and homozygous lethal. Salivary chromosomes normal.

pn^{69}: prune 69. 1-0.8 Nitrosoguanidine induced in Ore-R male. Eye color like pn^{2}.

r^{ntg}: rudimentary nitrosoguanidine. 1-54.5 Induced in Ore-R male. Phenotype like that of r in both males and females. Viability of male low. Homozygous females sterile to hemizygous males but weakly fertile to Ore-R males.

rb^{ntg}: ruby nitrosoguanidine. 1-7.5 Induced in Ore-R male. Eye color like ruby, ocelli colorless.

w^{crr2}: white carrot 2. 1-1.5 Nitrosoguanidine induced in Ore-R male. Eye color orange on emergence darkens to reddish brown with age. Ocelli colorless. No sexual dimorphism. Salivary chromosomes normal.

w^{ntg}: white nitrosoguanidine. 1-1.5 Induced in Ore-R male. Phenotype like w^{1}. Salivary chromosomes normal.

w^{69}: white 69. 1-1.5 Nitrosoguanidine induced in Ore-R male. Phenotype like w^{1}. Salivary chromosomes normal.

y^{ntg}: yellow nitrosoguanidine. 1-0.0 Induced in Ore-R male. Phenotype like y^{2}. y/y^{ntg} females are y^{2} in phenotype.

Report of Dr. P. Mostashfi and G. Koliantz

In Iranian natural populations of D. melanogaster, three new mutations have been discovered which are presented here.

tb: thin bristles Spontaneous recessive mutation, causing the phenotype with thin and slightly short bristles. The location is at 81.6 ± 0.5 on the second chromosome.

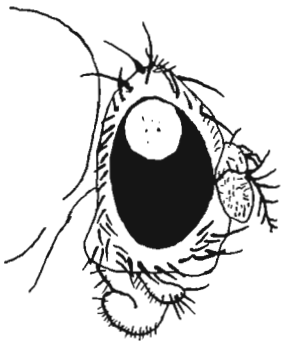
cmd: carminoid Spontaneous carmin-like recessive eye color. Its gene map is 68.2 ± 0.3 on the third chromosome.

rw: red wine Spontaneous recessive mutation, with winy eye coloration. The location is at 22.7 ± 0.6 on the third chromosome.

The pure stocks of these mutations are available. For further information write to: Dept. of Biology, Faculty of Science, the University of Tehran, Tehran, Iran.

Report of Om Parkash.

pe^{ts}: pupilla eccentrica, temperature-sensitive 3-65,0±1%. Of spontaneous origin in a thymidine-induced sex-linked-lethal culture kept at 26°C. At this temperature, the eye has the appearance as shown in the diagram. The pigment-free circular area (the pupilla) has a diameter of about 2/5 of the long axis of the eye, is more or less sharply demarcated and has some pigmented spots scattered in it. At temperature below 26°C the boundary between the pigment-free and the pigmented part gets increasingly unsharp and the size of the pupilla is reduced till at 16°C this genotype practically resembles the wild type, the expression of the gene 'pe' being completely suppressed. The eye color in the pigmented part varies from dull red to light brown, the surface of the whole eye appearing somewhat rough. Ocelli colorless. Third instar larvae (pe/pe) transferred from 26° to 16°C and kept at the lower temperature for the rest of the developmental period result in eye resembling the wild type, whereas, 0-24 hr. old pupae when transferred result in the typical 'pe' eye, indicating that it is a L/P-boundary mutant. No gross changes detected in salivary chromosomes.



Report of D.J. Fox.

$Idh\text{-}NADP^{F}$: NADP-dependent isocitrate dehydrogenase, fast form

$Idh\text{-}NADP^{S}$: NADP-dependent isocitrate dehydrogenase, slow form. These two forms of ioscitrate dehydrogenase are resolvable by agarose gel electrophoresis using the EBT buffer system of Ursprung and Leone (J. Exptl. Zool. 60: 147-154). Electrophoresis is routinely conducted for 25 minutes at 250 volts and 23 milliamps. In this system the "fast" variant ($Idh\text{-}NADP^{F}$) migrates more rapidly toward the anode than does the "slow" variant ($Idh\text{-}NADP^{S}$) as judged by the relative positions of formazan deposition. Additional bands of intermediate mobility are obtained when extracts of heterozygotes are used. Fifty of the fifty-five laboratory stocks surveyed were homozygous for the "slow" variant; the other five stocks were polymorphic. The fact that several bands are obtained when homozygotes are used may indicate the existence of isozymes. The Idh-NADP locus maps to 3-27.1±0.4.

Report of W.J. Ouweneel

art: aristatarsia Ouweneel 69e. Spontaneous in a Sevelen wild stock. Major recessive factor on the third chromosome, but considerable effect of the second chromosome. Arista replaced by a tarsus-like structure. Penetrance more than 70%, expression varying over the length of the arista. art does not interact with ss^{a}, but it strongly enhances the effect of $Antp^{B}$ (penetrance $Antp^{B}/+$ less than 1%; ntp^{B}/art more than 60%), and of $Antp^{49}$, with the latter often producing completely developed antennal legs.

hl: halteroptera Ouweneel 69g. Spontaneous in an $Antp^{B}/art$ strain. Recessive factor on the third chromosome, no effects of other chromosomes observed. Halteres replaced by often well-developed wings (sometimes half as long as the normal wings), inflated, with well-formed border bristles and wing veins. Metathorax partly replaced by a mesothoracal structure. Interaction with bx alleles.

Report of F.M. Butterworth, M. Nolph, L. Au, F. Gottschalk, N. Nadler, and G. Tuma.

ap^{69c1} : $apterous^{69c1}$ Au 69c. EMS induced in bw^D males and isolated as a compound of ap^{56f}. The stock genotype is ap^{69c1} bw^D/ In(2LR)Cy,SM5. The most salient feature of this mutant is that the wing rudiments are unlike those of the other recessive ap alleles. Although the rudiments are only 10-15% of the length of the wild-type, they possess all five hair types (see Butterworth and King, Genetics, 52: 1153-1174). In addition to the five hair types, the wing rudiments of one homozygote have costal and subcostal veins and a short portion of the radius$_1$vein. The rudiments of compounds $ap^{69c1}/ap^4$and ap^{69c1}/ ap^{56f} have all the hair types; however compounds of ap^{69c1} with ap^6, ap^{69c3}, and ap^{Xa} only have hair types 3, 5, and a few 2. The rudiments of ap^{Xa} compounds possess dome organs. The adult homozygotes are female sterile and live only a few days. Compounds of ap^{69c1} with ap^4 and $M(2)S2^4$ are also female sterile and short-lived; compounds with ap^{56f} have normal fertility and longevity.

ap^{69c2} : $apterous^{69c2}$ Gottschalk 69c. EMS induced in bw^D males and isolated as a compound of ap^{56f}. The stock genotype is $ap^{69c2}bw^D$/ In (2LR)Cy,SM5. Homozygotes are not found in the adult stage suggesting preadult lethality. Compounds of ap^{69c2}/with ap^4 and $M(2)S2^4$ are female sterile and short-lived; compounds with ap^{56f} are fertile and have normal life spans. The wing rudiments of the above compounds are short and possess type-3 hairs only.

ap^{69c3}: $apterous^{69c3}$ Nadler 69c. EMS induced in bw^D males and isolated as a compound of ap^{56f}. The most salient feature of ap^{69c3} is that the homozygotes are viable and fertile, similar to ap^{56f}. Furthermore, compounds of ap^{69c3} with ap^4 and $M(2)S2^4$ are also fertile and viable. Wing rudiments of the homozygotes are less than 10% of the normal wing and possess type-3 hairs only. The halteres are very short, the supraalar thoracic bristles are absent, and only half the normal number of the postalars are present. However, the thoracic bristle distribution and halteres of ap^{56f} homozygotes are normal. It should be noted that most of the traits (viability, female fertility, wing length, wing hair types, etc.) of stocks such as ap^4 and ap^{56f} used earlier (Butterworth and King, Genetics, 52: 1153-1174) have remained the same after about 130 generations. However, the distribution of the various thoracic bristles of ap^4 , ap^{56f}, and ap^6 homozygotes and the haltere morphology of ap^{56f} have changed markedly over that period. Consequently the system of classifying ap alleles according to thoracic bristle distribution should be used with caution.

(Supported by N.S.F. Grants GY 5834 and GB 6144)

Report of C. Beckman

sk stuck 3.80 Spontaneous in Oregon-R. A sex limited condition which causes males to have difficulties separating from females after copulation. Penetrance variable, near 100% in original isogenic stock but presently 78% in sibmated descendants of the original stock. The present mortality due to copulation is 14% of mated pairs. Affected males often show narrowing of the abdomen however this characteristic is not consistent enough to separate affected individuals from normal. Map distance approximate - obtained by pair mating techniques.

Report of Sergey Polivanov

lz^{63i} : $lozenge^{63i}$ Arose spontaneously. First was found as a single male in the egg sample taken from an experimental population. Phenotypically similar to lozenge-clawless (lz^{cl}) or lozenge (lz^{59}) and functionally allelic to the latter. But eye hairs are not completely absent and eyes are slightly larger than in lz^{59}. Males are fertile. Fertility of females is highly reduced.

Report of Ruby Valencia

Following is a list of new rearrangements obtained in my "total genetic damage" experiments to date. All were induced in mature spermatozoa by X-rays. In the 4000r series, the visible mutations carried on the chromosomes are y^2/cho^2; red sbd; ci or spa^{pol} and in the 2000r series they are y; red; ci. Some X chromosome rearrangements are carried in stocks in which the marked autosomes have been replaced by normal autosomes and some autosomal rearrangements are carried with a normal X. Many, however, are still in their original form with markers in X, 3 and 4. All are with appropriate balancers. To reduce the length of this listing, individual stock compositions are not given. Salivary breakpoints are given and the new order given only in multi-break cases. Viabilities and fertilities are indicated in parentheses (v=viable, st=sterile, f=fertile, l=lethal, sl=semi lethal, d=detrimental) and any visible effect is indicated. Salivary chromosome breakpoints were determined by Juan Valencia.

4000r series

In (1)V7-7. 3C9-10; 16C10; 18B2-3 (sl)
In (1)V7-12. 5B; 11A7 (l)
In (1)V10-7. 3A-B; 16C (l)
In (2L)V11-3. 22A; 40E (vf) black
In (3LR)V9-10. 66D; 99F (l)
In (3R)V9-8. 81-82A; 89A-B (l)
In (3LR)V10-7. 80C; 97A-B (d, male st)
In (3LR)V5-25. 69C; 92E-F (vf)
In (3LR)V12-1. 68D-E; 94E (l)
T(X;2)V9-2. 6A2-3; 38F (male st)
T(X;2)V11-2. 40-41; 20 (vf)
T(X;2)V12-1. 11E-F; 25C-D (male st)
T(X;2)V12-2. 12C9; 40-41A (male st)
T(X;3)V5-2. 13A8-9; 84E (male st)
T(2;3)V6-1. 54D-E; 55E; 70F (segment from 2 inserted in 3, order not determined) (df)
T(2;3)V8-2. 30B; 97F-98A (l)
T(2;3)V9-1. 40-41; 62F (l)
T(2;3)V9-10. 35A; 90F (l)
T(2;3)V10-7b. 56D-E; 89D (l)
T(2;3)V10-7m. 26A3 to 26F deleted and inserted at 86E, order not determined (l)
T(2;3)V11-1. 49C-D; 65F (vf)
T(2;3)V11-3-3. 54C; 62A1 (l)
T(2;3)V11-3e. 46D; 63C (l)
T(2;3)11-3g. 24A-B; 53B; 81F New Order: 21A-24A/81F-61A; 60F-53B/24B-53B/81F-100F (l)
T(2;3)V12-1-6. 55F; 62E Combined with In(2L) 21F;29B-C (l)
T(2;3)V12-1-10. 42F-43A; 81-82 (l)
T(2;3)V12-1-32. 59F; 79F (l)
T(2;3)V12-1-41. 22E; 91F (l)
T(2;3)V12-1a. 88B to 91B deleted and inserted at 55B, order not determined (l)
T(2;3)V12-1b. 31D; 85D (l)
T(2;3)V12-2-2. 44E; 50B; 80 New order: 21-44E/50B-60; 61-80/44E-50B/80-100 (d, male st)
T(2;3)V12-2d. 31F-32A; 41; 53B; 78F; 94C New order: 21-31F/78F-94C/41-53B/94C-100; 61-78F/41-32A/53B-60 (l)
T(2;3)V13-1. 35B; 96E Combined with In(3L)V13-1, 70B; 71E-F (l)
T(2;3)V13-1b. 33B; 79F (l)
T(2;3)V13-2a. 41A to 50C-D deleted and inserted at 100A-B, order not determined (l)
T(2;3)V13-2b. 60D; 96F Combined with Df(2R)49D-E to 50B (l)
T(2;3)V13-3. 40-41; 85F (l)

2000r series

In(2LR) V26. 26F; 41A (1)
In(3L)V4-14. 70B; 80B Includes a possible deficiency in 70C (1)
Df(1)V51. 19E1-2 to 3-4 (1)
Df(2)V44. 29E5-6 (1)
Df(2)V30. 41B-C (1)
Df(2)V106. 37B (1)
Df(3)V127. 71C1-2 (1)
Tp(3L)V13. 61F to 62A deleted and inserted at 64C with 61F missing (sl and s st)
T(X;2)V101. 20A(C-D?); 60F5 (male st)
T(X;2)V153. 20A3-4; 56F5 (male st)
T(X;2)V154. 12A3; 40-41 (1)
T(X;2)V161. 8B4; 40 (1)
T(X;3)V105. 12D3; 81 (male st)
T(X;4)V46. 7D10-11; 101F-102A (X is vf, 4 is l)
T(2;3)V3-2. 27C-D; 74C-D (sl) Phenotype: arched wings
T(2;3)V4-8. 65F to 79E-F deleted and inserted at 43E, order not determined (1)
T(2;3)V4-13. 58B-C; 78B (1)
T(2;3)V8. 56C-D; 64D (1)
T(2;3)V14. 40; 62C (sl)
T(2;3)V16. 60B-C; 84A Combined with In(3R)93F; 99C (d)
T(2;3)V103. 58F; 67B4 Combined with In(3R)85A; 96E (1)
T(2;3)V116. 41C; 75B (1)
T(2;4)V24. 60A-B; 102D-E (1)

Report of J.B. Jenkins

dp: dumpy 2-13±. The following dumpy mutants were induced with EMS. Unless otherwise noted, the phenotypic expression is standard for each pseudoallele mentioned (see Carlson, Genetics 44:347, 1959), and each mutant described is balanced over Cy Stw L^4.

'olv^{em4}
'olv^{em5}
'olv^{em6}
'olv^{em7}: picked up as a double mosaic with 'o^{em9}.
'olv^{em8}
'olv^{em9}
'olv^{em10}: almost completely lethal over 'ov^1.
'olv^{em11}: variable phenotype over 'ov^1 from ov to extreme olv.
'lv^{em1}: has a crinkly-wing effect over 'ov^1.
'lv^{em2}
'lv^{em4}: mild lv phenotype over 'ov^1
'lv^{em5}: has a crinkly-wing effect over 'ov^1.
'ol^{em1}
'ol^{em8}
'ol^{em9}
'ov^{em2}: very mild o and v over 'ov^1; less extreme than 'ov^1/'ov^1.
'o^{em2}
'o^{em9}: picked up as a double mosaic with 'olv^{em7}.
'o^{em10}
'o^{em11}
'v^{em3}: very mild v
'o^{em2} 'olv^{sp1}/'o^{em2}: 'olv^{sp1} arose spontaneously in 'o^{em2}/'o^{em2}.

Report of G. Lefevre Jr.

Corrected and new information regarding mutants listed in Lindsley and Grell 1968

Df(1)N^{63b} Corrected information: genetics: Deficient for N. Carries w^{63b}, a white allele resembling w^{sp} in its interactions with w^1 and w^-.

T(1;2)51b Corrected information: cytology: T(1;2)3C1-2;3D6-3E1;20A;52E. New order: 1-3C1|20A-3D6|20A-20F; 21-52E|3C2-3D6|52E-60.

T(1;2)SP10 New information: cytology: T(1;2)10D4-6;50D5-7.

T(1;3)SP38 Corrected information: cytology: T(1;3)10B10-12;85A8-12.

T(1;3)v New information: cytology: T(1;3)10A1-2;93B7-10.

T(1;3)w^{m49a} Corrected information: cytology: T(1;3)3B1-2;3E2-3;81. New order: 1-3B1|3E3-20; 61-81|3E3-3B2|81-100.

*T(1;A)pn-ec Corrected designation:

*T(1;2)w^{62g26} cytology: T(1;2)2D6-2E1;4A1-2;(40-41) position of autosomal break in 2L or 2R undetermined. New order: 1-2D6|4A2-20 21-40|(2E1-4A1)|41-60 (for example) Genetics: Deficiency segregant uncovers pn and ec; male-lethal; duplication segregant does not cover pn (which is possibly mutant rather than deficient in the X-chromosome). The w+ locus in the duplication is not variegated, even though inserted in the centric heterochromatin of chromosome 2.

Report of J.P. Hjorth

Pgm^1: phosphoglucomutase^1
Pgm^2: phosphoglucomutase^2. These two codominant alleles of a new locus Pgm were revealed by horizontal starch-gel electrophoresis on buffer extracts of single flies followed by development for phosphoglucomutase as described by Spencer et al. (Nature, 1964, vol.204 p.742). Normal Oregon wild type, for instance, showed one single fast moving band and was homozygous for Pgm^1, whereas several mutant stocks had a single slow moving band and were homozygous for Pgm^2. Hybrids had both bands and segregated fast banded, double banded and slow banded in a proportion 1:2:1, defining a PGM-controlling locus. This Pgm locus was localized at the chromosome 3, and a mapping experiment involving the Oregon wild type (Pgm^1/Pgm^1) and a th st cp stock (Pgm^2/Pgm^2) placed the Pgm locus between th and st at position 43.4. Details to be published shortly in Hereditas.

Report of J.S.F. Barker and Barbara Hollingdale

scal: scabrous-like, 2 - 11.7. Found in an abdominal bristle number selection line, derived from an outbred wild-type cage population. The eyes are rough in appearance, and slightly bulging, and there is an increase in the number of abdominal and scutellar bristles (scal/scal 45.2, +/- 34.7 bristles on the fifth abdominal sternite in females of the selection line). Wings are broad, slightly spread and curved; longtitudinal vein L II and the posterior crossvein are irregular. Semi-lethal; females almost completely sterile.

dhm: dark hairy margin, 3 - 43.2, but proximal to thread. Found in an abdominal bristle number selection line, derived from an outbred wild-type cage population. Wings appear darker than wild-type, with hairy margins and thick veins, and there is an increase in the number of abdominal and scutellar bristles (dhm/dhm 36.4, +/+ 32.1 bristles on the fifth abdominal sternite in females of the selection line). Viability and fertility are good.

LINKAGE DATA

Report of G. Lefevre, Jr.

cho cho is located to the right, not left, of ec. Its map position should be listed as 5.5+. Neither cho^1 nor cho^2 is uncovered by $Df(w\text{-}ec)^{64d}$, whose right breakpoint is adjacent to or through 3F1-2. Two crossovers have been obtained between cho^1 and ec, demonstrating unequivocally that ec is to the left of cho.

fw and ras On the standard linkage map, fw is located at 1-38.3, 2.2 crossover units from m at 1-36.1; ras at 1-32.8, 0.2 crossover units from v at 1-33.0. Results from large-scale crossover tests that included ras, v, m, and fw as markers indicate that the standard map positions of both ras and fw are incorrect. In control crosses, 255 ras-v recombinants were observed among 40,693 total progeny (0.63%); 1250 v-m recombinants among 40,693 progeny (3.07%); and 196 m-fw recombinants among 25,837 progeny (0.76%). These data may be augmented by including information from crosses that also involved recessive lethals in adjacent intervals. Overall, the following values were obtained: ras-v, 0.59% (836/140, 629); v-m, 3.14% (2,491/79,279); m-fw, 0.77% (255/33,278). All data were obtained, in 5 broods, from females that were between 4 and 17 days of age.

Through the courtesy of E.B. Lewis, the original linkage records of C.B. Bridges were obtained. For ras-v, Bridges recorded 0.8%. Data to support the standard map position of ras could not be found in the existing records. By contrast, Bridges' original records for fw indicate a value of 2.0%, or more, for the m-fw interval. Thus, the low values in the present experiments are difficult to explain; they cannot result from chromosome abnormalities because the reduction is seen only in the m-fw interval, not in the v-m nor in the ras-v intervals, which were measured simultaneously.

rst The standard linkage map locates rst at 1-1.7, 0.2 crossover units from w at 1-1.5. The original determination was made by Gruneberg (1937) using rst^2, a deficiency. It is true that rst^2 exhibits approximately 0.2% recombination with w; but at the same time rst^2 shows about 0.3% recombination with spl (1-3.0). A new EMS-induced rst allele was obtained from E.B. Lewis. This mutant shows no cytological deficiency and fails to exhibit any of the bristle anomalies associated with rst^2; further, the mutant is quite viable and fertile in the homozygous condition.

Crossover tests with Lewis's EMS-induced rst allele provided a more accurate map position for the rst locus. In crosses of $rst^{16172.646}$/ y w spl x y w spl, all daughters exhibiting recombination between w and spl were saved for progeny tests. Among 81 such females, 40 carried crossovers between w and rst, 41 between rst and spl. Thus, the rst locus should be placed on the standard map about halfway between w and spl, that is at 1-2.2. This localization is supported by studies on two additional EMS-induced rst alleles, produced in this laboratory. (See report of New Mutants.) Of interest is the fact that both of these exhibit the vt bristle syndrome of rst^2, yet neither is obviously deficient as far as can be judged from a close study of the salivary chromosomes. One is female sterile, the other not.

Report of J. Valentin

In (2LR) Rev: recombination in right arm outside inversion The cross, In (2LR) Rev/cn bw x cn bw permitted estimation of the recombination length of the right arm outside the inversion. The progeny obtained during 4 egg-laying periods of 2 days each from ♀♀ not older than 3 days at beginning of first period was:

Rev + +	584	+ cn bw	577
Rev + bw	56	+ cn +	60

There is thus 9.1% recombination between the right breakpoint of In (2LR) Rev and bw.

Report of S. Nørby.

Recombination frequency between rudimentary and forked In recent experiments recombination frequencies of 1.3 and 1.4% have been found between rudimentary (r) and forked (f). These results are in conflict with the currently accepted positions of these loci. The literature seems to support a demand for revision of the relative map positions of the genetic markers in question.

Introduction: The first reliable crossover data for r and f were published by Morgan and Bridges in 1916 (1). The summarized data of four experiments gave a crossover value of 1.4 (20 in 1456). The two loci were placed at 55.1 and 56.5 respectively, with Bar (B) at 57.0. In the same year Muller published data (2) showing a recombination frequency of 1.3% (9 in 712) between the two markers. In 1921, however, Bridges (3) moved the r-locus to 54.5. B and f were still located at 57.0 and 56.5 respectively. No experimental data are given in the paper.

The present map positions of the three loci have been held for valid since 1931. In that year Morgan, Bridges and Schultz (4) proposed the generally accepted f-B distance of 0.3 units. As the B- and r-loci were kept at their positions from 1921, the relocation of f resulted in a distance between r and f of 2.2 units. It is interesting to note, however, that in the same report it says (p.414): "The locus of uneven is less than 0.1 units to the left of that of rudimentary, which is itself provisionally put at 54.5, though this may be too far to the left".

In his description of uneven (un) Mohr (5) reports a distance between un and f of 1.2 units. Actually, a recalculation of his data gives a recombination frequency between the two loci of 1.3% (13 in 1033).

Experimental results: In three experiments involving a rudimentary allele spontaneously arisen in our laboratory in May 1968 recombination frequencies of 1.3 and 1.4% were found between r and f. One experiment also involved un, which mapped 0.1 unit to the left of rudimentary. Another, involving B, gave 0.2% crossing over between f and B.

These results, as well as the data presented in the literature cited, suggest that a revision of the relative map positions of un and r on one side and f and B on the other is needed.

Experimental data: 1. P: r/v,f females x + males. F_1-males: (N = 5128)

non crossovers		crossovers		
r: 2035	v,r: 478	r,f: 32	v,r,f: 2	
v,f: 2048	f: 496	v: 35	+: 2	

recombination frequency between r and f: 1.4% (71 in 5128).

2. P: un/r,f females x + males F_1-males: (N = 7333)

non crossovers		crossovers	
r,f: 3609	r: 51	un,r,f: 2	
un: 3623	un,f: 42	+: 6	

recombination frequency between r and f: 1.3% (93 in 7333)
recombination frequency between un and r: 0.1% (8 in 7333)

3. P: B/r,f females x + males F_1-males: (N = 8272)

non crossovers		crossovers	
r,f: 4053	r,B: 50	r,f,B: 8	
B: 4084	f: 66	+: 11	

recombination frequency between r and f: 1.4% (116 in 8272)
recombination frequency between r and B: 1.6% (135 in 8272)

Literature cited: 1. Morgan, T.H., and C.B. Bridges: "Sex-linked Inheritance in Drosophila", Carnegie Institution of Washington, publication no. 237 (1916). 2. Muller, H.J. "The Mechanism of Crossing-over III"., The American Naturalist, 50: 350-366 (1916). 3. Bridges, C.B.: "Current maps of the location of the mutant genes of D.m." Proc. Nat. Acad. Sci., 7: 127-132 (1921). 4. Morgan, T.H., C.B. Bridges and J. Schultz: "The constitution of the germinal material in relation to heredity.", Carnegie Institution Year Book, No 30, pp. 408-415 (1931). 5. Mohr, O.L.: "Contribution to the X-chromosome map in D.m.", Nyt Magazin for Naturvidenskaberne, 65: 265-274 (1927).

NEWARK, DELAWARE: UNIVERSITY OF DELAWARE
Department of Biological Sciences

D. virilis (wild)

LINCOLN, NEBRASKA: UNIVERSITY OF NEBRASKA
Department of Zoology

See DIS 43

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 4

12 cd

Chromosome 5

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

Multichromosomal

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

Tropical strains of Drosophila from the eastern Caribbean, about 3-4 years in laboratory:

D. melanogaster

Mona Is.
Guanica (Puerto Rico) xerophytic scrub and forest
Culebra Is. (off P.R.)
Algodones (off P.R.)
Ramos (off P.R.)
Blanquilla (off P.R.) xerophytic scrub
Surprise Key (off P.R.) sesuvium cover on rock
Hassel Is. (off St. Thomas)
Little Camanoe (British Virgin Islands)
Big Camanoe (British Virgin Islands)
Marina Key (British Virgin Islands)

D. simulans

Desecheo Is. (west of P.R.)
Maricao (P.R. at 3,000 feet) 4 strains
Ramos (off P.R.)
Culebra Is. (off P.R.)
Hassel Is. (off St. Thomas)
Hans Lollik (off St. Thomas)
Inner Brass Is. (off St. Thomas)
Thatch Key (British Virgin Islands)
Guana Is. (British Virgin Islands) (3 strains)
Cooper Is. (British Virgin Islands)
Tortola (coastal) (British Virgin Islands)
Tortola (Sage Mt.) (British Virgin Islands)
Virgin Gorda (British Virgin Islands)

D. nebulosa

Guanica (P.R.) xeric scrub and forest
St. Thomas (U.S. Virgin Islands)
Cooper Is. (British Virgin Islands)
Marina Key (British Virgin Islands)

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D. affinis: Bethany, Conn.; Greenwich, Conn.; Sleeping Giant, Conn.
D. algonquin: Bridgeport, Conn.; West Haven, Conn.
D. americana americana: Independence, Ohio; Western
D. americana texana: Florida
D. ananassae: Cristobal; Cairns
D. bifasciata; sex-ratio; Pavia normal
D. borealis: Kent, Conn.
D. busckii: Lankenau (Abington, Pa.)
D. duncani: New Canaan, Conn.
D. equinoxialis: Puerto Rico, normal and sex-ratio
D. flavomontana: Yampa River, Colo.
D. funebris: Rexburg, Idaho: Stockholm, Sweden; Upperville, Va.; white eye; Yucatan
D. gibberosa: South Mexico
D. hydei: Chile; New Haven, Conn.; Vera Cruz; Zurich, Switzerland; sca cn vg; pb sca cn vg; bb4
D. immigrans: DeKalb, Illinois; New Haven, Con Conn.; North Canaan, Conn.; Sharon, Conn.; Washington, Conn.
D. lacicola: Fairbanks, Minn.
D. lebanonensis
D. littoralis: Switzerland
D. melanica: St. Louis, Mo.
D. mirim
D. montana: Cottonwood Canyon, Utah
D. nebulosa: Haiti, normal and sex-ratio
D. nigrohydei
D. nigromelanica: Cold Spring Harbor, N.Y.; Marlborough, Conn.; Stafford, Conn.
D. novamexicana
D. paramelanica: Hamden, Conn.; Killingly, Conn.
D. pararubida: Port Moresby
D. paulistorum: Belem; Bucamaranga; Cantareiras; Lancetilla; Trinidad
D. persimilis: Whitney, Calif.
D. polychaeta
D. prosaltans: Belem; Chilpancingo (stellata)
D. pseudoobscura: Pinon Standard
D. repleta: Philadelphia, Pa.; Prospect, Conn.
D. robusta: Fairfield, Conn.; Hebron, Conn.; Kent, Conn.; New Canaan, Conn.
D. rubida: Cairns
D. serrata: Cairns
D. setifemur: Cairns
D. simulans: Lankenau
D. tripunctata: Bridgeport, Conn.; Fairfield, Conn.
D. virilis: Japan
D. willistoni: Barbadoes-3; Belem; Recife-3; Recife Pop. 168; ebony; pink eyes; white eyes; sex-ratio
Zaprionus vittiger: South Africa

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

Note: 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.

POUGHKEEPSIE, NEW YORK: MARIST COLLEGE
Department of Biology

D. pseudoobscura

Payson, Ariz. (3 wild 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. (5 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)
Montrose, 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

Princeton, N.J. (1 strain)
Poughkeepsie, N.Y. (3 strains)

D. affinis

Princeton, N.J. (3 strains)
Poughkeepsie, N.Y. (3 strains)

D. nigromelanica

Poughkeepsie, N.Y. (1 strain)

D. algonquin

Poughkeepsie, N.Y. (1 strain)

D. melanogaster

Princeton, N.J. (1 strain)
Poughkeepsie, N.Y. (2 strains)

BUFFALO, NEW YORK: STATE UNIVERSITY COLLEGE
Department of Biology

D. pseudoobscura
Chromosome 3

or pr (ST)
or px (AR)
or ru (TL)

Zaprionus

multistriate
tuberculatus
vittiger

LEEDS, ENGLAND: THE UNIVERSITY
Department of Zoology

D. buskii D. funebris D. obscura D. phalerata (several strains) D. subobscura

MISIMA, JAPAN: NATIONAL INSTITUTE OF GENETICS

D. ananassae

Wild Stocks

Texas
Barro Collorado, Panama 69 (low elevation)
Turrialba, Costa Rica 101 (high elevation)
Baton Rouge, Louisiana
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 (" ")

Chromosome 1

y (Hinton)
w65 49
w65 f51
w65 y49 51
w65 f65 y51
w sn y

kk
w19 kk y51
od

Chromosome 2

b
b65
ba65
bwR
ma T (Hinton)
se
Arc bwR T
Arc se
b maT
b seR
bw baR 65
cd ba65 -12
cd bwR T
cd se ba65
j b
j b seT
maT ba65
se67 ba65
bn
b bn67
cd bwR bn67
j b ma
pe

Chromosome 3

px
px2
ru
ru2
sm
bri Rf
bri Rf px
bri ru
Rf ru
M65 px
M65 px E+
M px E-
M-b
M65 ru
bri M65 ru
M-c
M-c px
pc
Snp M-c
Snp bri ru
Snp

Chromosome 4

bb67
bb2

Multichromosomal

f;cd(Hinton)
f;cd;pxT
f;cd se66
fy65;cd ba65
w ;pxT
b seR;px2
bwR;bri
bwR;ruT
j b seT;ru
ma;bri
w;bb67
seT;ru
b seT;px2s

Undetermined

pxd50
sk66
ab-a
Bn px-b bb67
Bn bb67
gp-2
tr
tr gp
round
Sv

NEDLANDS, WESTERN AUSTRALIA: UNIVERSITY OF WESTERN AUSTRALIA
Department of Zoology

Drosophila, Subgenus Sophophora, melanogaster group:
seguyi and 2 other undescribed sibling species
melanogaster
simulans

PADOVA, ITALY: UNIVERSITA DI PADOVA
Istituto di Biologia Animale 1A Cattedra di Zoologia

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

FUKUOKA, JAPAN: KYUSHU UNIVERSITY
Faculty of Agriculture, Department of Biology

D. bifasciata (normal and sex-ratio)
D. equinoxialis (normal and sex-ratio)
D. nebulosa (normal and sex-ratio)
D. paulistorum Cantareiras
D. pseudoobscura
D. robusta (normal and sex-ratio)
D. willistoni Barbados-3 (normal and sex-ratio)

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

D. ambigua - Spanish stocks
D. busckii - Spanish stocks
D. buzzati - Armentera, Spain
D. cameraria - Prat, Spain
D. funebris - Bilbao, Spain
D. hydei - Barcelona, Spain
D. immigrans - Prat, Spain
D. littoralis - Prat, Spain
D. mercatorum - Prat, Spain
D. obscura - Spanish stocks
D. phalerata - Spanish stocks
D. repleta - Barcelona, Spain
D. simulans - Spanish stocks
D. subobscura - Spanish stocks, mutant stocks
D. testácea - Bilbao, Spain
D. transversa - Prat, Spain
Megaselia scalaris - Barcelona, Spain

KALYANI, WEST BENGAL, INDIA: UNIVERSITY OF KALYANI
Faculty of Science, Department of Zoology

D. ananassae	Wild Stocks	Chromosome 2	Chromosome 3
	a^6 Calcutta	bw vs ss^a	pc

HELSINKI, FINLAND: UNIVERSITY OF HELSINKI
Department of Genetics

D. obscura (12 strains)
D. bifasciata (1 strain)
D. subobscura (12 strains)
D. funebris (17 strains)
D. hydei (2 strains)
D. phalerata (1 strain)
D. testacea (1 strain)
D. transversa (13 strains)
D. busckii (8 strains)
D. littoralis (5 strains)
Scaptomyza pallida (1 strain)

Note: All these stocks are of Finnish origin, collected from natural populations.

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

D. ananassae Mutants

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

SÃO PAULO, BRASIL: UNIVERSIDADE DE SÃO PAULO
Faculdade de Filosofia, Ciências e Letras, Departamento de Biologia Geral

See DIS 43. Correction: D. austrosaltans in the place of D. anstrosaltans

ATHENS, GREECE: COLLEGE OF AGRICULTURE
Department of Genetics

D. simulans
D. obscura
D. ambigua
D. virilis

D. subobscura

$A_{ST}, J_1 Aph^2\ Est_2\ Est_3^1, U_{ST}\ MDH^1, E_{ST}, O_{ST}\ Est_1^{23}\ Lap^0$.

$A_2\ , J_1 Aph^1\ Est_2^2\ Est_3^2, U_{1+2}\ MDH^1, O_{3+4}\ Est_1^3\ Lap^1$

$J_{ST} Aph^2\ Est_2^2\ Est_3^2, U_{ST}, E_{ST}, O_{3+4}$ ch cu $Est_1^{12}\ Lap^1$

$A_{ST}, J_1\ Aph^S\ Est_2^2\ Est_3^1, U_{ST}\ MDH^1\text{-}MDH^2,$

$J_1\ Aph^1\ Est_2^S\ , U_{ST}\ , O_{3+4}\ Est_1^2$.

cu Est_1^{23}

and several others homozygous for inversions and/or enzyme genes.

HEVERLEE-LOUVAIN, BELGIUM: THE UNIVERSITY
F.A.Janssens Memorial Laboratory for Genetics

Wild Stocks
- D. subobscura (Belgium)
- D. subobscura (Küssnacht) (Standard homozygous)
- D. virilis

TURKU, FINLAND: UNIVERSITY OF TURKU
Department of Genetics

D. simulans	Chromosome 1	Chromosome 2	Chromosome 3
wild	v	net	H^h pe
	y w		jv se st pe
			st pe

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

50 D. funebris : wild
51 D. busckii : wild
52 D. hydei : wild
53 D. simulans : v
54 D. virilis : wild
55 D. pseudoobscura A 333

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

bipectinata (1 strain)
brachynephros (2 strains)
angularis (1 strain)
nigromaculata (1 strain)
virilis (2 strains)
ezoana (2 strains)
lacertosa (1 strain)
pseudosordidula (3 strains)
sordidula (3 strains)

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

D. busckii: Chile (La Serena)
D. camaronensis: Chile (Azapa)
D. funebris: Chile (La Serena, Valdivia, Tierra del Fuego y Punta Arenas)
D. gasici: Chile (Arica), Bolivia (Cochabamba), Colombia (Bogotá)
D. gaucha: Brazil (M. Capoes, C. de Jordan and Taimbas), Argentina (Córdoba, San Luis)
Mutants - Chromosome 1. w, 2. y
D. hydei: Chile (Camarones, Azapa, Copiapó, Antofagasta and El Tabo), Bolivia (Cochabamba)
D. immigrans: Chile (El Tabo and Valdivia)
D. mercatorum: Chile (Arica and Antofagasta)
D. mesophragmatica: Bolivia (La Paz), Perú (Machu-Picchu)
D. pavani: Chile (Copiapó, Vallenar, La Serena, El Tabo, Viña del Mar, Olmué, Bellavista, Arrayán, Los Alpes, Colbún, Los Queñes, Chillán), Argentina (Mendoza)
D. simulans: Chile (Arica)
D. viracochi: Perú (Machu-Picchu), Colombia (Bogotá)
D. virilis: Chile (Santiago)

MISIMA, JAPAN: NATIONAL INSTITUTE OF GENETICS

Wild Stocks

D. pseudoobscura
ST 5 lines
AR 6 "
CH 5 "
PP 6 "

D. virilis	2 lines	D. kikkawai	1 line
D. lutea	4 "	D. simulans	1 "
D. busckii	3 "	D. rufa	1 "
D. auraria	2 "	D. nigromaculata	1 "
D. hydei	2 "	D. equinoxialis	1 "
D. immigrans	2 "		

MELBOURNE, AUSTRALIA: UNIVERSITY OF MELBOURNE
Department of Genetics

D. simulans
701 SIM + S 51

D. hydei
702 HYD + S 51

D. immigrans
703 IMM + C 60

D. funebris
704 FUN + C 51

D. serrata
705 SER + Q 59

D. persimilis
706 PER
707 Dl;or:Cy

D. pseudoobscura
708 or pr in Santa Cruz gene arrangement
709 or pr in Standard
710 or Bl L Sc pr cv (Standard)/lethal (Cuernavaca)
711 or Bl Sc ru pr cv (ST)/or L (SC)
712 or Bl L (SC)/lethal (Cueranavaca)
713 Standard, Mather California
714 Chiricahua, Mather California
715 Arrowhead, Mather California
716 gl

UMEÅ, SWEDEN: UNIVERSITY OF UMEÅ
Institute of Biology, Department of Genetics

D. littoralis D. americana D. simulans D. texana D. virilis

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

persimilis

1 Porcupine Flat
2 Quesnell
3 Sequoia

Mutant

4 Delta or Cy

Wild strains homozygous for Chromosome 3 inversions

Arrowhead (7 strains) Pinon, Calif.
Chiricahua (8 strains) Pinon, Calif.

Mutant

gl

simulans

2 wild strains from N.S.W. and Victoria

Mutants

1 y
2 v
3 st
4 p
6 stp
7 net pm (b py sd)

Other Species

nebulosa

MYSORE, INDIA: UNIVERSITY OF MYSORE
Department of Zoology

D. ananassae

Bandipur
Bangalore
Chamundi hills
Chitradurga
Chinthamani
Coimbatore
Davangere
Goa
Harihar
Hiriyur
Hassan
Holalkere
Hosadurga
Hyderabad
Jagalur
Leucern garden
Mangalore
Mallur
Mayamudi
Mercara
Molakalmur
Mysore
Nyamthi
Sringeri
Thirupathi
Tumkur
Widyanagar

D. bipectinata

Holalkere
Mysore

D. melanogaster

Amarapur
Coorg
Davanagere
Hindupur
Hosadurga
Mayamudi
Mysore
Nyamthi
Shimoga
Widyanagar
Dehradun

D. melarkotliana

Bandipur
Chamundi hills
Chidambarum
Chitradurga
Hosadurga
Karamudi
Dehradun
Leucern garden
Palibetta

D. immigrans

Mercara

D. nasuta

Chitradurga
Coorg

D. jambulina

Leucern garden
Srirangapatam

D. mysorensis

Mysore
Leucern garden
Mercara
Srirangapatam

Zapionus mysorensis

Mercara
Mysore
Leucern garden
Srirangaparam

D. serrata

Mercara

D. takahashi

Leucern garden
Krishnaraja Sagar
Srirangapatam

D. meridiana

Udupi

D. repleta

Chitradurga
Mysore
Leucern garden
Srirangapatam

D. montium

Mercara

D. rajasekari

Chitradurga
Hosadurga
Mysore
Leucern garden
Srirangapatam

TOKYO, JAPAN: TOKYO METROPOLITAN UNIVERSITY
Department of Biology

D. ananassae

Wild Stocks

		(Inversion Karyotypes) In2L:In3L:In3R
001	Texas	AA : AB : AB
002	TL_3	AB : AA : AA
00 '	TL_{3-4} .	AA : AB : AB
003	Barro Collorado, Panama 69 (low elevation)	AB : AB : AB
004	Turrialba, Costa Rico 101 (high elevation)	AB : AB : AB
005	Christobal, Panama	AA : AB : AA
006	Baton Rouge, Louisiana	AA : AA : AA
007	$2L\text{-}A^H$	AA : AA : AA
008	$2L\text{-}B^H$	BB : AB : AA
009	Hawaii	AB : AB : AA
010	D-rar	
011	D-pp	AA : AA : AA
012	L-pp	
013	Cuba	AB : AB : AB
014	Hawaii (Wh)	BB : AB : AB
015	D-Niue	AA : AA : AA
016	Panama	AB : AB : AA
017	D-Tonga	AA : AB : AA
018	Yukatan	AB : AB : AA
019	IM-1 (Madras, India)	AA:: BB : AB
020	IM-2 (")	AA : BB : AB
021	IM-4 (")	AA : BB : AA
022	IM-5 (")	AA : BB : AB
023	Port Rico	
024	L-Upolu (light)	
025	D-Upolu (dark)	AA : AA : AA
026	L-Taputimu	
027	D-Taputimu	AA : AA : AB
029	F4 (Peng-Hu Is.)	AB : AB : AB
030	F5 (")	BB : AB : AB
031	F7 (")	BB : AB : AB
032	F8 (")	AB : BB : AA
032'	Fm (")	
033	V-Majuro, Marshall Islands	AA : AA : AA
034	V-Truk, Carolina Islands	AA : AA : AA
035	Calcutta, UCC-a66	
036	College Street, Calcutta, UCC-a99	
037	Dakshineswar, UCC-a111	
038	Port Blair, Andaman, UCC-a222	

Chromosome 1

102	y_{65}(Hinton)	AB : AB : AA
103	$w_{65}{}^{49}$	AA : AB : AA
104	$w_{65}{}^{f\,51}$	
105	$w_{65}{}^{y}$	AB : AB : AA
106	$w_{65}{}^{f\,49\,y\,51}$	AB : BB : AA
107	$w\,{}^{y\,51}sn^{65}$	AB : AB : AA
108	kk_{65}	BB : AA : AA
109	$w\,{}^{y\,51}kk$	

Chromosome 2

201	Arc_{65}	BB : AA : AB
203	b^{65}	BB : AB : AA
204	$ba_R{}^{65}$	AA : AA : AA
205	bw^R	AA : AA : AB
207	ma_T(Hinton)	BB : AB : AA
208	se^T	AB : AA : AA
209	Arc b^R	
210	Arc bw^R	
211	Arc ma_T	
212	Arc se^T	
213	b ma_T	BB : AB : AA
214	b se^T	BB : AB : AB
215	$bw^R\,ba_{65}$	AA : AA : AB
217	cd ba_{65}-7	AA : AA : AA
218	cd ba_R-12	AA : AA : AA
219	cd $bw_T{}^{65}$	
221	cd $se^T\,ba^{65}$	
222	j b^T	BB : AA : AA
223	j b se_{65}	
224	$ma_T ba_{65}$	
225	$se_T ba_{65}$-1	
226	$se_T ba_{65}$-2	
227	$se_T ba_{65}$-6	
228	$se_T ba_{65}$-11	
229	$se_T ba_{65}$-12	
230	$se_T ba^{67}$-13	
231	bn^{67}	AB : BB : AA
232	b bn^{67}	BB : AB : AA
233	cd $bn_R{}^{67}$	
234	cd $bw\,bn^{67}$	

Chromosome 3

301	bri (Hinton)	AA : AA : AA

303	px_2	BB : AA : BB
304	px^{2}	AA : BB : AA
305	Rf	BB : BB : AB
306	ru_2	AA : AB : AB
307	ru^{66}	AA : AB : AB
308	sm^{66}	AA : BB : AA
310	bri Rf	
312	bri ru	AA : AA : AA
313	$Rf_{65}px$	
314	$M_{65}px$	
315	M_{65}bri px	AB : AA : AB
316	M_{65}px E^{+}	BB : AA : AB
317	M_{65}px E^{-}	
318	M-b	AA : AB : AB
319	M_{65}b ru bri	
320	$M_{65}ru$	
321	M_{65}ru bri	AB : AA : AA
322	M-c px	BB : AB : AB
324	pc	BB : AA : AA
325	M-b pc	

Chromosome 4

401	bb^{67}	

Multichromosomal

501	f;cd	
502	f;cd^{66}	
504	f y^{+};cd ba^{65}	
505	f^{Sa};se^{T}	
506	f^{Sb};se^{T}	
507	f^{Ua};se^{T}	
508	f^{Ub};se^{T}	
509	$f^{U'}$;se^{T}	
521	w^{65};px^{T}	
531	b se^{T};px^{2}	
532	ba^{65};M^{65}	
533	bw^{R};bri	AA : AA : AB
534	bw^{R};ru^{T}	
535	j b se^{T}; ru	
536	ma;bri^{T}	
537	se^{T};ru^{2s}	
538	b se^{T};px	BB : AA : AB
539	b ma;M-c	

Undetermined

701	pxd^{50}	BB : BB : AB
702	pxd (Hinton)	
703	Bn_{65}(Hinton)	
704	ms^{66}	AB : BB : AB
705	sk^{66}	AA : BB : AA
706	ab-a	BB : BB : AB
707	Bn (bb?)	BB : AA : AA

D. virilis

Wild Stocks

Tokyo
Pasadena
Texas 1801.1
15 strains from different parts of Japan

Chromosome 1

y_4
v_{62}
w^{4}
y v_{62}
y w

Chromosome 2

eb
b.
tx
si
eb b

Chromosome 3

cn_{61}
cn_{61} t^{2}

Chromosome 4

cd

Chromosome 5

st
es
pe
st es
dc es pe

Multichromosomal

w;cn(1,3)	Est-5
b;cn(2,3)	Est-6
tx;cn(2,3)	Est-7
eb;cn(2,3)	Est-8
cd;pe(4,5)	Est-9
eb;cd;pe(2,4,5)	Est-10
eb;cd;es(2,4,5)	$Est\text{-}2^{B}$ Est-1
tx;cd;pe(2,4,5)	$Est\text{-}2^{B}$ Est-3
eb b;cn;es(2,3,5)	$Est\text{-}2^{B}$ Est-4
b;tb gp;cd;pe(2,3,4,5)	$Est\text{-}2^{B}$EEst-5
	$Est\text{-}2^{B}$ Est-6
Esterase isozyme variants-Chromosome 2	$Est\text{-}2^{B}$ Est-7
	$Est\text{-}2^{B}$ Est-8
$Est\text{-}2^{A}$	$Est\text{-}2^{B}$ Est-9
$Est\text{-}2^{B}$	$Est\text{-}2^{B}$ Est-10
$Est\text{-}2^{C}$	$Est\text{-}2^{A}$ Est-5
$Est\text{-}2^{D}$	$Est\text{-}2^{A}$ Est-9
Est-1	$Est\text{-}2^{C}$ Est-6
Est-3	$Est\text{-}2^{D}$ Est-1
Est-4	$Est\text{-}2^{D}$ Est-5
	$Est\text{-}2^{D}$ Est-6
	Esterase-null

LEIDEN, THE NETHERLANDS: GENETISCH LABORATORIUM DER RIJKSUNIVERSITEIT

D. bifurca
D. buzzatii
D. eohydei
D. hydei Leiden
D. hydei Saopaulo
D. hydei Alicante
D. hydei Madeira
D. mercatorum
D. mulleri
D. neohydei
D. nigrohydei
D. obscura
D. repleta
D. simulans
D. victoria
D. virilis
Zaprionus chesquierei

STOCKHOLM, SWEDEN: UNIVERSITY OF STOCKHOLM
Institute of Genetics

D. pseudoobscura — ST 1 line — AR 2 lines — TL 1 line — (all phenot. wild)

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

D. azteca
D. pseudoobscura
D. virilis
D. hydei
D. ananassae
D. eohydei
D. neohydei
D. simulans

FREIBURG, GERMANY: BIOLOGICAL INSTITUTE OF THE UNIVERSITY

D. bifurca
D. eohydei
D. neohydei
D. fulvimacula, wild
" , y
" , y/+ *)
D. repleta
D. simulans
D. virilis

*) See: New mutants, other species, report of O. Hess

D. hydei

1 wild

Chromosome 1

2 $t^{tomato\ 2}$
3 f^{2}
4 $g\ a\ y\ m$
5 w
6 $yellow^{1}\ miniature^{1}\ cherry^{tomato-1}$
7 m^{tu-2}
8 w lt/Y, & +/Y
9 v sc sn y m cherry bb
10 $v\ f^{tu-1}$
11 v (Y-autosome translocation)
12 v^{3} (T(X,2), homozygous lethal)
13 w
14 $w\ lt$
15 w^{m1}
16 w^{m2}
17 w^{m3}
18 N/w lt (Df.(1))
19 v f/Y & w lt/Y
20 y m ch/Y & w/Y
21 w/Anp

Chromosome 2

22 e^{Du}

Chromosome 3

23 cn

Chromosome 5

24 red eye
25 or

Multichromosomal

26 bb;p;vg (1;2;5)
27 st;sca;jv (2;3;5)

Several strains with mutant Y's
Many strains with T(X,Y)

Several strains with T(X, autosome)
Many mutants of D. hydei have been described in DIS 40:37, ff

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
21 H^h pe

Other Species

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

MATSUE, JAPAN: SHIAMNE UNIVERSITY
Department of Biology

spinofemora: 2 strains
hypocausta: 1 strain
formosana: 1 strain
nasuta(komaii?): 10 strains

SWANSEA, GLAMORGAN, WALES: UNIVERSITY COLLEGE OF SWANSEA
Department of Genetics

D. simulans

8 Caribbean strains
8 Italian strains
3 Californian strains
3 Columbian strains
w
v
Est^{6F} dh b py sd pm
Est^{6S} jv st pe
Est^{6F}

MANCHESTER, ENGLAND: PATERSON LABORATORIES
Department of Cytogenetics

D. ananassae
Wild Strains

CO (alleged to have CO in ♂)
NCO (alleged to have no CO in ♂)
3L (homozygous for a sequence in 3L)

Chromosome 1

w, y and f singly and in combinations

Chromosome 2

cu (curled) ma (maroon) se (sepia)
cd (cardinal) b (black) bx (bithorax)
singly and in various combinations

Chromosome 3

pc (peacock) px (plexus) ru (rough) up
(upward) singly and in combinations

PÔRTO ALEGRE, BRAZIL: UNIVERSIDADE FEDERAL DO RIO GRANDE DO SUL
Instituto de Ciências Naturais, Secção de Genética

D. willistoni

Wild Strains from: Florida, Perú, Cuba, Guatemala, Jamaica, Equador, Brazil: Tracua, Serra do Navio (Amapá), Manaus and Tabatinga (Amazonas), Pôrto Velho (Guaporé) Belém (Pará), Maranguape (Ceará), Salvador, Cassarongongo and Pedra de Una (Bahia), Xingú (Mato Grosso), Brasília, 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^h	S Hk abb bw (Inv)/lethal	pink (Inv)/lethal
w^e	S Hk abb bw (Inv)/cn	
w^+ y sn ru (Inv)/lethal	Em ph	

D. paulistorum

Wild Strains from: Apoteri (British Guiana); Brazil: Belém (Pará), Xingú (Mato Grosso), Maranguape (Ceará)

D. nebulosa

Radiosensitive and radioresistant strains

BHAGALPUR-7, INDIA: BHAGALPUR UNIVERSITY
Department of Zoology, Drosophila Laboratory

D. ananassae

Chromosome 2	Chromosome 3
ST^2/ST^2	ST^3/ST^3
AL/AL	DE/DE
GA/GA	ST^3/DE
ST^2/AL	ST^3/ET
AL/GA	DE/ET
ST^2/GA	

NORWICH, ENGLAND: JOHN INNES INSTITUTE

D. simulans

NAMUR, BELGIUM: FACULTÉS UNIVERSITAIRES N.D. DE LA PAIX
Medical School, Laboratory of Genetics

D. subobscura

MILANO, ITALY: UNIVERSITA' DI MILANO
Istituto di Genetica

D. simulans

Wild Stocks	Chromosome 1	Chromosome 2	Stocks selected for tumor manifestation
1 Aspra	2 st	3 net	4 tu Bl
			5 tu Aspra

SEOUL, KOREA: CHUNGANG UNIVERSITY
Department of Biology

D. suzukii

D. auraria : Race A (15 wild strains)
Race B (3 " ")
Race C (10 " ")

D. nigromaculata
D. pseudoobscura
D. virilis (5 wild strains)

CHANDIGARH, INDIA: PANJAB UNIVERSITY
Department of Zoology

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

DROSOPHILA SPECIES - NEW MUTANTS

Report of J. Grossfield

D. auraria Type C

st: scarlet Eye color bright red, darkening slightly with age. Ocelli colorless through lifespan. Autosomal recessive. Spontaneous in wild stock. Viability and fertility excellent.

D. auraria Type A

w: white Eye color and ocelli pure white. Sex linked recessive. EMS induced in wild stock. Viability and fertility excellent. Probably homologous with white locus in D. melanogaster.

saf: saffron Eye color and ocelli slightly yellowish. Sex linked recessive. EMS induced in wild stock. Viability and fertility excellent. Probably homologous with white locus in D. melanogaster.

gaucha

Report of Patricia Iturra

w: white Iturra 1965 Sex-linked recessive. Spontaneous in a mixed strain from different geographic localities of Brazil. Eyes white; ocelli, Malpighian tubes and testis sheath colorless. Salivary gland chromosome analysis by Brncic revealed no associated chromosomal aberration.

y: yellow Iturra 1966 Sex-linked recessive. Spontaneous in a mixed strain from different localities in Brazil. Body color rich yellow. Hairs and bristles brown with yellow tips. Wing hairs and veins yellow. Larval setae and mouth parts indistinguishable from dark brown of wild type. Salivary gland chromosomal analysis by Brncic revealed no associated chromosomal aberrations.

raychaudhurii

Report of S.P. RayChaudhuri and O. Kaul

dct: dichaete Wings stretched out like the aeroplane wings. Expression similar in both sexes. Viability reduced in females. Fertility lower than the wild stock. 90%.

dm: domed Wings sloping laterally, a little diverging, similar expression in both sexes. Penetrance incomplete.

or: orange Eye color translucent orange at eclosion, changes to dark after some days. Body color lighter. Penetrance complete, autosomal.

sn: singed All bristles and hairs smaller than usual, curled wavy or twisted; especially the bristles of thorax and scutellum reduced to thick stumps. Ocellars absent or hairy. Expression better in males. Viability and fertility reduced in both sexes.

crvn3: additional-crossvein Additional crossvein between L_3 & L_4, just near the anterior crossvein. Expression in both sexes similar. Sometimes 3 or more crossvein seen only in one wing.

cvn: crossveinless Posterior crossvein absent. Expression equal in both sexes. Wing shape normal. Sometimes a few flies with incomplete posterior crossvein appear in the culture. Autosomal.

se: sepia Eye color brown on hatching darkening to sepia.

virilis

Report of S. Ohba

tx: taxi Ohba 66d. Spontaneous in Tokyo strain. Wings held out at about 75° from body axis, somewhat narrow. Slightly slow development. Good viability. Chromosome 2.

si: ski Ohba 67e. Spontaneous in Tokyo strain. Slightly upturned wing tips in homozygotes. Chromosome 2.

w^{62}: white-62 Ohba 62k. Spontaneous in Tokyo strain. Likely reoccurrence of w.

cn^{61}: cinnabar-61 Ohba 61k. Spontaneous in Tokyo strain. Allelic to cn.

Est-1: Esterase-1 Naturally occurring allele. One of nine positively migrating α-esterases which react to α-naphthyl acetate when both α- and β- naphthyl acetate were used together as substrates after agar gel electrophoresis of single fly homogenates. The mobility of Est-1 is the slowest among nine α-esterases. Location: chromosome 2.

Est-1^-: Esterase-1^- Naturally occurring allele. Allelic to Est-1. Homozygote shows no esterase activity in the position of Est-1. Est-1/Est-1^- heterozygote produces the same band as Est-1/Est-1 homozygote but esterase activity is low.

Est-2^A: Esterase-2^A
Est-2^B: Esterase-2^B
Est-2^C: Esterase-2^C
Est-2^D: Esterase-2^D
Est-2^O: Esterase-2^O

Naturally occurring multiple alleles of β-esterases which react specifically to β-naphthyl acetate when mixed substrates were used. In homozygous conditions the mobility increases in the order from A/A to D/D. Est-2^O/Est-2^O homozygote shows no band of esterase activity. Heterozygotes A/B, A/C, A/D, B/C, B/D, and C/D contain the parental enzymes plus a hybrid enzyme of intermediate mobility. A/O, B/O, C/O and D/O heterozygotes produce the same esterase bands as A/A, B/B. C/C and D/D homozygotes respectively but esterase activity is low. Chromosome 2.

Est-3: Esterase-3
Est-3^-: Esterase-3^-

Naturally occurring α-esterase alleles. Mobility of Est-3 in agar gel electrophoresis is faster than Est-1. Est-3^- is a silent allele which produces no esterase band in the position of Est-3. Chromosome 2.

Est-4: Esterase-4
Est-4^-: Esterase-4^-

Naturally occurring α-esterase alleles. Mobility of Est-4 is faster than Est-3. Est-4^- is a silent allele like Est-3^-. Chromosome 2.

Est-5 and Est-5⁻: Esterase-5 and Esterase-5⁻

Est-6 and Est-6⁻: Esterase-6 and Esterase-6⁻

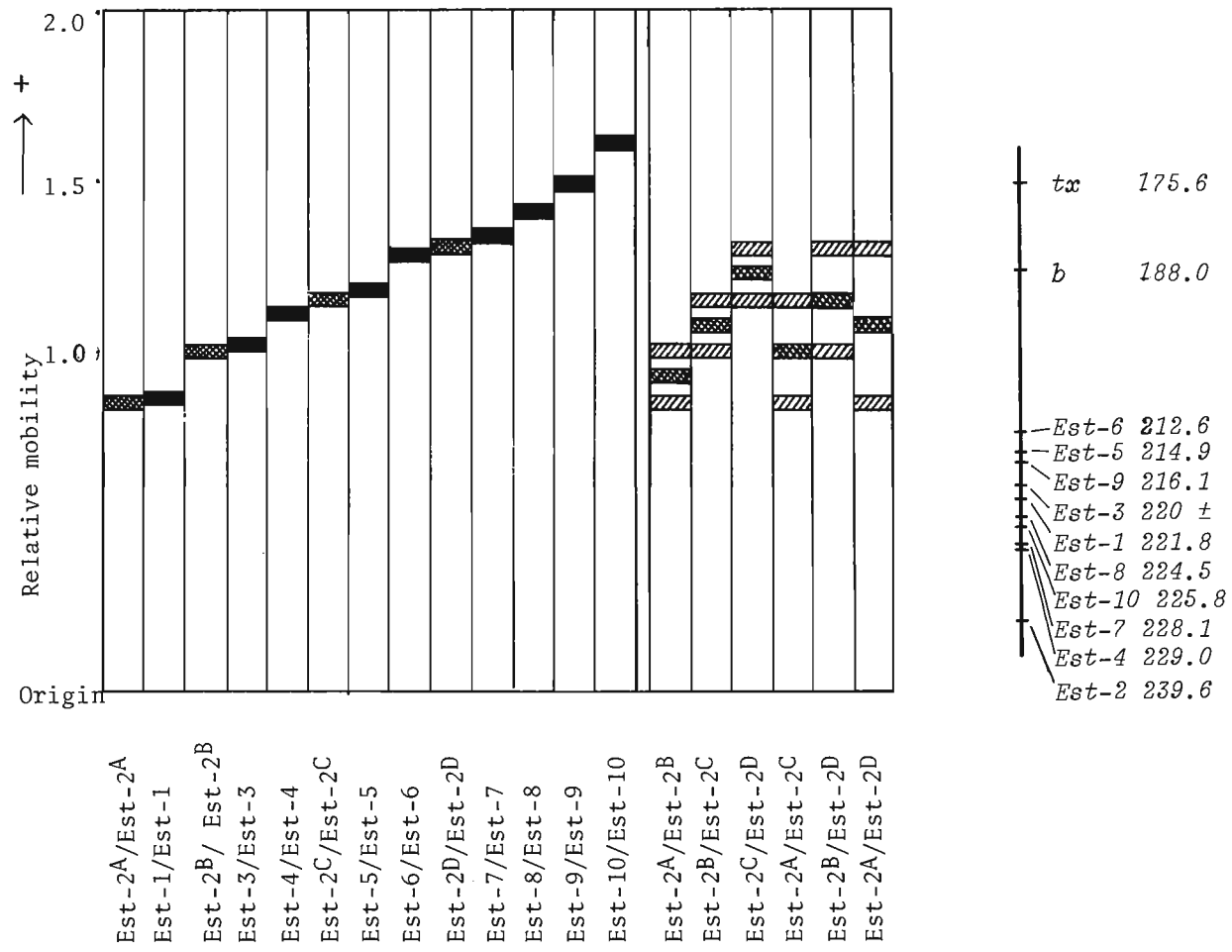
Est-7 and Est-7⁻: Esterase-7 and Esterase-7⁻

Est-8 and Est-8⁻: Esterase-8 and Esterase-8⁻

Est-9 and Est-9⁻: Esterase-9 and Esterase-9⁻

Est-10 and Est-10⁻: Esterase-10 and Esterase-10⁻ These six pairs of α-esterase alleles were also found at different loci on chromosome 2. Zymograms of these esterase alleles and a tentative chromosome map are shown in the following figure:

Phenotypes of esterase variants and tentative map of the Second chromosome



fulvimacula

Report of O. Hess

y: yellow I. Gretzmacher, 1967 Chromosome 1 (sex-linked). X-ray induced in ♀, recovered as single ♂. Yellow body, light brown bristles; RK1; viability and fertility considerably reduced. Mitotic and salivary gland chromosomes apparently normal. Note: in the progeny of a large number of females, X-irradiated in order to receive sex-linked markers, yellow has been recovered five times. In contrast to this, other mutations have so far never been found.

yellow-attached-X R. Freye, 1967 X-ray induced in females homozygous for y (see above). Neuroblast metaphases show a large metacentric chromosome.

ananassae

Report of D. Moriwaki

M-b: Minute-b Moriwaki 67d22. 2-. Spontaneous 1 in a cross wfy x kk. Minute bristles. Dominant. Homo. lethal.

app: approximate Moriwaki 67j23. 3-. Appeared as single in M-b stock. Posterior crossvein shifts toward anterior crossvein obliquely.

bb^{67}: bobbed-67 Moriwaki 67j23. 4-. Spontaneous in +F5 wild stock. Only in bristles shortened and often abdomen etched. Male shows neither characteristic. Normal allele exists in Y-chromosome too.

bn^{67}: broken-67 Moriwaki 67k21. 2-R. Spontaneous in +F8 wild stock. Posterior crossvein missing or broken. Emerging the later, false normals appear the more. Penetrance is low in male.

M-c: Minute-c Moriwaki 68a11. 3-. Recovered as single in a cross bb^{67} x bb^{67}/Rf bri. Minute bristles. Dominant. Homo. lethal.

Bd: Beaded Moriwaki 68e16. Spontaneous as single from a cross px x M-c px/+F8. Wings reduced by marginal excisions. Low viability and fertility. Dominant. Homo. lethal.

Report from Paterson Laboratories, Manchester, England

bx: bithorax Chromosome 2. A much milder form of the bithorax condition as seen in melanogaster. Spontaneous origin in curled flies (which made it more visible). Two small lumps of bristle-covered tissue, presumably of metathoracic origin, lying on each side of the mid-line above the balancers. Expression - variable. Penetrance complete. Viability and fertility good.

up: upward Chromosome 3. Wings held up and slightly turned, resembling the position in an over-etherised fly. Spontaneous origin. Expression variable. Penetrance about 70%. Viability and fertility good.

TECHNICAL NOTE

Oliver, Dorothy V. and J.P. Phillips. Department of Zoology, University of Texas, Austin, Texas. Fruit fly fractionation.

Our interests in Drosophila enzymology have prompted us to develop a method for the fractionation of the adult fruit fly into its basic morphological components. The following describes a method for rapidly obtaining gram quantities of Drosophila heads, bodies (abdomen-thorax complex minus appendages), and legs.

Flies are collected in a clean dry milk bottle and frozen on Dry Ice for thirty minutes. The bottle is then rapped sharply 15 or 20 times against a hard rubber pad. The fractured flies are then shaken through stacked wire sieves of 20, 30 and 40 mesh, respectively.

Wings fragment easily and coat the inside of the milk bottle. Bodies and undecapitated flies are retained on the 20 mesh screen. Some bodies, mostly male, pass through and collect on the 30 mesh screen. Heads collect on the 40 mesh screen, which passes the fragmented leg parts. Those heads which remain stuck in the 30 mesh screen can be freed with a camel hair brush.

With a little practice essentially pure heads, bodies and legs can be obtained in amounts limited only by the amount of starting material.

Pipkin, S.B. and T.A. Bremner. Howard University, Washington, D.C. Coordinate activity of octanol dehydrogenase isozymes and its breakdown in Drosophila interspecific hybrids.

The octanol dehydrogenase (ODH) isozyme complex of the sibling species D. metzii, D. pellewae, and D. leticiae is observed in zymograms using agar gel electrophoresis as at least 15 bands, including those seen in different developmental stages of variant strains (Fig. 1). Genetic evidence has been interpreted as supporting the hypothesis of a tetramer subunit structure of isozymes in positions 3 to 7 which are supposed to contain subunits coded by two structural genes, ODH_1 and ODH_2 (Pipkin, 1968, 1969a, 1969b). ODH isozymes anodal to position 3 and cathodal to position 7 have been hypothesized to depend on duplicate ODH structural genes (Pipkin, 1969b). Genetic studies indicate that isozyme patterns of true breeding A and B type variants (Fig. 2) depend on regulatory alleles ODH^A_{1c} and ODH^B_{1c} affecting the time and rate of subunit synthesis by the ODH structural genes, ODH_1 and ODH_2 (Pipkin, 1968, 1969a, 1969b). In the progeny of crosses of A and B type variants extracted from the Barro Colorado Island strain of D. pellewae,

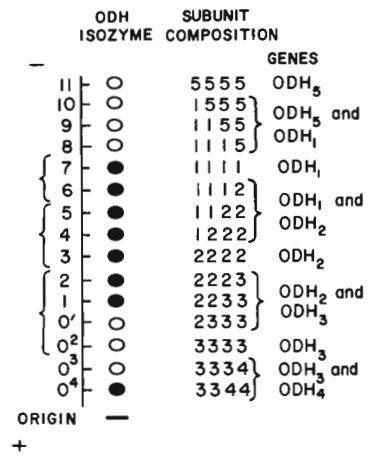


Fig. 1. Duplicate gene hypothesis explaining the subunit structure of ODH isozymes in D. metzii & D. pellewae.

the maternal ODH pattern is seen in A♀/B♂ hybrid embryos 24 hours old (Fig. 3a) and in B♀/A♂ hybrid embryos of the same age (Fig. 3e). In addition, these embryos display new slowly migrating isozymes at positions 1, 2 and 0^1. Both the maternal pattern affecting isozymes in positions 3 to 7, and the new embryonic isozymes disappear in late first instar larvae. At this time synchronous activity of both paternal and maternal regulatory alleles is indicated by the appearance of a 3, 4,5 triplet pattern in both A♀/B♂ (Fig. 3b,c) and in B♀/A♂ (Fig. 3f-j) hybrid first instar larvae.

Coordinate activity of two groups of isozymes is observed in the 24 hour A♀/B♂ and B♀/A♂ embryos of D. pellewae, respectively. In A♀/B♂ embryos (Fig. 3a), the isozymes at positions 3 and 1 show strong staining, and the #5 isozyme is weak or sometimes undetectable. In B♀/A♂ embryos, on the other hand, the #5 isozyme shows strong staining and the #3 and #1 isozymes are faint (Fig. 3e). The correlation of activity as judged by the intensity of formazan staining of the isozymes at positions 3 and 1 can be explained by assuming that in embryos, ODH_2 and duplicate gene ODH_3 share subunits in the #1 isozyme of the A variant, whereas a strongly staining isozyme at position 5 in the B variant indicates subunit sharing by ODH_2 and ODH_1.

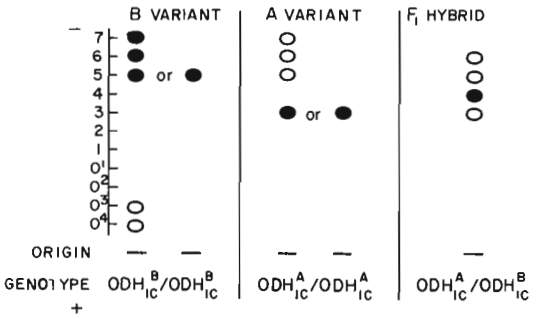


Fig. 2. ODH isozyme patterns of B and A type variants and of their hybrid.

In interspecific hybrids all development is retarded. Moreover, the 3,4,5 triplet pattern expected in post-embryonic stages is not always observed. This is believed to be due to the failure of operation of either the maternal or the paternal regulatory alleles or to their acting with altered timing. As a result, certain third instar larvae of leticiae B♀/metzii A♂ hybrids showed only a single isozyme at position 3 instead of the expected 3,4,5 hybrid pattern, indicating absence of detectable action of the maternal regulatory allele, ODH^B_{1c}. In brown pupae of the same hybrids (Fig. 4,c,d) both maternal and paternal regulatory alleles were apparently acting to cause structural genes to code for subunits in isozymes at positions 4 and 5, but the rate and/or time of activity of structural gene ODH_2 was altered so that the #3 isozyme, the supposed homotetramer composed of "2" subunits, was undetectable. Third instar larvae of the reciprocal cross, metzii A♀ x leticiae B♂, showed isozymes at positions 3 and 4 but not at 5 (Fig. 4e,f), indicating reduced or faulty activity of the paternal regulatory allele, ODH^B_{1c}. In brown pupae of metzii A♀/leticiae B♂ hybrids, an expected 3,4,5 triplet

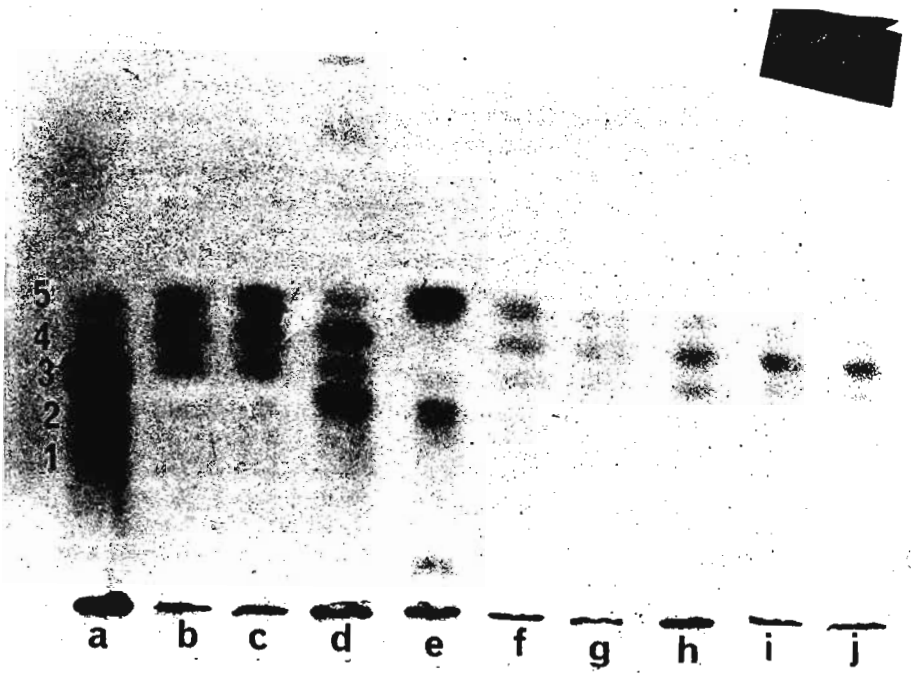


Fig. 3. Reciprocal hybrids of A and B type variants of D. pellewae: a, A♀/B♂; e, B♀/A♂ 24 hour embryos; b,c, A♀/B♂ first instar larvae; f-j, B♀/A♂ first instar larvae.

pattern was observed in the individuals assayed in Fig. 4g,h, indicating synchronous activity of maternal and paternal regulatory alleles. However, a difference in pupal ODH patterns of reciprocal hybrids of D. metzii and D. leticiae was sometimes observed, suggesting a breakdown of normal regulation of the structural gene ODH_2 and its duplicate gene ODH_3. Normally the slowly migrating embryonic isozymes at positions 2,1,0^1, and 0^2 are undetectable in post-embryonic stages of both D. metzii and D. leticiae except in concentrated mass homogenates (i.e., electrophoresed aliquots of 100 females per 0.25 ml of 0.2 M tris buffer) of D. metzii. This suggests that the ODH_3 structural gene is active in the embryonic period when it shares subunits with ODH_2 but shows little or no activity in post-

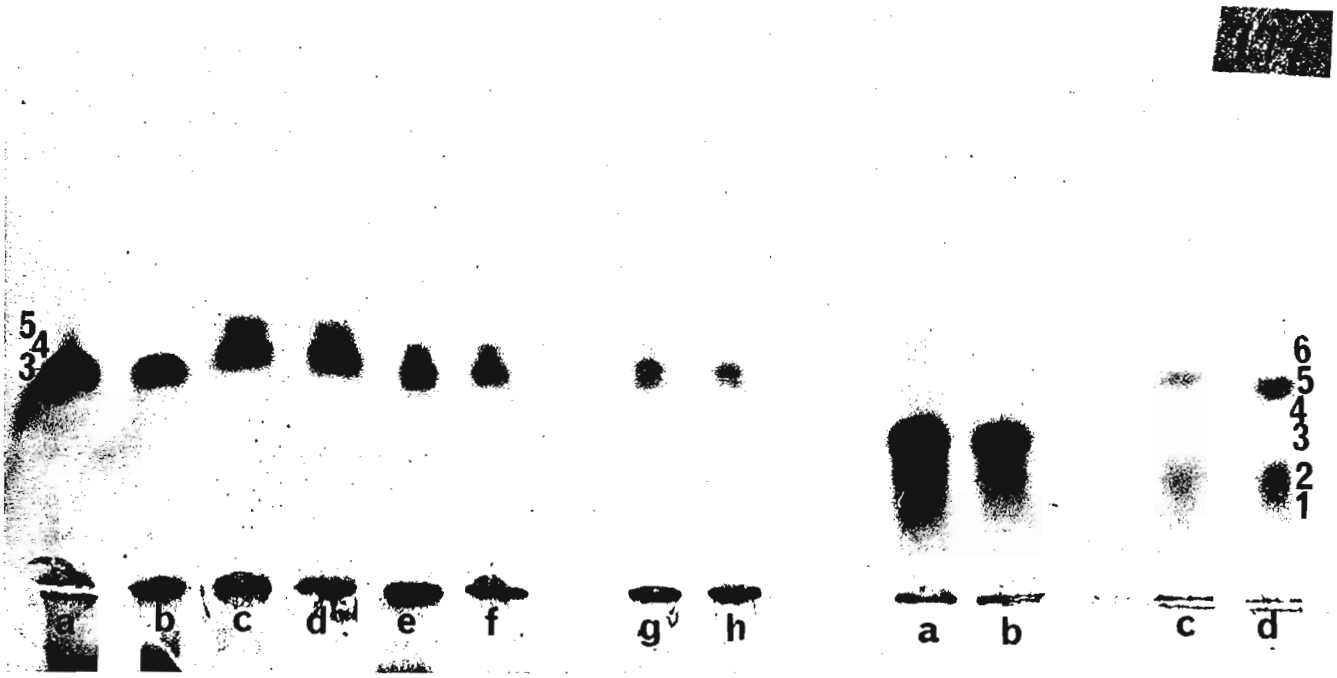


Fig. 4 (left). leticiae B♀/metzii A♂ hybrids: a,b single third instar larvae; c,d single pupae metzii A♀/leticiae B♂ hybrids: e,f single third instar larvae; g,h single pupae.
Fig. 5 (right). a,b metzii A♀/leticiae B♂ hybrids: single pupae; c,d leticiae B♀/metzii A♂ hybrids; single pupae.

embryonic stages. In interspecific hybrid pupae of D. metzii and D. leticiae, strong formazan staining of isozymes at positions 2,1, and 0^1 is sometimes observed, indicating abnormally high activity of structural gene ODH_3 during the pupal stage. For example, the pupae of both metzii A♀/leticiae B♂ hybrids in Fig. 5a,b and of leticiae B♀/metzii A♂ hybrids (Fig. 5c,d) showed strongly staining isozymes at positions 2,1, and 0^1, instead of the expected 3,4,5 pattern. Metzii A /leticiae B hybrids assayed as single adult females sometimes showed the expected 3,4,5 triplet pattern, but often only a single isozyme at position 3 was observed, indicating that activity of the paternal ODH^B_{1c} allele was undetectable. In conclusion our studies indicate that regulation of subunit sharing between ODH structural genes may be disturbed in third instar larvae, pupae, and adult stages of hybrids of D. metzii and D. leticiae. The parental species were shown by Pipkin (1968) to differ in multiple translocations. Similar results regarding ODH isozyme patterns have been obtained for D. metzii/D. pellewae hybrids.

This work was supported by PHS Grant 14937 and National Science Foundation Grant GB 8770.

References: Pipkin, S.B., 1968, Evol. 22: 140-156; Pipkin, S.B., 1968, Genetics 60: 81-92; Pipkin, S.B., 1969a, DIS 44: 59-61; Pipkin, S.B., 1969b, In press, Oct. Genetics.

Mukherjee, A.S. and A. Das. University of Calcutta, India. A recombination associated segregation - distortion in D. ananassae.

A case of segregation distortion has been observed in the inbred laboratory strain, px-pc, of D. ananassae. It is comparable to the SD action of D. melanogaster (Sandler et al, 1959), but unlike SD, this phenomenon is associated only with the recombinant classes and in both sexes. The recombinant classes px + and + pc from the px pc/+ + (male or female) parent are not complementary to each other; px flies appear in the progeny in a much higher proportion than the pc. The proportion of the complementary non-recombinant classes is close to 1:1 (the mean K values, i.e. the proportion of px pc among all non-recombinants, vary from 0.46 to 0.55). There is considerable inequality of the complementary recombinant classes in both sexes (i.e. px pc/+ + male as well as female) but it is unusually high in the male (K values, i.e. the proportion of px among all recombinants, are always close to 1.0)(Table 1). In testcrosses with px +/+ pc,

Table 1
Distribution of testcross progeny and K values in heterozygous males and females. Genotypes of F_1 parents: px pc/+ + x px pc/px pc in Expts. 1-6 and px +/+ pc x px pc/px pc in Expts. 7-8

Expt. No.	Sex of heterozygote parent	$+_a+$	$px_b pc$	$\bar{K}$ b/a+b	px/c	pc/d	$\bar{K}$ c/c+d	No. of crosses
1	Female	1892	1694	0.48	1028	323	0.76	45
2	Female	460	448	0.49	345	75	0.83	13
3	Female	681	750	0.52	631	84	0.88	19
4	Male	1091	979	0.46	399	1	0.99	29
5	Male	709	746	0.51	405	4	0.99	20
6	Male	370	451	0.55	215	2	0.99	12
7	Female	219	355	0.62	859	740	0.53	21
8	Male	38	147	0.80	982	931	0.53	26

the non-recombinant px and pc classes were in equal proportion and the recombinant px pc and + + classes were highly disproportionate, thus conforming with the data of the previous set (Table 1). Tests on viability and penetrance of the mutants px and pc, in relation to the wild type (a6+) or px pc double recessive, do not show any abnormality. It is, therefore, suggested that there may be certain genetic factor or factors closely associated with the px locus, whose function is to prevent the recovery of that recombinant class which is separated from the factor following the exchange. It may be noted that this case of segregation distortion perhaps records the first example of the phenomenon in a species in which spontaneous crossing over in males is quite frequent, unlike other species of Drosophila. The results presented above, however, do not exclude the possibility of a type of nonrandom disjunction (Novitski, 1967, Ann. Rev. Genet., 1: 71-86), somehow operating in both sexes.

Fahrig, R.* Genetisches Institut der Justus-Liebig-Universität, Giessen, Germany. The influence of temperature upon the concentration of the free amino acids of D. melanogaster.

The free amino acids of Drosophilae cultivated for some generations at a distinct temperature are very constant in their concentrations. A change of the temperature is correlated with a change of the concentration of many amino acids. In this work we have determined the concentration of 19 different amino acids by using an automatic amino acid analyzer of Beckman. The concentration changed in nine amino acids in larvae (96 h old), in ten in pupae (24 h old) and only in one in adults (72 h old). The concentration of ammonia which has also been determined is not influenced by temperature.

AMINO ACIDS	LARVAE			PUPAE			ADULTS		
umol wt/100mg wet weight	18°C	24°C	30°C	18°C	24°C	30°C	18°C	24°C	30°C
Histidine	0.28	0.28	0.28	0.38	0.33	0.30	0.49	0.47	0.48
Lysine	0.27	0.18	0.05	0.27	0.20	0.07	0.06	0.05	0.05
Arginine	0.30	0.34	0.38	0.37	0.30	0.22	0.36	0.37	0.35
Ammonia	0.24	0.23	0.24	0.25	0.24	0.26	0.43	0.40	0.42
Aspartic acid	0.12	0.11	0.12	0.15	0.14	0.15	0.17	0.17	0.17
Glutamic acid	0.42	0.40	0.39	0.56	0.56	0.55	0.53	0.54	0.53
Threonine	0.18	0.11	0.05	0.13	0.10	0.08	0.08	0.08	0.08
Serine	0.16	0.21	0.31	0.18	0.13	0.10	0.17	0.17	0.18
Proline	0.36	0.34	0.35	0.17	0.18	0.17	0.32	0.30	0.32
Glycine	0.23	0.23	0.22	0.18	0.16	0.17	0.29	0.29	0.30
Alanine	0.64	0.63	0.64	0.35	0.26	0.16	0.39	0.40	0.41
Valine	0.05	0.04	0.05	0.22	0.17	0.12	0.05	0.06	0.05
Methionine	0.01	Traces	Traces	Traces	Traces	Traces	Traces	Traces	Traces
Isoleucine	0.01	Traces	Traces	0.12	0.07	0.01	Traces	Traces	Traces
Leucine	0.07	0.07	0.07	0.40	0.24	0.11	0.04	0.04	0.05
Tyrosine	0.37	0.34	0.32	0.16	0.16	0.17	0.08	0.08	0.08
Phenylalanine	0.01	0.01	0.01	0.07	0.05	0.03	0.02	0.02	0.02
β-Alanine	0.02	0.02	0.01	0.03	0.03	0.03	0.45	0.38	0.33
γ-Aminobutyric acid	0.02	0.02	0.03	Traces	Traces	Traces	0.07	0.06	0.06
Ornithine	0.03	0.03	0.04	Traces	Traces	Traces	Traces	Traces	Traces
TOTAL	3.79	3.59	3.55	3.99	3.32	2.70	4.00	3.88	3.88

A rise of temperature in the cultures results in a decline of the concentration of all amino acids being influenced in larvae, pupae and adults with exception of arginine and serine in larvae.

Amino acids	Larvae	Pupae	Adults
Histidine		+	
Lysine	+	+	
Arginine	-	+	
Ammonia			
Aspartic acid			
Glutamic acid	+		
Threonine	+	+	
Serine	-	+	
Proline			
Glycine			

Amino acids	Larvae	Pupae	Adults
Alanine		+	
Valine		+	
Methionine	+		
Isoleucine	+	+	
Leucine		+	
Tyrosine	+		
Phenylalanine		+	
β-Alanine			+
γ-Aminobutyric acid			
Ornithine			
TOTAL	8	10	1

The total amount of all amino acids shows little differences in larvae, high differences in pupae (in accordance with Anders, Drawert, Anders and Reuther 1964) and no differences in adults.

References: Anders, F., Drawert, F, Anders, A and Reuther, K.H., 1964, Z. Naturforschung 19b: 495-499.

* New address: Zentrallaboratorium für Mutagenitätsprüfung, 7800 Freiburg/Breisgau, Breisacherstrasse 33.

Browning, L.S. University of St. Thomas, Houston, Texas. A radiation dose rate effect occurring in developing reproductive cells of male Drosophila.

About 600 specimens of D. melanogaster in the late third instar larval stage (after the larvae have become motionless) or the prepupal stage were removed from culture medium, mixed in a beaker of water, then divided into four lots of 150 each. Twelve hours later one group was subjected to continuous gamma radiation from a Ce^{137} source over a 64-hr period for a total dose of 2000 r (0.52 r/min). Each of the other groups was given 2000 r gamma radiation from a Co^{60} source over a period of ten minutes (200 r/min), one group being irradiated at the beginning of the 64-hr period ("0 hrs"), another at the middle of the 64-hr period, and the third at the end of the 64-hr period. About sixty males hatched from each group. Recessive lethals occurring in the paternal X chromosomes (of genotype y sc^{S1} In49 sc^{8}) were scored by crossing the males to Canton S females and individually testing the daughters for lethals. The table below shows the results.

Brood (2-da)	Low Intensity (0.52 r/min) 0-64 hrs			High Intensity (200 r/min) Irradiated at 0 hrs			32 hrs			64 hrs			Unirradiated Controls		
	NO.	L	%	No.	L	%	No.	L	%	No.	L	%	No.	L	%
1	306	27	12.2	282	1	0.4	136	0	0	151	0	0	599	0	0
2	378	29	7.7	390	0	0.0	328	0	0	409	1	0.2	202	0	0
3	250	8	3.2										55	0	0
4	314	9	2.9												
1-4	1,277	83	6.5	672	1	0.2	464	0	0	560	1	0.2	856	0	0
6-11	2,127	9	0.4	192	1	0.7	391	0	0	395	1	0.3			

The difficulties of interpreting data on mutation production when germ cells are undergoing maturation at the time of treatment are well known. However, the data given in the table show an unexpected and almost complete absence of sex-linked recessive lethals recovered after acute treatments with 2000 r applied at times so widely spaced as the time of pupation ("0 hrs") when the testes contain mostly spermatocytes ready to undergo meiosis plus a few spermatogonia, at 32 hrs later when many spermatocytes are undergoing meiosis and post-meiotic cells are present, and at 64 hrs when most of the cells are post-meiotic with many spermatids and spermatozoa present ("Biology of Drosophila," Demerec, Ed., 1950, pp 282-3; 302-4.) One possible explanation for the obliteration of the lethal rate which occurred at each of these stages is that all metabolizing cells undergoing spermatogenesis were killed by the radiation, only the radiation-resistant spermatogonia being left to repopulate the testes and manifest mutation after radiation.

Previously it had been noted in an experiment done in connection with NASA's biosatellite project that when pupae were exposed to 2460 r of gamma radiation continuously over a 64-hr period, a lethal frequency of 7.8% (58/740) in broods 1 thru 4 was produced, but in broods 5 thru 10 only 1 lethal was found in 2,625 tested chromosomes (0.04%). The untreated stock had previously given a rate of 0.05% (9 lethals in 11,625). As shown in the table, when this experiment was repeated, 9 lethals were found among 2,127 chromosomes, making it appear that no depression of the frequency below that of the spontaneous frequency had actually occurred, and the relatively high frequency in the earlier broods (6.5%) was consistent with the earlier experiments. Oster (J. of Cellular and Comp. Physiology 58: 203-7, 1961) has reported a lethal frequency of 0.7% or 9 lethals in 1,247 after an acute dose of 2000 r when young larvae containing only spermatogonia were irradiated. High lethal frequencies have been observed by us in the early broods in three separate experiments in which exposure to approximately 2000 r has been started at approximately the time of pupation and continued at a rate of about 0.5 r per minute.

Acute doses of 500, 1000, and 1500 r applied at the time of pupation have not produced a drastic drop in the lethal frequency, the lethal frequencies in broods 1-3 being 6.7, 4.5 and 2.0%, respectively. This inverse relationship to dose may be a further manifestation of the killing of potential lethal-bearing cells by the higher acute doses, although this must remain speculative until more data have been obtained. The conclusion is justified, however, that doses of acute radiation of 2000 r applied at various times during the pupal stage result in a drastic reduction in the number of recovered lethals, but that at 1500 r and below the effect

is diminished and recovery of recessive lethals is possible. (Work supported by NASA Contract NAS2-4849.)

Fahmy, O.G. and M.J. Fahmy. Institute of Cancer Research, Chalfont St. Giles, England. Design for testing specific mutability at the bobbed locus.

In our studies of the genetic effects of carcinogens, it was felt desirable to undertake specific mutability tests on some heterochromatic gene loci, of which bb was an obvious representative. A major difficulty with bb, however, is that different alleles show considerable variation in viability as well as phenotypic expression, and most homozygous stocks tend to show declining phenotypes on keeping. A strong allele of bb, in combination with f and mal^{bz}, has now been found which remained stable when balanced against sc^{S1} B InS w^a sc^8 (M-5). The homozygous triple-marker females invariably showed an extreme expression of bb, both with regard to the reduction in the size of the bristles and the etching of the abdominal sclerites, but their viability was substantially reduced. The heterozygous females, against a standard-X (f mal^{bz} bb/+), had slightly shortened thinner bristles, indicating that the bb allele had a "semi-dominant" effect. The hemizygous triple-marker males appear bb^+ against a normal Y, but are lethal against Y^{-bb}.

The f mal^{bz} bb/M-5 stock has been successfully used in specific mutability tests at the various marker loci (including w^a on the M-5 chromosome), using several chemical carcinogens. Where activity on bb^+ was required, the stock females were mated to + Y^{-bb} non-bobbed treated males, to ensure the elimination of the background bb mutations from the test. The F_1 consisted of only three of the expected classes; f mal^{bz} bb/Y^{-bb} males were lethal. The F_1 females carrying the M-5 chromosome heterozygously were scored for w^a mutations and a sample was bred on for the assay of the sex-linked recessive mutation frequency in the F_2, by the usual Muller-5 technique. The alternative class of F_1 females (non-M-5) were scored for f, mal^{bz} and bb and all suspected mutants were subjected to confirmatory genetic tests. In particular, flies showing reduction in bristles were backcrossed to the stock bb allele, to distinguish the true sex-linked instances from the autosomal dominant Minutes.

The phenotypic expression of 59 bobbed alleles induced by a carcinogenic hydrocarbon in various test crosses.

Phenotypic expression	Test crosses		
	Homozygous	bb with f mal^{bz}	Y^{-bb}
Wild type	0	0	5
Bristle effect: slight	28	2	2
: intermediate	15	22	29
: extreme	3	6	3
Bristle and abdomen effects	13	29	16
Lethal	0	0	4

Details of the genetic testing of 59 bb alleles induced by the carcinogen 7-bromomethyl-12-methyl benz(a)anthracene are given in the accompanying table. On the whole, alleles with clear expression homozygously also showed with more exaggerated phenotype when crossed to the test stock bb or Y^{-bb}, while those with only slight effects were rendered scorable. The stock bb was more useful in this respect since it revealed the majority of the induced mutants with both bristle and abdomen effects; also with Y^{-bb}, 5 alleles overlapped wild-type and 4 were lethal. It would appear, therefore, that our stock bb was an appreciable size deletion which permitted the recovery of a range of induced deletions within the bb^+ locus, particularly those of smaller size : of slight expression homozygously. Conversely, however, induced deletions of a size approaching that of the test marker, could have been inviable, which might have resulted in underestimating the activity of the tested compounds. The test stock is now being modified to overcome this difficulty.

Ogonji, G.O. Howard University, Washington, D.C. Genetic control of the octanol dehydrogenase isozymes in D. albirostris.

The existence of octanol dehydrogenase (ODH) in multiple molecular forms in D. melanogaster was reported by Courtright, Imberski, and Ursprung (1966). Since then, the genetic control of ODH in D. metzii and D. pellewae has been studied by Pipkin (1968, 1969a, 1969b). Using an agar gel electrophoresis method, true breeding ODH isozyme variants were extracted from polymorphic strains of D. albirostris from El Valle, Panama (Fig. 1); Darien, Panama; Summit Gardens, Panama; Rio Raposo, Colombia; and Leticia, Colombia. The isozyme patterns of the extracted variants were of three main types: A, B, and B^1. The A type variants from Leticia and Summit Gardens, designated L-A and S-A, respectively, always possessed an isozyme at position 3 and occasionally additional isozymes were seen at positions 5 and 7, or 5,6, and 7. These positions are marked relative to those of extracted variants of D. metzii and D. pellewae, which belong to the same subgroup of the tripunctata species group as D. albirostris (see Fig. 1 of Pipkin, this issue of DIS). The two B type variants, designated EV-B4 and EV-B13 from El Valle, Panama, always possessed an isozyme at position 4.5 or 5 and 6, but differed in the manner in which they reacted in interstrain hybrids. The B^1 type

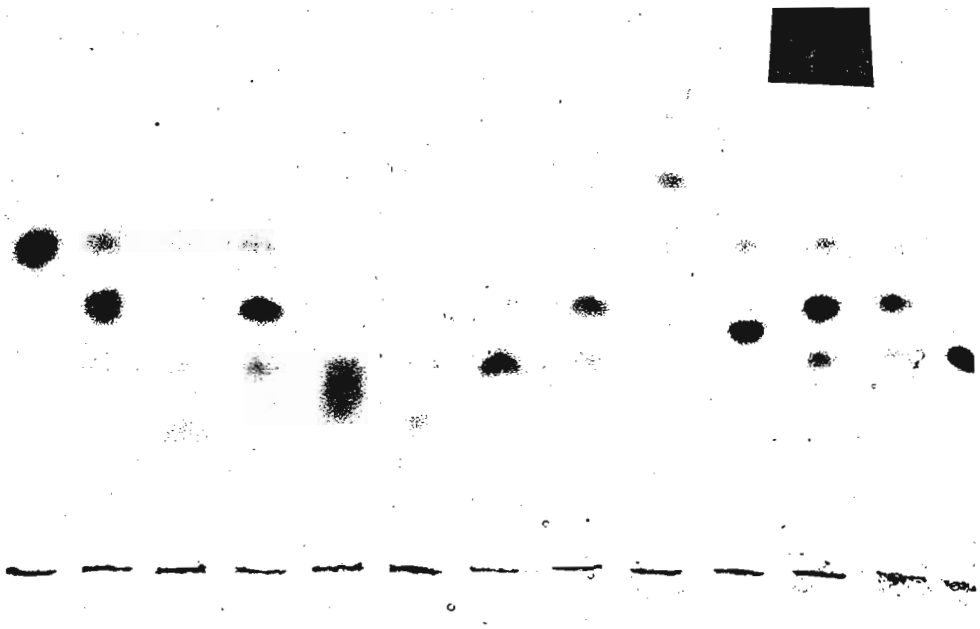


Fig. 1. ODH isozyme patterns of single adult females of D. albirostris, El Valle, Panama strain.

variant extracted from Darien, Panama, possessed a single isozyme at position 5. However, the B^1 variant extracted from the Rio Raposo, Colombia strain had an isozyme at position 5 and often at either 6 or 7. The F_1 hybrids between distinct variants usually displayed a triplet isozyme pattern in 8 day old adult females. A/B hybrids possessed a 3,5,7 pattern; A/B^1 hybrids, a 3,4,5 pattern; and B/B^1 hybrids, a 5,6,7 pattern. The frequencies of parental and heterozygote isozyme patterns occurring in F_2, backcross, and outcross progeny indicate a monofactorial inheritance. The A,B, and B^1 variants are believed to differ in multiple alleles of a single locus. The multiple allele interpretation is borne out by the ODH isozyme patterns of the segregating progeny of the outcross EV-B13/S-A x $D\text{-}B^1$. The parental pattern EV-B13 appears in Fig. 2a; that of $D\text{-}B^1$, in Fig. 2b; and S-A, in Fig. 2c. Among the outcross progeny, Fig. 2d, e,f,h,i,k show individuals with the triplet pattern 5,6,7 characteristic of $EV\text{-}B13/D\text{-}B^1$ heterozygotes; and Fig. 2g and j show individuals with the triplet pattern 3,4,5 typical of $D\text{-}B^1$/S-A heterozygotes.

An unusual single isozyme pattern was observed where triplet pattern was expected in certain progeny from crosses of both EV-B13♀ x S-A♂ and EV-B4♀ x L-A♂. Among the F_1 progeny from the EV-B4 x L-A cross, Fig. 3a,c,e,g,h,i,and j show individuals with the expected 3,5,7 pattern characteristic of EV-B4/L-A heterozygotes. Fig. 3b,d, and f show individuals with the aberrant single isozyme pattern 3 which suggests that mechanisms controlling synchronous

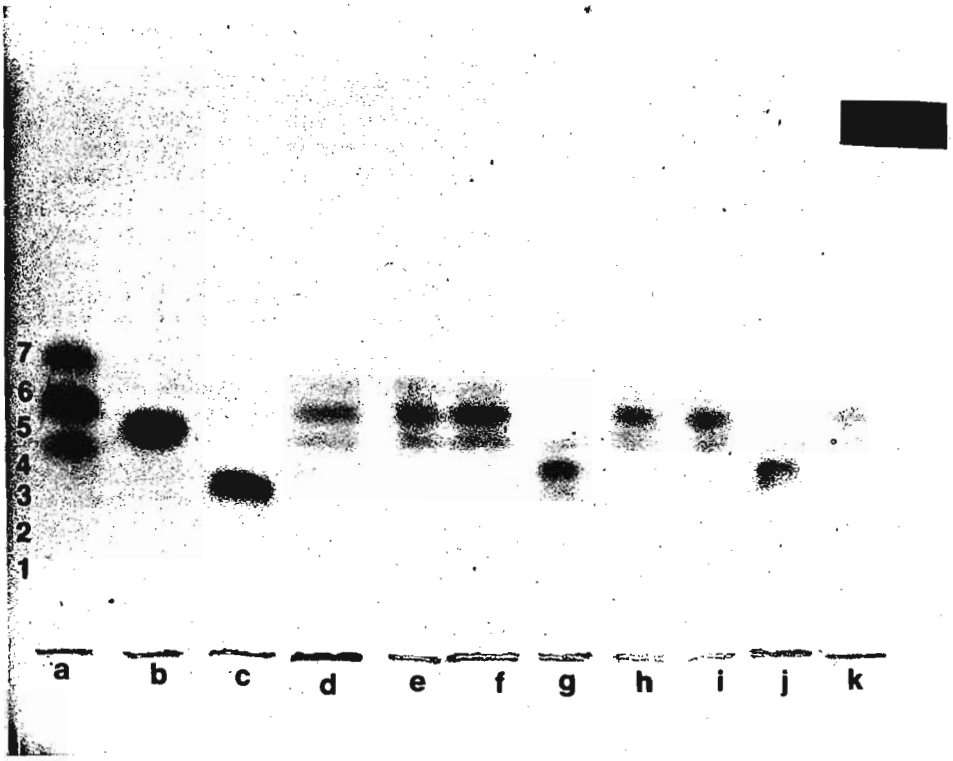


Fig. 2. Parental strain ODH isozyme patterns and patterns of the progeny of the cross EV-B13/S-A x D-B^1: parental strains: a, EV-B13; b, D-B^1; c, progeny: d,e,f,h,i,k, patterns of EV-B13/D-B^1 heterozygotes; g,j,3,4, 5 pattern of D-B^1/S-A heterozygotes.

activity of the maternally derived allele may break down occasionally as Pipkin and Bremner (this issue of DIS) have found for interspecific hybrids of D. metzii and D. leticiae.

According to developmental studies, D. albirostris embryos have in addition to adult isozyme patterns, slowly migrating ODH isozymes at positions 2,1,0^1, and 0^2. Furthermore, embryos of B and B^1 types of extracted lines possessed an isozyme at position 3 which is not detectable in imagines. This is taken as evidence that two structural genes code for sub-

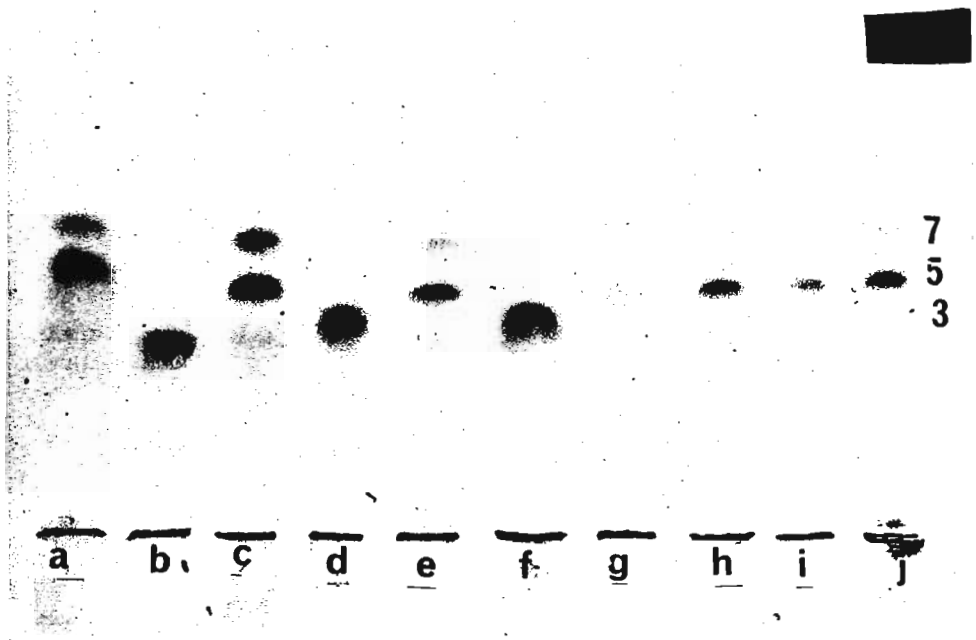


Fig. 3. F_1 progeny of the cross EV-B4 x L-A: a,c,e,g,h,i,j individuals with the expected 3,5,7 ODH pattern; b,d with the aberrant single isozyme at position 3.

units that may be present in isozymes at positions 3 to 7. Preliminary studies on heat lability of embryonic isozymes indicate that when treated with 50°C for 30 minutes, isozymes at positions 5,6,7 were heat labile, but those at positions 3,2,1,0^1, and 0^2 were still enzymatically active. This is further evidence that more than one structural gene codes for subunits that form the isozymes at positions 3 to 7. The three extracted variant types are considered to be regulatory variants that control the rate and/or time of subunit synthesis by structural genes, similar to the lactate dehydrogenase variant studied in mouse erythrocytes by Shows and Ruddle (1968).

This work was supported by PHS Grant 14937.

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Bairati, A. and M.E. Perotti, University of Milan, Italy. Occurrence of a compact plug in the genital duct of D. females after mating.

Some experiments have been performed to control the previously reported assumption (1) that the ejaculatory bulb secretion is injected with sperms into the female genital duct during mating. Females (10 for each interval) have been separated from males at various intervals from the beginning of mating. Their genital apparatus has been dissected in saline isotonic solution and observed with dissection, phase contrast and electron microscopes.

The following results have been obtained: 1) during the first 5 minutes from the beginning of mating no material is observed in the female genital duct. 2) between 5 and 7 minutes a compact plug appears filling the uterus lumen. It is cylindrical and made up of a homogeneous, thick and translucent substance. Before the appearance of the plug no sperms are present in the uterus and at about 7 minutes only few sperms have been observed in the most caudal portion of the female genital duct. 3) at 10 minutes, the mass acquires its largest size and many sperms appear within the uterus beyond the plug. Furthermore, some sperms are observed beating between plug surface and uterus walls. 4) at 12 minutes a very large number of sperms is assembled in the cephalic portion of the uterus. Some sperms are present also in the ventral receptacle. Within 14 minutes the sperms fill the receptacle and the spermathecae. 5) the plug is visible in the uterus since 5-7 minutes up to 6 hours - 6 hours and 30 minutes from the beginning of mating and disappears after the first egg has been laid.

Histochemical stainings demonstrated that both the bulb secretion and the plug inside the uterus possess the same staining properties, viz.:i) they stain with Sudan III and Sudan Black. ii) they reduce and osmium tetroxide solution, acquiring a deep dark coloring. iii) the material can be extracted and staining prevented when the material is treated with fat-dissolving solutions. iv) PAS staining is not positive. The foregoing findings further substantiate the assumption that the plug which is found inside the uterus after mating is formed by the secretion produced by the ejaculatory bulb. As to the nature of such a secretion, it may be assumed to consist mostly of fatty material; in point of fact, in view of the viscosity and compactness of the secretion, the latter may be presumed to be of a waxy nature. As to the functional interpretation of the plug, its homegeneousness and compactness would suggest a mechanical kind of function in the first place. If the plug were formed at the end of the mating, after the sperms have been introduced, the most obvious supposition would be that of the plug acting as an obstacle to the outflow of the sperms. As, however, it is found before sperms are introduced, its function is likely to be that of a factor favoring the travel of the sperms from the vagina to the spermathecae and to the seminal receptable. The fact should be remembered that Drosophila sperms are very long cells endowed with a spiral motion. A likely assumption is that the waxy plug works as a central axis which aids the sperm progress, forcing the sperms to swim between the surface of the plug and the walls of the uterus. Besides, by causing the uterus to dilate, the plug helps the sperms to reach the opening of the storage organs. The foregoing hypothesis is backed by observations performed with electron microscopy on uteri of females that had been separated 10 minutes after the beginning of mating. The electron microscope pictures demonstrated that bundles of sperms were located between the uterus walls and a homogeneous granular mass which fills the central portion of the uterus cavity. As far as the chemical function of the plug is concerned, no data are available at present that may either substantiate or rule out the

possibility of its containing such substances as may increase sperm motility or affect some unknown activity either of the sperms or of the female reproductive organs.

At any rate, the waxy plug may be regarded as a fertility factor. As a decrease in ejaculatory bulb secretion has been observed following repeated matings (1), variations in fertility rates may be caused not only by a reduction in accessory gland secretion (2) but also by inadequate activity of the ejaculatory bulb.

Finally, it must be definitely said that, on the strength of all the findings reported, the plug which is found in the uterus after a mating has simply nothing to do with the fluid secretion which Patterson (3) and other workers have reportedly noticed inside the genital duct of the Drosophila genus as a reaction to insemination. The fact must not be overlooked, indeed, that the plug is present after 5 to 8 minutes since mating beings, before any sperm is present and before any reaction is exhibited by the female genital duct's mucosa - and, more important still, the fact should be remembered that the plug is formed by the ejaculatory bulb secretion. This does not mean that a reaction to insemination may not occur, as noted particularly with interspecies matings, but merely that the waxy plug should not be regarded as the product of such a reaction. At this stage, two different assumptions should be investigated: either the waxy plug is the only material contained within the female genital duct of D. melanogaster besides the sperm after mating, or, together with it, the duct also contains the fluid secretion produced by reaction to insemination. Should the first hypothesis be verified, the plug and fluid secretion would be one and the same thing, and the actual existence of a secretory activity primed by insemination would then call for further investigation. As reaction to insemination is generally regarded as an effective selection mechanism in interspecies matings in the Drosophila genus, the finding we have just reported would seem to acquire a general biological and genetical significance as well as to warrant further, more systematic, investigations.

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Gateff, E. and H. A. Schneiderman. Case Western Reserve University, Cleveland, Ohio. Long term preservation of imaginal disc cell lines at low temperature.

When lines of imaginal disc cells with novel developmental capacities arise in the course of in vivo culture (Hadorn, 1965) one wants to maintain them for further study. To do this involves repeated subculturing in adults at intervals of one or two weeks. The time intervals can be lengthened to a month by implanting the tissue fragments into adults of D. virilis which are larger. But as more and more novel lines arise the investigator is forced to destroy certain lines because of the difficulties of keeping them continuously subcultured. We have modified a preservation technique originally designed to preserve bacterial cultures at low temperatures (Bouroncle, 1965).

The preservsng medium is a solution of 75% Drosophila Ringer's, 15% calf serum and 10% dimethylsulfoxide. One ml. of this solution is placed in a sterile ampoule. The adult abdomen containing the fragment of tissue to be preserved is separated from the thorax and cut open at the posterior tip. This leaves the abdomen open at both ends. The abdomen containing the imaginal disc fragment is placed in the vial which is then sealed in a flame and placed in a dry-ice-acetone bath at -80°C and then into a -78°C deepfreeze.

When the tissues are needed, the ampoule is thawed in a 40°C waterbath and then cut open. The abdomen is washed three times in Ringer's and the implanted tissues may now be used. These frozen tissues retain the capacity to grow when cultured in adult abdomens and to differentiate when implanted into larvae. The longest time tissues were kept at low temperatures was three and one-half months. When thawed, both the frozen implanted tissues and the organs of the frozen adult host abdomens appeared normal.

Hadorn, E. 1965. Brookhaven Symp. Biol. 18: 148-161. Bouroncle, B. A. 1965. Proc. Soc. Exp. Biol. and Med. 119: 958-961.

Lakovaara, S. and M. Sorsa, University of Helsinki, Finland. Distribution and the chromosomal characteristics of a newly described species, D. (Hirtodrosophila) subarctica Hackman.

The species in question was described from material captured in Finland in 1969 (Hackman, 1969 Notul. ent. 49: 69). Although a rather effective trapping for Drosophilids has been going on during the last few years in various parts of Finland, Drosophila subarctica has been captured only in the northernmost part of the country. Its southern line of distribution seems to be surprisingly accurate passing parallel to the Arctic Circle not more than about 20 kilometres southwards. The find locality farthest north was near to the northernmost point of Finland at Utsjoki, Kevo (69° 45' latitude). In this distributional area of D. subarctica the species is obviously rather common, as estimated from samples of several hundred individuals from 14 different trapping sites. Outside Finland, a find of a male individual has been made, apparently belonging to the same species from Northern Norway in Rosta (69° 00' latitude; Basden & Harnden, 1956 Trans. R. ent. Soc. London 108: 147). As yet D. subarctica is not known elsewhere.

The strictness of the southern line of distribution suggests that the species may need an uninterrupted illumination period of several days for its reproduction. D. subarctica seems to represent a "long-day" type of insect in relation to its photoperiodic response. This hypothesis is supported by some preliminary results obtained in experimental light-box cultivations of this species.

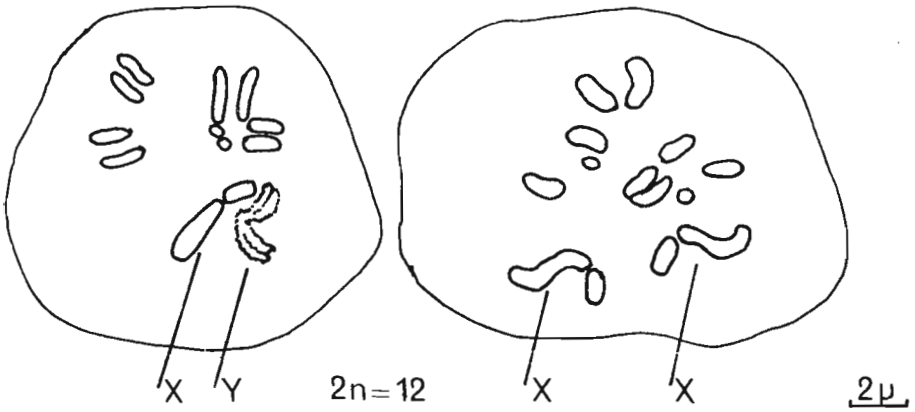

Fig. 1. Metaphase chromosomes from male and female larval ganglion cells of Drosophila subarctica.

The somatic chromosome number of D. subarctica, as determined from ganglion cells of third instar larvae is 2n=12, comprising of five pairs of autosomes and a sex chromosome pair. Four pairs of the autosomes are acrocentric rod chromosomes, while one is a dot chromosome pair. The sex chromosomes are the only ones in the chromosome complement with a median centromere, the chromosome X being submetacentric, while the primary constriction in the Y chromosome seems to be more precisely in the middle. The Y chromosome has a tendency for negative heteropycnosis in somatic metaphases of the ganglion cells. One pair of the acrocentric autosomes is slightly longer than the three other pairs.

The salivary cells of D. subarctica possess large nucleoli and beautiful and precisely banded polytene chromosomes. There are five giant chromosomes sticking out of the chromocentre which suggests that the Y chromosome and the short arm of the X chromosome are located in the chromocentre mass.

Ondřej, M. Institute of Experimental Botany, Prague, Czechoslovakia. Genetic effects of Edta alone and in combination with radiation.

Edta (ethylenediaminotetraacetic acid) showed synergical effect with radiation in the induction of dominant lethals in Habrobracon juglandis[1], and aberrations in the meiotic cells of Tradescantia[2]. Edta is known to increase frequency of crossovers in Drosophila females[3] but no other genetic effects on Drosophila were studied in detail.

We investigated the effects of Edta on aberrations, mutations and crossovers. In all experiments we used the injection application of Edta in 5mM concentration. This treatment caused temporary immobility of flies, which lasted 1-2 hours. If twofold concentration was applied, the toxicity was so high, that lethality immediately after treatment exceeded 90%.

Table 1
Frequencies of dominant lethals after treatment by 5 mM Edta and X-rays in the dosis 1 500 r.

Treatment	Eggs counted	% of unhatched eggs
Control	2,018	3.0
Edta	1,236	4.4
X	1,453	39.8
Edta + X	1,932	40.4

Irradiation by X-rays in the dosis of 500 r was carried out before injected flies recovered from the immobilizing effect of Edta. Two-day mating scheme was used throughout the experiments. The only exception was the series with dominant lethals, where only the first brood, lasting three days, was scored.

Dominant lethals were tested in the stock Oregon K. Edta induced just a very small frequency of unhatched eggs (1.4%), but its effect was quite independent of the effect of irradiation. Large fragments in the X-chromosome were tested by mating Oregon K males to attached $\overline{XX}$ y v f females. The frequencies of y^+, v^+, f^+ phenotypes and their combinations in F_1 females were scored. The effect of Edta was very slight. No indications of enhancement of X-ray effect by Edta were found. Crossing over in Drosophila females was scored in F_1 of the cross b cn vg x Suchumi. It is given only for the region b-vg. Edta, as

Table 2. Frequencies of large chromosomal fragments.

	X		Edta + X		Edta	
Brood	F_1 females	% of exceptions	F_1 females	% of exceptions	F_1 females	% of exceptions
I	3,596	0.25	3,547	0.25		
II	1,422	0.28	2,764	0.25		
III	450	0.89	1,259	0.64		
IV	434	0.00	417	0.24		
V	981	0.10	587	0.00		
VI	1,425	0.00	935	0.00	altogether	
VII	2,152	0.00	105	0.00	22,202	0.01

well as radiation, enhances strongly the frequency of crossovers, but when both agents act together, the resulting effect is rather smaller, than the sum of effects of the two agents. Crossing over in Drosophila males after Edta treatment was scored in F_1 of the cross dp b cn bw x Oregon K, in both spermatocytes and spermatogonia. There were 0.042% of crossovers in 16,504 individuals. Spontaneous frequency under similar conditions was 0.021%[4]. The differences between both frequencies were just on the verge of statistical significance. Edta did not induce any sex-linked recessive lethals. In all stages of spermatogenesis we scored altogether 3792 cultures and we get 0.24% of recessive lethals. Negative results are in agreement with earlier finding of other authors[5]. In our experiments after Edta treatment we found 0.02% of mutations in the dp locus between 10,776 individuals scored; the spontaneous frequency is in the verge 0.02% - 0.04% and therefore our results are negative.

Table 3. Frequency of crossovers in Drosophila females in the region b-vg.

	Control		Edta		X		Edta + X	
	p	s_p	p	s_p	p	s_p	p	s_p
I	10.19	0.41	10.05	0.83	15.03	0.59	15.65	0.63
II	11.82	0.39	12.85	1.06	12.52	0.60	12.15	0.54
III	9.10	0.33	13.00	0.92	12.11	0.67	11.20	0.46
IV	8.20	0.33	10.94	0.80	10.60	0.42	99.78	0.38
V	8.41	0.39	12.22	1.07	12.19	0.57	10.55	0.48
VI	7.59	0.33	11.50	0.90	8.69n	0.55	9.87	0.58
VII	5.87	0.34	6.01	0.79	8.88	0.54	9.67	3.86

Under our experimental conditions Edta increased the frequency of crossing over in the females, induced just a very low frequency of unhatched eggs, very low frequency of fragments of the X-chromosome and it induced a frequency of crossovers in males, which was on the verge of statistical significance. Edta never showed synergical effect with radiation; on the contrary the effect of both agents acting together was always a little bit smaller than the sum of individual effects of Edta and X-rays, acting on their own.

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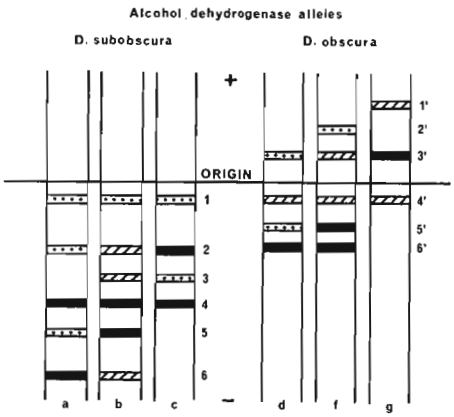
Saura, A. and S. Lakovaara, University of Helsinki, Finland. A study of alcohol dehydrogenase isoenzymes in D. subobscura and D. obscura.

We have recently studied the isoenzyme patterns of the D. (Sophophora) obscura group by means of starch and agar gel electrophoresis at pH 8.6. The flies are reared on the malt medium devised by Lakovaara (1969 DIS 44). ADH isoenzyme patterns are observed using ethanol or isopropanol as substrate. We have analyzed a total of 17 strains of D. subobscura collected from different natural populations in SW Finland and 23 strains of D. obscura collected from natural populations in Finland and N Norway along with strains of D. alpina, D. ambigua, D. bifasciata, D. pseudoobscura and D. silvestris.



The most common ADH pattern of D. subobscura is marked 'c' in the figure. It has two strongly staining bands at positions 2 and 4, and two very weakly staining bands at 3 and 1. The pattern 'c' has been found in all populations of D. subobscura studied this far, and it is identical with a pattern found in D. alpina and D. pseudoobscura. Like 'c', pattern 'a' also breeds true and it is found in two Finnish populations with pattern 'c'. The hybrid progeny of 'a' and 'c' shows the pattern 'b' (shaded bands 2, 3 and 6 are minor ones but stronger than the stippled ones.)

Patterns 'd' and 'g' appear to be identical with D. melanogaster Adh^{Slow} and Adh^{Fast}, respectively.

ADH of D. obscura shows two true-breeding forms 'd' and 'g', and a hybrid between these, 'f'. Pattern 'd' is found also in D. bifasciata and D. silvestris, whereas 'g' is found in D. ambigua. Most Finnish populations of D. obscura contain all three types, only four being homozygous for 'g'. Type 'd' has not been found homozygous in any population of D. obscura.

Moree, R. Washington State University, Pullman, Washington. Heterozygosis and segregation ratio in D. melanogaster.

In connection with the use of the Drop mutant (Dr; 3-99.2; homozygous lethal) as a marker, heterozygosity variations can be made high or low in the 3rd chromosomes of the marker type, in the 3rd chromosomes of their wild type competitors, and in the backgrounds of both. The eight resulting combinations (three factors, two levels each) were made by using the following four strains of flies: a Canton-S strain into which Dr was introduced by 35 generations of back crossing; a wild type strain collected at Wawawai, Washington on 27 September 1964; two derived strains, one having Canton-S 2nd and Wawawai 3rd chromosomes, and the other the contrary, constructed by the use of a double balancer, SM1/Pm;TM6/Dl^7. The X chromosomes consist of material from the balancer, Canton-S, and Wawawai lines in about a 4:1:1 ratio. The crosses, the eight heterozygosity combinations, the total number of flies for each combination, and the percentage of Dr carriers are summarized as follows:

1)	S/S;S/S x S/S;S/S^o ⟶ S/S;S/S^o and S/S;S/S	3162	47.94*
2)	S/S;S/S^o x W/W;S/S ⟶ W/S;S/S^o and W/S;S/S	3082	48.51
3)	S/S;S/S x S/S;W/S^o ⟶ S/S;S/S^o and S/S;W/S	2022	35.16***
4)	W/W;S/S x S/S;W/S^o ⟶ W/S;S/S^o and W/S;W/S	3012	47.01**
5)	S/S;W/W x S/S;W/S^o ⟶ S/S;W/S^o and S/S;W/W	2652	49.32
6)	W/W;W/W x S/S;W/S^o ⟶ W/S;W/S^o and W/S;W/W	3050	48.33
7)	S/S;S/S^o x S/S;W/W ⟶ S/S;W/S^o and S/S;W/S	2703	48.58
8)	W/W;W/W x S/S;S/S^o ⟶ W/S;W/S^o and W/S;W/S	3186	49.27

S indicates a Canton-S chromosome, S^o a Canton-S chromosome carrying Dr, and W a Wawawai chromosome. *, **, and *** indicate statistically significant deviation from 50% at the 5%, 1%, and 0.1% levels, respectively. Further X^2 tests show that combination 3 differs signifi-

cantly from all other combinations and that, aside from this, no other combination differs significantly from any other with respect to Dr carrier frequency. Sex and Drop phenotype frequencies were found to be independently distributed.

It is clear that changes in heterozygosity can change the relative viability of the Drop carriers, and hence the segregation ratio, but only under certain conditions; these conditions are summarized in the following table:

Combination	1	2	3	4	5	6	7	8
Drop type	(L:L)	(H:L)	(L:L)	(H:L)	(L:H)	(H:H)	(L:H)	(H:H)
Wild type	(L:L)	(H:L)	(L:H)	(H:H)	(L:L)	(H:L	(L:H)	(H:H)
% Drop type	47.94*	48.51	35.16***	47.01**	49.32	48.33	48.58	49.27

For a given type and combination, in parentheses, relative heterozygosity is given as background:3rd chromosomes and may be either low (L) or high (H) for a given category, i.e., (H:L) for Drop type of combination 4.

In combination 1, Drop frequency falls significantly below 50% while in combination 2 it is intermediate between that of combination 1 and 50% without being significantly different from either; the intermediacy ostensibly relates to the increased background heterozygosity in both types. In combination 3 Drop frequency is drastically reduced; interestingly, the difference between the total heterozygosities of the two types is proportionally greater in this combination than in any other. In combination 4 Drop frequency very significantly increases relative to combination 3; the difference between the total heterozygosities of the two types is proportionally less than in combination 3 owing to the increase in background heterozygosity. Combinations 5 and 6 might reasonably be expected to give results just the opposite of those of combinations 3 and 4, but obviously do not. The wild type flies of combinations 5 and 6 have 3rd chromosomes W/W, rather than S/S, as occurs in all other combinations in which the two members of a pair are from the same strain. Third chromosomes W/W and W/S appear to be about equal as far as viability effects are concerned. Why do the S and W chromosomes differ in this respect? Canton-S is an old laboratory stock, necessarily somewhat inbred, and has probably accumulated mildly detrimental mutations that would ordinarily be eliminated by the rigors of selection in nature. Wawawai, by contrast, is a new laboratory strain taken from a natural population about five years ago. It seems possible that such a difference may characterize a fair proportion of chromosomes taken from laboratory and natural populations. It is reasonable to suppose that if Dr were transferred to a Wawawai chromosome (now under way) the viability relations $W^o/W^o < W^o/S > S/S$ would obtain since the carriers of W^o/S would be highly heterotic. In combinations 7 and 8 Drop frequencies are intermediate, as in combination 2; and, as in combination 2, the total heterozygosities of the two types are essentially equal in both combinations.

Excluding the apparent effect of the Drop gene (or region) and the exceptional behavior of the W/W 3rd chromosomes, the simplest consistent explanation of the results, applicable to wide deviations from the theoretical 1:1 ratio (combinations 3 and 4) and to failure to depart significantly from it (combinations 2, 5, 6, 7 and 8) may be summarized as follows:

(a) If total heterozygosities of two coexisting types tend toward equality, their frequencies tend toward equality also, whether background heterozygosity is high or low; if background heterozygosity is higher, the tendency toward equality is slightly greater (combinations 1, 2, 7, and 8, exclusive of the Dr effect).

(b) If total heterozygosities of two coexisting types are unequal, the less heterozygous type has the lower frequency; the difference is more pronounced when background heterozygosity is low, less when it is high (combinations 3 and 4).

(c) Aside from the heterozygous effect of the Dr gene (or region), differences in segregant viability are correlated with differences between the total heterozygosities of the two segregants. Genetic background is effective to the extent, and only to the extent, that it contributes to the magnitude of this difference.

(d) The results depend as much on the distribution as on the mere quantity of heterozygosity, in a given combination.

(Aided by funds from the State of Washington Initiative Measure No. 171 for the Support of Biological and Medical Research. I wish to thank E.B. Lewis for TM6 and J.D. Krivshenko for Dr.)

Armstrong, C.E. Howard University, Washington, D.C. A thermostability study of octanol dehydrogenase isozymes in D. metzii and D. pellewae.

Following the study of octanol dehydrogenase (ODH) of D. melanogaster by Ursprung and Leone (1965) and Courtright, Imberski, and Ursprung (1966), this enzyme has been the object of extensive developmental and genetical analysis in the sibling species D. metzii, D. pellewae (Pipkin 1968, 1969 in press), and D. albirostris (Ogonji, this issue of DIS). To this date little work has been done on the characterization of ODH isozymes. This report describes the first in a series of experiments to characterize the ODH isozymes of D. metzii and D. pellewae.

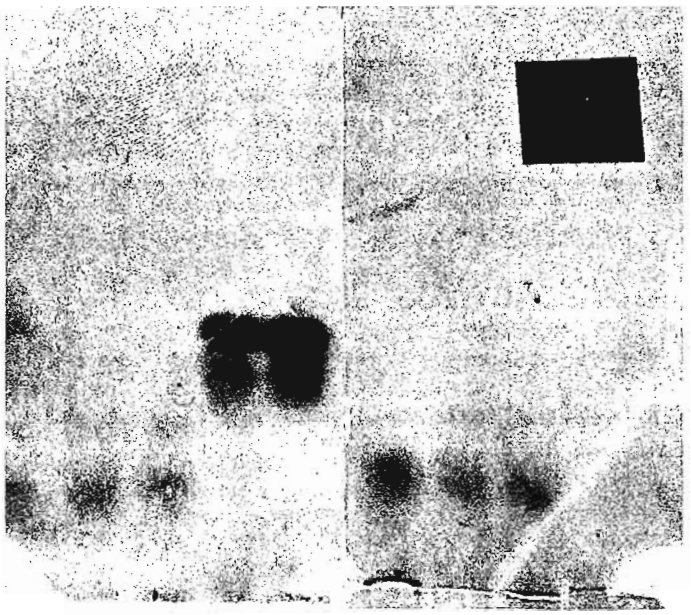


Fig. 1. Left, control ODH isozymes of homogenates of six day old adult females of true breeding D. metzii and D. pellewae strains; right, gel treated with 55°C for 35 minutes shows only the #1 isozyme still enzymatically active.

Differences in the thermostability of certain ODH isozymes separated by agar gel electrophoresis have been found in the crude homogenate obtained from four virgin females aged for six days, derived from eight different strains of D. metzii and D. pellewae. Known isozymic patterns of these experimental strains have been altered by timed exposure to high temperature ranges.

Experimental results have shown that the maximum thermal range of all the ODH isozymes was 55°C with a forty minute exposure time. At the same temperature, however, with a 35 minute exposure time, isozymes located at positions 1 and 2 were found to be heat stable and isozymes located at positions 3,5,6, and 7 were found to be heat labile (Fig. 1). No detectable difference in thermostability of isozymes at positions 3,5,6, and 7 has been observed. It is also noted that the thermal studies on third stage larval isozyme patterns agree with the results found in the adults.

The absence of a difference in the heat stability of isozymes at positions 3,5,6, and 7, and the finding of such a difference between the number 1 and 2 isozymes and all the other isozymes is in agreement with the duplicate gene hypothesis as outlined by Pipkin (1969 and her Fig. 1, this issue DIS).

This work was supported by National Science Foundation Grant GB 8770.

References: Courtright et al, 1966, Genetics 54: 1251-1260; Ogonji, G. 1969, DIS (this issue); Pipkin, S.B., 1968, Genetics 60: 81-82; Pipkin, S.B., 1969, Genetics (in press); Ursprung et al, 1965, J. Exptl. Zool. 160: 147-154.

Lim, J.K. Wisconsin State University, Eau Claire, Wisconsin. A selective system for testing reversibility of the sex-linked recessive lethals carried in males.

A genetic selection system for quick detection of apparent reverse mutations of the sex-linked recessive lethals utilizing the special Y-chromosomes and the attached X-chromosome has been tested. The results from a preliminary test indicate that the system works well in practice. Each of the sex-linked recessive lethals located at the proximal end and at the center of the X-chromosome, near the v locus, was made self-maintaining in males as follows:

lethals at the proximal end of the X-chromosome

y f:=/y^+·Y·ma-l^+ and l/y^+·Y·ma-l^+

lethals at the center, near the v locus, of the X-chromosome
y f:=/B^S•v^+•Y•y^+#1 and 1/B^S•v^+•Y•y^+#1

Of the eight lethals maintained in males, l(1)M41 induced by MMS in y w ct f X-chromosome was tested. The lethal was found to be located at 36.0 and the polytene chromosome of the stock appears quite normal. In testing the reversibility of the lethal, a large number of virgin females of the genetic constitution y f:=/y^+•Y•ma-1^+ was obtained from the cross between y f:=/Y and l(1)t2-14a/y^+•Y•ma-1^+ [l(1)t2-14a was induced in the Canton X-chromosome by W.D. Kaplan and was localized by him at 65.0] and were mated to y w ct f l(1)M41/B^S•v^+•Y•y^+#1. Of the 27,827 progeny, from the cross, were the following viable males: 7 w ct f males, 1 y w ct f male, 2 w ct f B males, and 1 fB male. The w ct f males and 1 y w ct f male represent apparent spontaneous revertants. Each of the seven w ct f males mated to y w ct f l(1)M41/FM6 produced y w ct f females indicating either a reversion of l(1)M41 or involvement of suppressor mutation for l(1)M41. The y w ct f male was sterile as expected. The w ct f B males were expected from non-disjunction in the males and the f B male can originate from separation of the y f:=. In addition to the above rare males were 11 f females, 59 y f females, and 1 y f B female. These rare females might have originated from non-disjunction in the females and/or separation of the attached X-chromosome.

A large number of f B virgin females (y f:=/B^S•v^+•Y•y^+#1) from the above cross can be mated to any of the lethals, in the proximal end of the X-chromosome, covered by the ma-1^+ segment of the y^+•Y•ma-1^+. In turn, a large number of f virgin females (y f:=/y^+•Y•ma-1^+) resulting from the above cross can be used to test the reversibility of the recessive lethals near the v locus carried in males with B^S•v^+•Y•y^+#1. Alternately introducing y^+•Y•ma-1^+ and B^S•v^+•Y•y^+#1, in this manner, into the eggs carrying y f:= should enable one to obtain a large number of virgin females, thereby providing an opportunity to test reversibility of sex-linked recessive lethals, covered by the v^+ segment of the B^S•v^+•Y•y^+#1 and those covered by the ma-1^+ segment of the y^+•Y•ma-1^+, carried in the males.

Mather, W.B. University of Queensland, Brisbane, Australia. Chromosomal polymorphism in D. rubida from Wewak and Oriomo River, New Guinea.

Samples of D. rubida from Wewak in the East Sepik and Oriomo River in the Western District of the Territory of Papua and New Guinea were taken in February and May 1968 respectively. The flies were collected from heaps of fermenting banana placed in rain forest. The flies were cytologically analysed by mating males to a standard inversion free laboratory strain and despermed females to males of the standard strain. In each case the giant chromosomes from seven larvae were scored (Mather 1961). The inversions recorded have been previously described: II LA, II RA, C and III A, B (Mather 1961), II RG, H, I (Mather 1966) and III H (Mather 1963). The most notable feature of the collections is the very high frequency of III A B H at Oriomo River.

	Wewak		Oriomo River	
Chromosome	♂%	♀%	♂%	♀%
II LA		13		
II RA	29	22		
C	76	72	50	91
G	6	22		
H	6			
I		6		
III A	18	6	100	100
B	18	6	100	100
H			100	100
Flies scored	18	16	8	11

References: Mather, W.B. 1961, Chromosomal polymorphism in D. rubida, Mather. Genetics Princeton 46: 799-810. Mather, W.B. 1963, Notes on the Inversions of D. rubida. D.I.S. 37: 104. Mather, W.B. 1966, New Inversions in D. rubida. D.I.S. 41: 125-126.

Browning, L. S. University of St. Thomas, Houston, Texas. Recessive lethals produced during oogenesis in D. melanogaster by ethyl methanesulfonate.

Females aged seven days or more were fed ethyl methanesulfonate in sucrose solution for 24 hours according to the method of Lewis (DIS 43: 193). This method of treatment should have insured the presence of one stage 14 oocyte in each ovariole. Two strains of females were used, one having approximately thirty ovarioles per ovary (Oregon R 60) and the other about twelve (Canton S). After treatment, ten bottles containing fifteen females each were mated to Basc males every other day for eight broods, and their F_1 virgin daughters mated individually to Basc males in order to detect recessive lethals in the X chromosome. Controls for the Canton S females showed a low spontaneous rate of 0.1% (4/4,407). The spontaneous recessive lethal frequency for the Oregon R 60 stock has not yet been measured. At the same time, Canton S males were treated and their daughters tested for recessive lethals by the Basc technique to confirm the mutagenicity of the chemical. The results are shown in the table below.

Broods (2-day)	Females: OR R 60 No.	OR R 60 L	OR R 60 %	Females: C S No.	C S L	C S %	C S Males No.	C S Males L	C S Males %	C S Controls No.	C S Controls L	C S Controls %
1	60	3	5.0	44	3	6.8	30	11	36.6	1,069	1	0.1
2	400	22	5.5	377	13	3.5	148	67	45.1	921	1	0.1
3	412	14	3.4	380	13	3.4	181	70	38.6	522	0	0.0
4	436	20	4.6	418	4	1.0	130	56	43.0	759	1	0.1
5	72	11	15.3	380	12	3.2	208	75	36.1	534	0	0.0
1-5	1,380	70	5.1	1,599	45	2.8	697	279	40.0	3,805	3	0.1
6	319	14	4.4	439	12	2.7	130	12	9.2	602	1	0.1
7	391	19	4.9	217	3	1.4	182	16	8.8			
8	202	8	4.0	-	-	-	64	1	0.1			
6-8	912	41	4.5	656	15	2.3	376	29	7.7	4,407	4	0.1

The continued appearance of recessive lethals through the sixteenth day after treatment shows that the mutagen is remarkably effective at all stages of oogenesis, including the germarium or oogonial cells. Since it takes approximately three days for an egg to pass from stage 1 to stage 14 at a maximum rate of egg-laying (R. C. King, 1957, Growth XXI, 95-102), eggs that were laid in broods 1 or 2 would probably have been in various stages of maturation at the time of treatment, and later broods would have been derived from cells that were oogonia at the time of treatment, although no egg counts were taken from individual females. Also, because of the mass mating of females, clusters or the presence of pre-existing lethals could not be detected. However, a record was kept of the lethals recovered from each bottle, and in both the Oregon R 60 and Canton S females the distribution of mutations and their frequencies were roughly similar. Even though the possibility exists that certain females retained their eggs much longer than others and so might have laid eggs that were in varying stages of sensitivity in broods subsequent to brood 2, it seems very unlikely that this would have persisted after brood 5, when they would have been 10 days post-treatment. It has been shown that the recessive lethal frequency in the second pair of autosomes is as low for eggs laid 10-15 days after acute irradiation with 4000r X-rays as for those laid later than 15 days after irradiation (Muller and Meyer, 1961, Genetics 46: 882). As will be seen from the table, a total of 56 lethals were recovered in 1,568 tests from the two types of females combined between the tenth and sixteenth days after treatment. Our average rate of 4.5% found in the Oregon R 60 females in these broods is double the $2.1\pm0.2\%$ found after acute X-ray irradiation of 4000r in very large-scale tests made by other workers (Muller, Oster, and Zimmering, 1963, Repair from Genetic Radiation Damage, Sobels, Ed., pp. 275-304). It might be pointed out, however, that this chemical produced from broods 1 through 7 far fewer mutations than are produced in the male germ line, in contrast to our finding that chloro-ethyl-methanesulfonate produces more mutations in the female than in the male germ line (Browning and Altenburg, 1965, Genetics 52: 431).

Parker has reported (1963, Repair from Genetic Radiation Damage, Sobels, Ed., pp. 11-19) that after X-irradiation, stage 7 oocytes are about one-half as sensitive as stage 14

oocytes when recessive lethals are measured but are one-tenth to one-twentieth as sensitive when hatchability is measured. He postulates that the increase in sensitivity of stage 7 oocytes with regard to recessive lethals may be due to an increased production of chromosomal aberrations, perhaps small deficiencies. It would be of interest to see if EMS behaves similarly when the same treatment techniques are applied.

Although an increase in recessive lethal frequencies is not shown clearly for each brood when Oregon R 60 females are compared with Canton S females, the table does indicate that a higher overall frequency for the Oregon R females probably exists, presumably due to the larger number of ovarioles. The use of this stock might then make the study of mutations arising in the female germ line less laborious.

Rai Chaudhuri, A. and A.S. Mukherjee. University of Calcutta, India. Developmental changes in puffing pattern in the mutant "ft" in D. melanogaster.

The mutant "fat" (ft;2:12.0) of D. melanogaster shows certain "vacuolar lipo-protein bodies" in the larval salivary gland cells (Slizynski, 1964; Rai Chaudhuri, 1968). This effect is accompanied by an initiation and increase in puffing activities in various sites of their chromosomes. Analysis of the sequential changes in the puffing pattern of these sites in ft during the late third instar to prepupae has been made and summarily presented below.

Table 1. Comparison of puffing activity in the wild type and ft third instar larvae and prepupae.

Group	puffing Sites	Oregon R+		ft	
		Larvae	Prepupae	Larvae	Prepupae
A	15CD	++	±	-	+
	18B	+	++	-	+
	53DE	+	++	-	+
	66D	+	±	-	+
	83EF	±	++	-	+
	85CD	±	±	-	±
	85EF	+++	++	-	++
B	42B	++	++	+++	-
	100EF	++	±	+++	±
C	7B	-	-	-	+
D	1A	-	-	+	+
E	2B	++	+	+++	++
	21B	++	+	+++	++
	61A	+	-	++	+
	74EF	+	-	+++	-
	75AB	+	-	+++	-
	47A	++	+	++	++
	50CD	++	+	+++	++
	72BC	+	+	++	+

Altogether 77 sites have been found to show activity during one or the other stages (from late third instar to prepupa). Among them, 42 were active during the late third instar, and the remaining 35 sites were active only during the prepupa; 23 puffs were active during both stages.

A comparative analysis of puffing patterns in ft larvae and prepupae with those in Oregon R+ shows (Table 1) that 7 puffs which are present either during the late third instar or prepupa in the wild type are absent in the ft larvae (Group A). Two puffs present in Oregon R+ larvae and prepupae are super-activated in ft larvae only (Group B). A single puff one each in Groups C and D is present either in prepupae (Group C) or in both stages of ft (Group D). Five puffs in ft larvae and three puffs in ft prepupae become more activated as compared to those in Oregon R+ (Group E). Three other puffs which are present in both stages of Oregon R+ and ft show a reduced activity in wild type strain as compared to ft larvae and prepupae (Group F).

Table 1. Legend:
+++ : Activity index 2 or more
++ : Activity index >1.6<2
+ : Activity index ~1.5
± : Activity index ~1.2 to 1.3
- : Activity index 1.0

References: Rai Chaudhuri, A., 1969, DIS 44: 118. Slizynski, B.M., 1964, Cytologia 29: 330-336.

Mukai, T., L.E. Mettler, and S.I. Chigusa. North Carolina State University, Raleigh, North Carolina. On the linkage equilibrium of isozyme genes in a Raleigh, N.C. population of D. melanogaster.

Three hundred and four second chromosomes were examined for alcohol dehydrogenase (ADH), α-glycerophosphate dehydrogenase-1(α-GPDH-1), and malic dehydrogenase-1(MDH-1). The frequencies of the fast alleles (F) are 0.237± 0.024 for ADH, 0.819±0.022 for α-GPDH-1, and 0.033±0.010 for MDH-1; hence, these three genes are located in the left arm. Using all chromosomes, no linkage disequilibrium was discovered between any two loci.

Two polymorphic inversions were discovered: Inversion A (breakage points are approximately 51-D and 57-A, and the frequency is 30/304) and Inversion C (breakage points are approximately 22-D and 33F - probably the same as In(2L)Cy but not associated with Cy with a frequency of 24/304). Although Inversion A is located in the right-arm of the chromosome, the associations between the inversion and the genes in question were examined. In ADH locus, F genes seem more associated with Inversion A than the chance, but not significantly so ($\chi^2_{d.f.=1}=3.45$, $0.05<P<0.10$). In the remaining two loci, no close association was detected.

With respect to Inversion C, a significant association was discovered between the inversion and S genes of the ADH locus ($\chi^2_{d.f.=1}=8.12$, $P<0.005$), although it is located outside the inversion (the distance is very small). This is linkage disequilibrium due either to some interaction between the inversion and the gene in question or to the lack of recombination between them. (The random genetic drift might not be significant because the effective population size has been estimated to be of the order of 10^4.) On the contrary, it was not possible to detect association between the α-GPDH-1 alleles and Inversion C although this locus is most probably located in this inversion. The MDH locus cannot be examined because of the low frequency of F genes.

Linkage disequilibrium was not detected using only the 241 completely inversion-free chromosomes.

Schalet, A. and V. Finnerty. University of Connecticut, Storrs, Connecticut. Is a deficiency for maroonlike lethal?

Recently, Lifschytz and Falk have presented 3 versions of a complementation map of the proximal region of the X chromosome of D. melanogaster in which the maroonlike locus as the only visible unit is a prominent feature. (DIS 1968; Mutation Research 1968, 1969). All three maps describe combinations of overlapping deletions yielding viable females that showed a "mal" phenotype. It was concluded that mal deficiencies were not lethal.

Because of our interest in the mal locus, Lifschytz and Falk were kind enough to send us four of their "mal" deletion stocks. We can report that none of the deletions have proven to involve the mal locus when tested against our extensive collection of viable and lethal mal mutants. We can confirm that females heterozygous for two of the deletions, A118/Q539 do survive and manifest a mutant phenotype. The eyes of these females sometimes display the mutant coloration as a large, irregular area. (The uneven distribution of pigment is clearly seen as a splotch in the eyes of pupae.) Adult females often have "material" protruding from the vagina and abnormal wings. Additional tests with non-mal mutants have located the locus in question to the right of mal and immediately proximal to lf. Salivary analysis by Lefevre shows that A118 and Q539 chromosomes carry deletions that overlap for at least band 19E7. The real maroonlike is located distal to lf, proximal to mel, and has been positioned cytologically at 19C4-19D3. (See note of Schalet, Lefevre and Singer in this issue.)

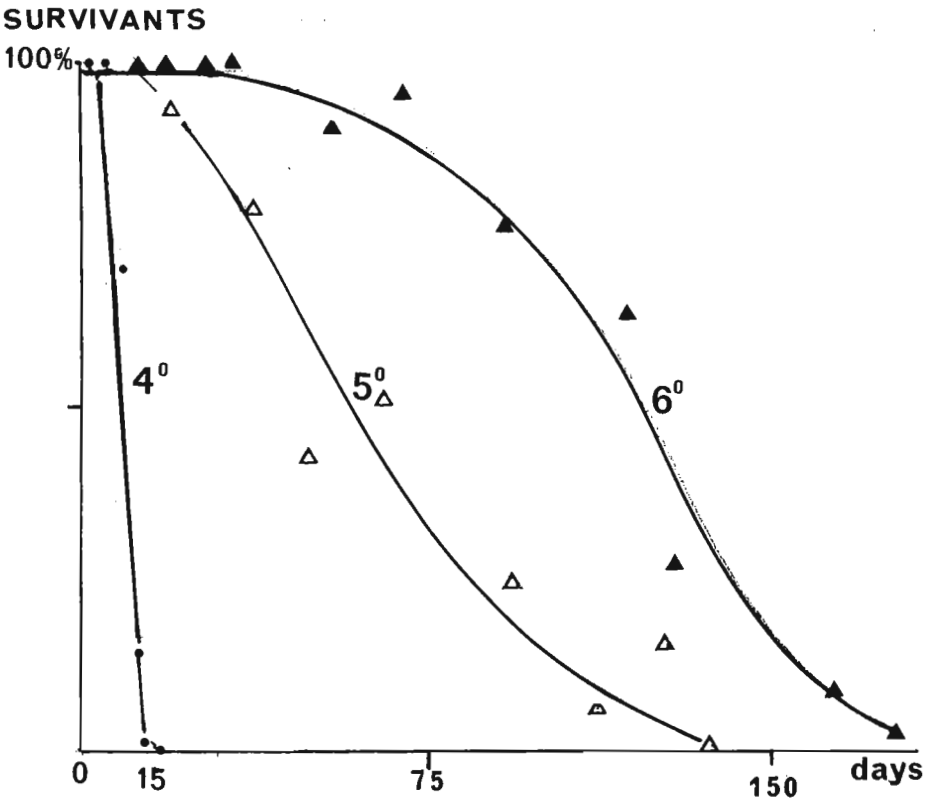
The question posed by the title of this note remains to be answered. Chovnick, Finnerty, Schalet and Duck (1969) have examined genetically 18 lethal mal mutants and all tested combinations have proved lethal. However, all lethal mal mutants behave like deficiencies in that each is lethal with at least one non-mal lethal locus adjacent to mal. Furthermore, all 7 deficiencies thus far examined by Lefevre show cytological deletions. Yet, the possibility that complete loss of mal alone may be lethal cannot be ruled out. Mal mutants lose the activity of three enzymes, xanthine dehydrogenase, aldehyde oxidase and pyridoxal oxidase. The loss of xanthine dehydrogenase activity alone is insufficient to produce lethality. At the rosy locus eye color mutants lacking only XDH activity are viable. Complete loss of the rosy locus is probably not lethal. This inference is drawn from the observation that the heterozygote, ry^{54}/ry^{74}, two probable overlapping deficiencies, is viable.

Anxolabehere, D. and G. Periquet. Faculté des Sciences, Paris. Cold resistance in D. melanogaster and its ecological implications.

The characteristics of natural populations of D. melanogaster stay generally the same from one year to another. But the problem of the survival of the flies at low temperatures is unknown. We are now looking at the cold resistance of adults.

One sampling of a natural population (M 68) originally from the French Mediterranean coast was kept at 4°, 5° and 6°C. The percentage of survival was scored from time to time. The curve (No. 1) made with both sexes, shows the great resistance of imagos to low temperatures. At 5°, a greater resistance of the females may be noted after 3 weeks; at 6°, this greater resistance appears only after 4 months. Two days after the end of the cold treatment, the fertility of the females was measured; it becomes 1/2 after 3 months, 1/3 after 4 months (Curve No. 2).

It is quite remarkable that the mean temperature of the coldest month in the area where the strain lives, is 7°C. So the resistance to cold of the strain allows it to survive during winter and recolonise in the spring. Nevertheless, it is still evident that this laboratory model must be tested in the field.



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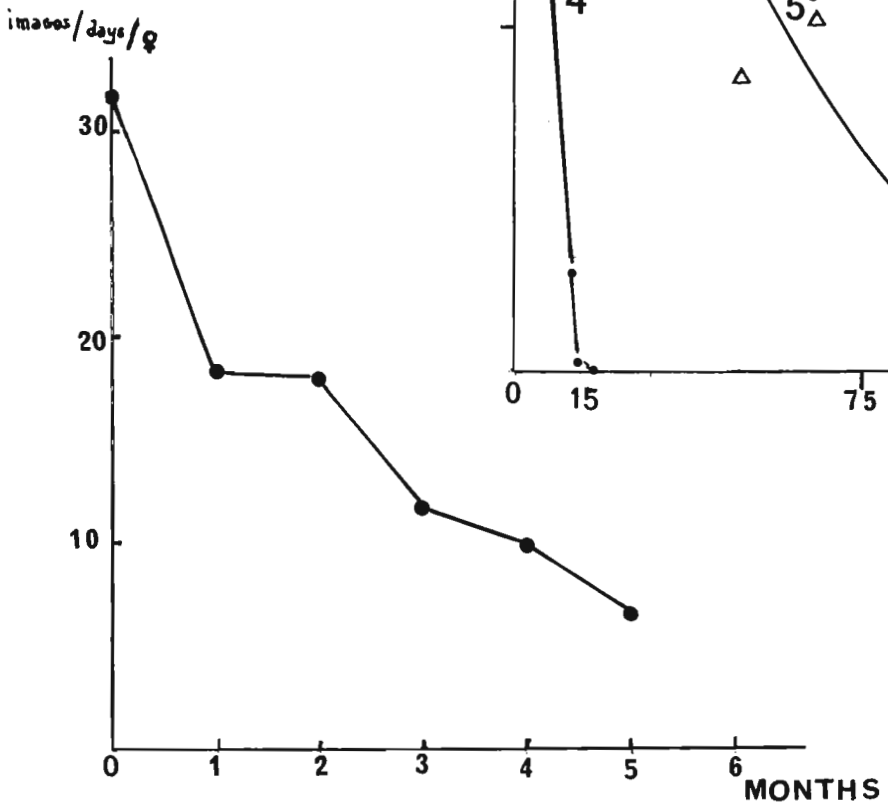


(2)

Voelker, R. and K. Kojima. University of Texas, Austin, Texas. Relative fitnesses of XO and XY males in D. affinis.

Miller and Stone (1962) and Voelker (1967) reported that XO males in D. affinis are viable and fertile. This indicates that no essential male fertility factors are present on the Y chromosome of this species. Since the Y chromosome is not necessary for male fertility, the possibility exists that XO males might be as fit as XY males. To test this possibility, population cages were set up in which the O and Y conditions were permitted to compete on two independently inbred (F=0.8) genetic backgrounds. The two genetic backgrounds were derived by eight generations of brother-sister pair matings of flies from a stock which was homosequential for all chromosomes, and carried a small Y chromosome. Subsequently, the O and large Y conditions were introduced into these backgrounds by five generations of backcrosses of males (either O or large Y) to females of the inbred backgrounds, which should have nearly restored the original degree of inbreeding. Two cages were started with each background, one with 75% O- and 25% Y-inseminated females and the other with 75% Y- and 25% O-inseminated females.

The frequencies of the O and Y conditions were determined by making larval ganglion squash preparations of male larvae taken directly from the cages. In all four cages the frequency of the O condition has decreased. This suggests that XY males are more fit than XO males irrespective of the background differences. One cage started with an O frequency of .75 became almost fixed for the Y condition at generation 10. The second cage, started with the O frequency of .75, still has the O frequency of about .10 at generation 14. Thus, there seemed to be some interaction among the backgrounds and the effect of large Y. The third and fourth cages, started with the O frequency of .25, became fixed for the Y chromosome before generation 10.

This work was supported by GM-15769, AT-(40-1)-3681 and 5 TO1 GM 00337.

Pelecanos, M. and A. Pentzos-Daponte. Department of Genetics, University of Patras, and Department of Biology, University of Thessaloniki, Greece. Rates of spontaneous autosomal recessive lethal mutations in D. populations of Northern Greece.

The present communication provides the first data ever collected in Greece on the frequencies of spontaneous lethal mutations in D. melanogaster populations. It is in this sense a preliminary report of an investigation which is undertaken in collaboration with other research workers.

The data presented here come from three non-isolated places in Northern Greece, namely: firstly, from the University farm in Thessaloniki, capital of Greek Macedonia, secondly, from the village Litohoron, which lies at the foot of the mountain Olympus 117 km. distant from Thessaloniki, and thirdly from the island of Thassos, which is approximately 25 km. S.E. of the port of Kavala (a town of eastern Macedonia, situated at a distance of 163 km. from Thessaloniki). In all three cases the flies were captured during autumn (September-October). Captured males were individually mated with virgin Cy L^4/Pm females in order to detect in each case the frequencies of second chromosomes bearing lethals.

Table 1. Rates of spontaneous autosomal lethal mutations

Locations	No. of parents tested	No. of chromosomes tested	No. of lethals	% lethals	% of parents which yielded lethals
Litohoron	130	648	149	22.99	92.0
University farm (Thessaloniki)	202	1,860	286	15.37	56.7
Island of Thassos (Limin)	18	274	20	7.30	50.0

Table 1 shows significant differences between the rates of lethals in all cases. Furthermore, tests for detecting reciprocal translocations between the II and III chromosomes indicate perhaps possible differences in different populations. (Litohoron samples had no translocations out of 2,680 gametes tested, while at the University farm we found 2 translocations out of 1,885 gametes tested.) Further investigation on the causes of the differences as well as on the identity of the lethals found are in progress.

Gethman, R.C. University of Chicago, Illinois. An age dependent, polarized effect on crossing over.

In a recent series of experiments designed to measure simultaneously crossing over and mutation in a particular X chromosome, a reduced frequency of crossing over was observed. As seen in Table 1, the frequency of recombination between yellow (y^2, 0.0) and singed (sn^3, 21.0) is reduced from an expected value of 21.0% to 14.8%, as measured in progeny from eggs laid on the first six days. In the second six day period, the frequency is not significantly different from the standard value. However, the frequency measured from 12 to 24 day old females was significantly higher. Note that the recombination frequency between singed and scalloped (sd, 51.5) did not deviate significantly from the standard map distance.

Table 1. Recombination frequencies in the cross of y^2 sn^3 sd/+ x y^2 sn^3 sd

	Age of the female parent (days)			
class	1-5	6-11	12-24	standard
y;sn	0.148	0.206	0.279	0.210
sn:sd	0.284	0.288	0.267	0.305
N	2062	1754	2542	

Subsequent crosses indicated that the effect was not a simple one, but was influenced by both X chromosomes. The results given in Table 2 are from a series of experiments designed to characterize this effect. The progeny from all of the crosses were counted for the first six days, and recombination was measured only between y^2 (or sc, 0.0) and sn^3 (or ct^6, 20.0). Ignoring cross #5, these results would seem to indicate that both the + and y^2 sn^3 sd chromosomes lower the frequency and operate in an additive fashion. In cross #5, an increase, rather than the anticipated decrease, was observed. Since male progeny are segregating for both signed and forked, a misclassification of the bristle phenotype is possible. However, since the heterozygous females were back crossed to y^2 sn^3 sd males, there should be no ambiguity in the bristle phenotype of the female offspring. Cross #5b lists only the female progeny, and the recombination frequency here is also high. Thus, it would appear that this increase in recombination is real, and is not due to any misclassification. The meaning of these results are not clear.

Table 2. Recombination frequencies between y^2 and sn^3 (crosses 1, 2, 5) or sc and ct^6 (crosses 3, 4).

	Age of female parent (days)				
Cross	1-2	3-4	5-6	average	N
1 y^2 sn^3 sd/+	0.128	0.103	0.168	0.129	1679
2 y^2 sn^3 sd/ORE-R	0.181	0.139	0.179	0.164	1415
3 sc ec cv ct^6 v g f/+	0.161	0.154	0.189	0.169	1489
4 sc ec cv ct^6 v g f/ORE-R	0.198	0.209	0.191	0.200	1254
5 sc ec cv ct^6 v g f/y^2 sn^3 sd	0.277	0.249	0.263	0.260	1925
5b (females only)	0.277	0.253	0.252	0.259	1000

The change in recombination frequencies cannot be due to viability; first, all the reciprocal classes were of similar sizes, and second, if it were due to viability, different classes would have to be lethal, depending on the age of the female parent. The effect is probably not due to any aberration, such as a small inversion, as all single recombinants in a cross of y z ec ct^6/+ were recovered. Finally, it should be noted that the crosses were made under standard mapping conditions, using newly emerged females. The females were singly mated to 4-6 males, and were raised in shell vials on standard cornmeal media.

The changes in crossover frequency seem to be restricted to the distal region of the X chromosome, and the direction of the change is dependent on the age of the parental female. It is not known whether this effect is also seen on the autosomes. The general behavior over the first six days is similar to that of polarized, meiotic mutants.

Kaufmann, B.P. and H. Gay. University of Michigan, Ann Arbor, Michigan. A single second chromosome carrying both the Cy and Pm markers resulting from crossing over between the In(2LR)SM1 Cy and the In(2LR)Pm second chromosomes of D. melanogaster.

In a study of the mutagenic properties of deoxyribonuclease, we have used the In(2LR)SM1, al^2 Cy cn^2 sp^2/In(2LR)Pm;H/Sb stock (Pm = bw^{V1}) for detection of reciprocal translocations between the second and third chromosomes. When virgin females having these markers are mated with treated males of a wild-type stock, four F_1 phenotypes are usually detected, namely, Cy;H, Cy;Sb, Pm;H and Pm;Sb. (Flies of each type are then tested individually to determine whether a 2;3 reciprocal translocation has been induced.) Occasionally, however, an F_1 fly carries two dominant (or the reciprocal recessive) markers in a single second or third chromosome, as evidenced by the detection of such phenotypes as Cy Pm;H, Cy;H Sb, or Pm;+ +. That these "unusual types" result from crossing over during oogenesis in Cy/Pm;H/Sb mothers has been deduced from cytological analyses of third-instar larval salivary-gland chromosomes of the progeny produced by mating F_1 Cy Pm males with Oregon-R wild-type virgin females. Analysis was restricted to the Cy Pm phenotype, since neither H nor Sb is associated with a gross chromosomal rearrangement.

The In(2LR)SM1 Cy chromosome is essentially metacentric, whereas the In(2LR)Pm chromosome is acrocentric. Sequences of rearranged subdivisions for each of these chromosomes (as reported by Lindsley and Grell, 1968, in Genetic Variations of Drosophila melanogaster) are given below. (The inserted asterisk denotes the approximate position of the centromere.)

21A-22A3/60B-58B1/42A3-58A4/42A2 * 34A1/22D2-33F5/22D1-22B1/60C-60F

21A-21C8/60D1-59E1/40F * 59D4/40F-21D1/60D2-60F

When these chromosomes synapse during meiosis, with their centromeres lying side by side, they should produce two large "inversion loops," separated by an intermediate region (encompassing roughly divisions 34 to 39) in which single exchanges can occur without producing dicentric or ascentric chromatids. Exchanges in the most distal subdivisions of 2L and 2R should also yield balanced, viable products.

Seven F_1 Cy Pm males were tested, but only four of the matings furnished viable progeny. They included in each case both Cy Pm and wild-type individuals. The patterns of banding in the Cy Pm salivary-gland chromosomes obtained from third-instar larvae could be determined by comparison with the band sequences in the normal wild-type second chromosomes of maternal origin. From such comparison we concluded that in the production of the Cy Pm chromosome one exchange had occurred in the 34 to 39 interval (mentioned above) and that another exchange had occurred at the left end in the 59F or 60A region. Diagnosis was based on the following considerations: the Cy Pm chromosome is acrocentric; the short limb often shows the 21EF and 22A subdivisions lying in contact with 60C; the 33F/22D and 40F/59D inversions are present in the long arm; the 42A/58A inversion of SM1 Cy is not included. Thus the tip of the Cy chromosome joins with a small piece of the left limb of Pm to form the short arm of the Cy Pm chromosome, whereas its long arm includes the proximal part of the right limb of Pm and the distal part of the right limb of Cy. The new order appears to be the following (in which X denotes a region of exchange):

21A-22A3/60B6-59F X 59E1/40F * 59D4/40F X 34A1/22D2-33F5/22D1-22B1/60C-60F

This sequence accounts for all the mapped bands, with the possible exception of small deficiencies between 22A3 and 22B1, and between 59D4 and 59E1. But the "deficiencies" may arise from our inability to identify precisely the points of breakage and recombination at these sites rather than from an absence of essential genetic material, since the fertile Cy Pm individuals gave rise to vigorous, fertile Cy Pm and wild-type progeny.

A total of 188 "unusual types" were found among 3453 F_1 flies whose fathers had been exposed to the action of DNAase dissolved in phosphate buffer, and 107 among 2019 flies - serving as controls - whose fathers had been treated with the buffer alone. The frequencies - in each case an overall value close to 5.4 percent, with 0.76 percent of the Cy Pm type - are much higher than those detected in our 1949 study, in which a Cy/Pm, ds^{33k};H/C Sb stock was used in screening for 2;3 reciprocal translocations induced by nitrogen mustard. Only a few "tandem dominants" were observed at that time; subsequent loss of the stock precludes presentation in this note of data about frequencies (and cytological characteristics of unusual types) for comparison with those given above for the SM1 Cy/Pm stock.

This work was supported in part by USPHS Research Grant GM-10499.

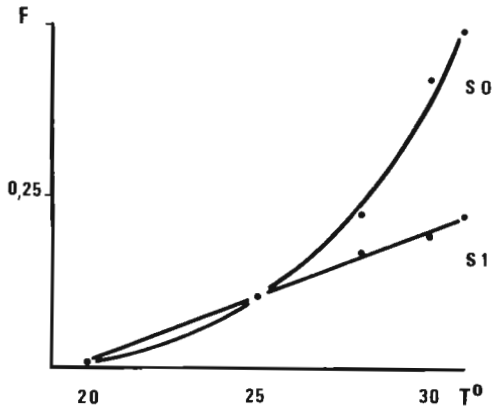
Periquet, G. Faculté des Sciences, Paris France. The maintenance of a semi-sterility factor in wild and experimental populations of D. melanogaster.

Penetrance and expressivity of ag character (see New Mutants) were investigated for their temperature dependency.

Six populations isolated from the same mutant strain were placed at different temperatures ranging from 20°C to 31°C (maximal non-lethal temperature for the strain). In every population there were flies with two normal gonads (S_2), one normal gonad (S_1) and none (S_0). The relative frequency of these types measured after one generation are given in the following table. (Curves No. 1 and No. 2).

	FEMALES					MALES				
T°	No. observed	S_1	S_0	Pene-trance	Expres-sivity	No. observed	S_1	S_2	Pene-trance	Expres-sivity
20°	210	0.014	0.014	0.028	0.50	250	0.024	0.004	0.028	0.14
25°	150	0.107	0.107	0.214	0.50	150	0.093	0.013	0.106	0.13
28°	65	0.169	0.231	0.400	0.56	61	0.181	0.082	0.262	0.31
30°	125	0.184	0.424	0.608	0.70	126	0.214	0.143	0.358	0.40
31°	217	0.221	0.489	0.710	0.69	266	0.233	0.252	0.485	0.52

One may see that in both sexes, penetrance and expressivity increase with the temperature of development. They are more important in females than in males (curves No. 3 and No. 4)



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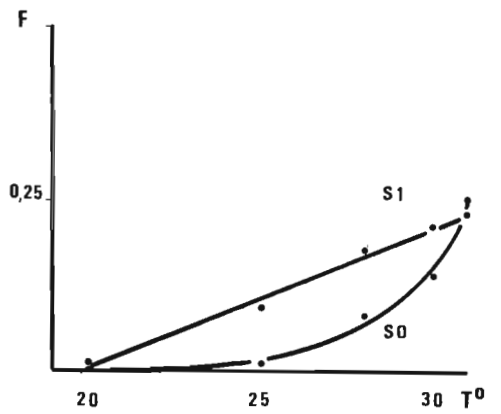

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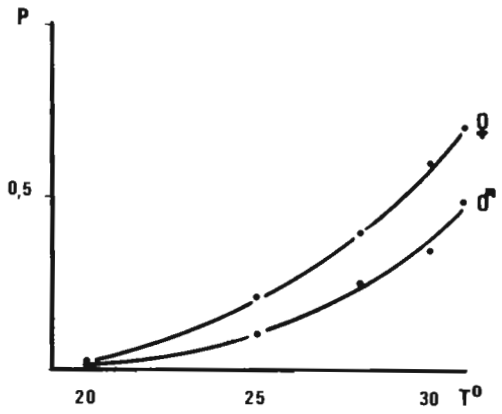

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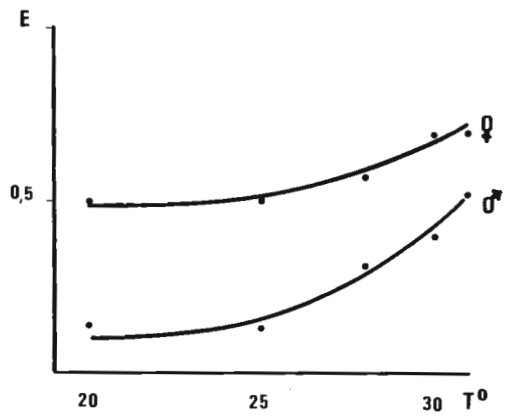


(4)

This character was seen to persist in a natural population during several years. As 20°C is nearly the environmental temperature of wild populations, the fact that both penetrance and expressivity are very weak at 20°C may partly explain the maintenance of the character.

Barr, H.J. University of Wisconsin, Madison, Wisconsin. Analysis of a putatively bb lethal Y chromosome.

When males from a laboratory stock of D. melanogaster homozygous for yellow and not known ever to have been exposed to a mutagen were crossed with C(1)DX females lacking the basal heterochromatin containing bobbed, no F_1 females were obtained. When crossed to attached-X females not lacking basal heterochromatin, such males gave progeny of both sexes.

Lines carrying the Y chromosome from the original, yellow stock were established by obtaining individual males whose Y chromosome had failed to rescue C(1)DX females at 25°C and crossing them to Oregon-R females. These lines are being maintained and studied. A total of 200 males randomly chosen from these lines proved fertile when tested individually. (Cf. Williamson, 1968, Genetics 60: 238) The yellow allele has been lost from these lines, probably by natural selection for the wild type.

These Y chromosomes are referred to as putatively bobbed lethal because the cross to C(1)DX females cannot rule out the possibility that the Y is deficient or mutant for a region of the basal heterochromatin that is necessary for survival other than the bobbed "locus". Thus Y chromosomes failing to rescue C(1)DX females may: (1) carry a deletion, mutation, or position-effect rearrangement involving the bobbed "locus"; (2) carry a deletion, mutation or position-effect rearrangement involving some part of the basal heterochromatin other than the bobbed "locus"; or (3) both (1) and (2). (Cf. Thompson & Braver, 1969, Genetical Res. 13: 325.)

Oregon-R and Canton-S stocks were tested for the presence of putatively bobbed lethal Y chromosomes by crossing singly 100 males from each to C(1)DX females at 25°C. No Y chromosomes that failed to rescue the F_1 C(1)DX females were found.

The pattern of "drift" toward wild type of the stocks carrying the putatively bobbed lethal Y chromosomes and the role of these chromosomes in suppressing variegating position effect have been studied and will be reported elsewhere.

(Supported by an N.I.H. Research Career Development Award, funds from the Graduate and Medical Schools of the University of Wisconsin, and an institutional grant from the American Cancer Society.)

Kinross, J. and A. Robertson. Institute of Animal Genetics, Edinburgh, Scotland. Egg laying and survival rates in population cages of D. melanogaster.

Our usual method of keeping population cages of D. melanogaster has been to add a pot containing 350 cc of standard agar food at weekly intervals, leaving each pot in the cage for 3 weeks at 25°C. The adult population then reaches a stable level of about 5,000. Attempts have been made to measure various characteristics of the life cycle using stocks containing marker genes substituted into a standard background. This involved putting known numbers of marked eggs onto pots in the cages at different times to measure survival to emergence and, by inference, the number of eggs laid on the pot in the cage. The results are as follows:

i. About 6,000 adults emerge from each pot. The average length of life of adult flies must then be somewhat less than a week.

ii. The number of eggs laid was highest on new pots (about 12,000 per day) and fell off as larval activity became greater. The average number of eggs laid by each female in her life time was around 20.

iii. The survival of eggs from laying to emergence was highest (about 40%) for eggs laid on the pot in the first two days but had declined almost to zero by the 5th day.

iv. It must follow from the rate of egg laying that, in order to maintain a stable population size, about 10% of all eggs laid will lead to adult flies.

v. The average weight of flies declined from an initial value of 1 mg to a minimum of 0.5 mg after 10 days of emergence and then increased once more.

vi. Since the average time from egg laying to emergence in these conditions is 15 days and the average length of life of adults is of the order of 7 days, it follows that the generation interval will be approximately 20 days.

O'Brien, S.J. Cornell University, Ithaca, New York. Functional and locational distinction between soluble and mitochondrial α-glycerophosphate dehydrogenase in D. melanogaster.

The α-glycerophosphate cycle of insects is critical in energy production, the intracellular NAD-NADH equilibrium, and in connecting carbohydrate and lipid metabolism (Sacktor in Physiology of Insecta, M. Rockstein, Ed., Academic Press, New York, ed. 2, 1965, p. 483). The two enzymes involved in the cycle in Drosophila have been the subject of investigation in our laboratory and this note is a preliminary report on our findings on the functional distinction between the soluble and mitochondrial α-glycerophosphate dehydrogenases.

Preparation of the soluble enzyme (αGPDH-1) involves mass homogenization of adults in .05 M Tris HCl pH 8.6 followed by precipitation of insoluble material by centrifugation at 30,000 g. The activity is recovered in the supernatant. Preparation of the particle associated enzyme (αGPDH-2) involves isolation of mitochondria by homogenization in .05 M phosphate pH 6.2 .001M EDTA, .38 M sucrose, followed by differential centrifugation between 500 g and 5000 g. The αGPDH-2 activity in the 5000 g pellet is particulate for the most part but can be solubilized by a variety of detergents, sonication, and enzymatic digestions. The most effective method is incubation of mitochondria with 1% Triton-X 100 for 2 hours, followed by centrifugation at 30,000 g. αGPDH-2 activity is found only in the supernatant after such treatment.

There are three general assays which we use to detect activity, (1) appearance of NAD at 340 nm, (2) reduction of PMS - INT read at 490 nm, (3) reduction of 2, 6-dichlorobenzenoneindophenole read at 600 nm. Qualitative electrophoretic detection employs only tetrazolium assays on cellulose acetate gels.

We can functionally distinguish between the soluble and mitochondrial enzyme by five different criteria. They are:

(1) Differential coenzyme specificity. αGPDH-1 shows a definite requirement for NAD in all assay procedures while αGPDH-2 show no activity dependence upon exogenous NAD. That the lack of coenzyme dependence does not depend upon mitochondrial impermeability to added NAD is demonstrated by identical independence of extracted "soluble" αGPDH-2 (see 2).

(2) Differential association of respective enzymes with the soluble and particulate fractions. Multiply washed mitochondrial preparations show no αGPDH-1 activity either spectrophotometrically (as determined by NAD stimulation) or electrophoretically (see 4). However, the soluble fraction always contains residual αGPDH-2 activity along with NAD stimulated αGPDH-1 activity (20x greater specific activity than αGPDH-2). This residual activity is presumably due to some αGPDH-2 which is normally soluble or solubilized by the isolation procedure.

(3) pH optimum - αGPDH-2 has a pH optimum between 6.1 - 6.4, while αGPDH-1 has an optimum above pH 9. These assays involve the oxidation of α-glycerophosphate.

(4) Electrophoresis - αGPDH-1 has a characteristic migration pattern which distinguishes electrophoretic variants on cellulose acetate strips in a .05 M Phosphate pH 7.4 system. Flies isolated in the absence of mitochondrial dissociating agents also show stain development of the origin independent of exogenous NAD. αGPDH-1 development depends on exogenous NAD. For a variety of reasons we think that the zone at the origin is the particulate αGPDH-2. Supernatant fractions show no development at the origin while mitochondrial preparations show only this development. Solubilized mitochondrial supernatant fractions show a variety of patterns depending upon the conditions of electrophoresis. None of these patterns which are seen in mitochondrial preparations correlate with those of the soluble enzyme. The former patterns can be detected in single flies and presumably represent αGPDH-2. The greatest homogeneity is detected with .05 M Acetate pH 4.8 at which pH there is inactivation of the αGPDH-1 enzyme.

(5) Presence of αGPDH-2 activity in αGPDH-1 deficient mutants. Four alleles of αGPDH-1 (O'Brien and MacIntyre DIS 43: 1968) which are deficient for αGPDH-1^B have been isolated by EMS mutagenesis. These have been tested and possess normal activity of αGPDH-2 as detected by electrophoresis and test tube assays.

Experiments designed to determine the genetic control of αGPDH-2 and to detect any genetic relationship between control of the enzymes are in progress.

This work was supported by Grant T1 GM 1035 from the National Institute of General Medical Sciences.

Zamburlini, P. and G.A. Danieli. University of Padua, Italy. A crylamide-gel electrophoresis of D. hydei proteins at different stages of larval development.

From cultures of synchronously developing larvae, samples were collected at different times of development. Larvae were collected in 2 M sucrose, washed twice in Tris-EDTA-Borate buffer and carefully dried on kleenex tissues.

The total soluble proteins of the larvae and the hemolymph specific proteins have been considered separately.

For the analysis of the soluble proteins, whole larvae were homogenized in 100 µl of the same buffer, containing 5% sucrose and P.T.C.

The omogenate was centrifuged at 20,000 x g for 15' and the clear supernatant was used as sample for the electrophoretic analysis as well as for the parallel protein content determination (Lowry et al. method).

For the analysis of the hemolymph proteins, larvae were dissected in a cold centrifuge tube containing 250 µl of the homogenization medium. The wall of the tube was washed with the same medium, up to a final volume of 0.5 ml. The tube was then centrifuged at 10,000 x g for 20' and the supernatant was considered as a dilution of the original hemolymph.

Acrylamide-gel electrophoresis was carried out in continuous buffer (Tris-EDTA-Borate, pH 9.4) at constant current (4 m A for tube); the run was stopped when the bromophenol front was at 1 cm from the lower end of the tube. Acrylamide-gels were stained overnight in acetic amido-black and then destained in 7% acetic acid.

Plate 1 reports the electrophoretic pattern of the total soluble protein content during the development from 52 to 196 hrs. calculated from the moment of oviposition at 24 hr intervals. Plate 2 reports the electrophoretic pattern of the hemolymph proteins in the same stages.

Acrylamide-gel patterns of total soluble proteins at different stages

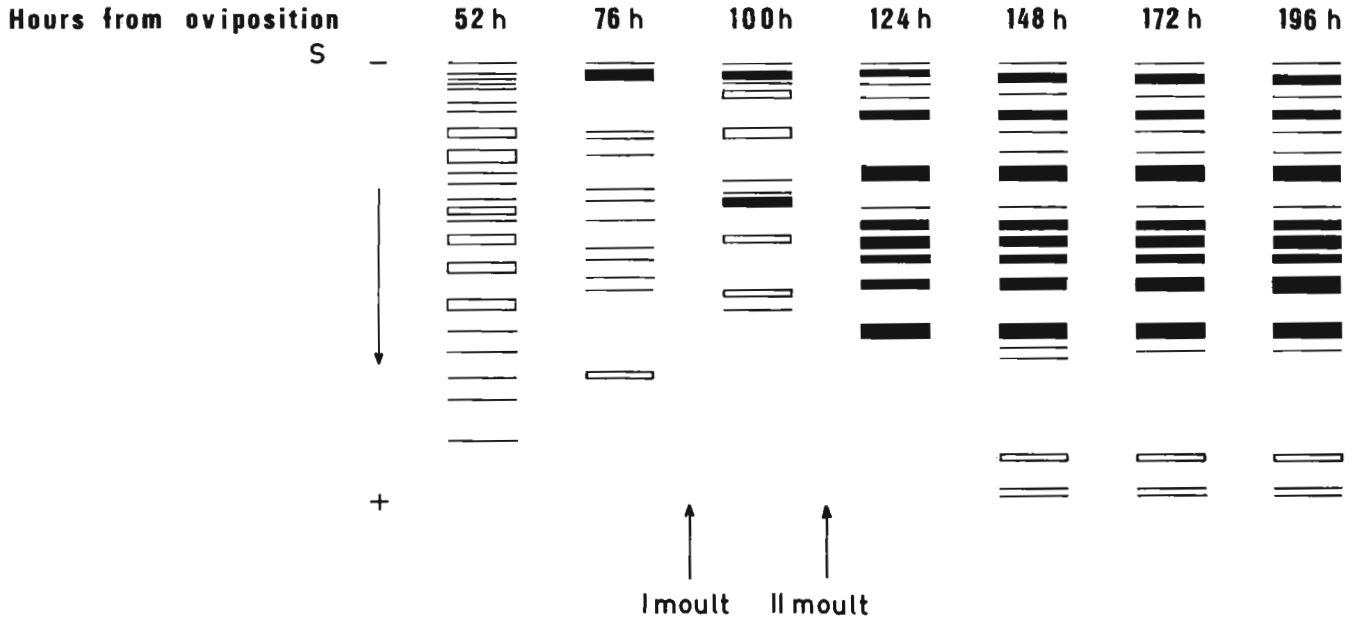


Plate 1

It is clear that the electrophoretic patterns under modifications during the development. In particular, it may be significant to note that some bands remain constant throughout the development (for instance the slow moving band, remaining near the cathode) while some other bands become visible in specific developmental stages; so the larval age can be recognized from the electrophoretic pettern of the larval proteins.

At the end of the development it is possible to identify at least 20 discrete bands. The

Acrylamide-gel patterns of hemolymph proteins at different stages

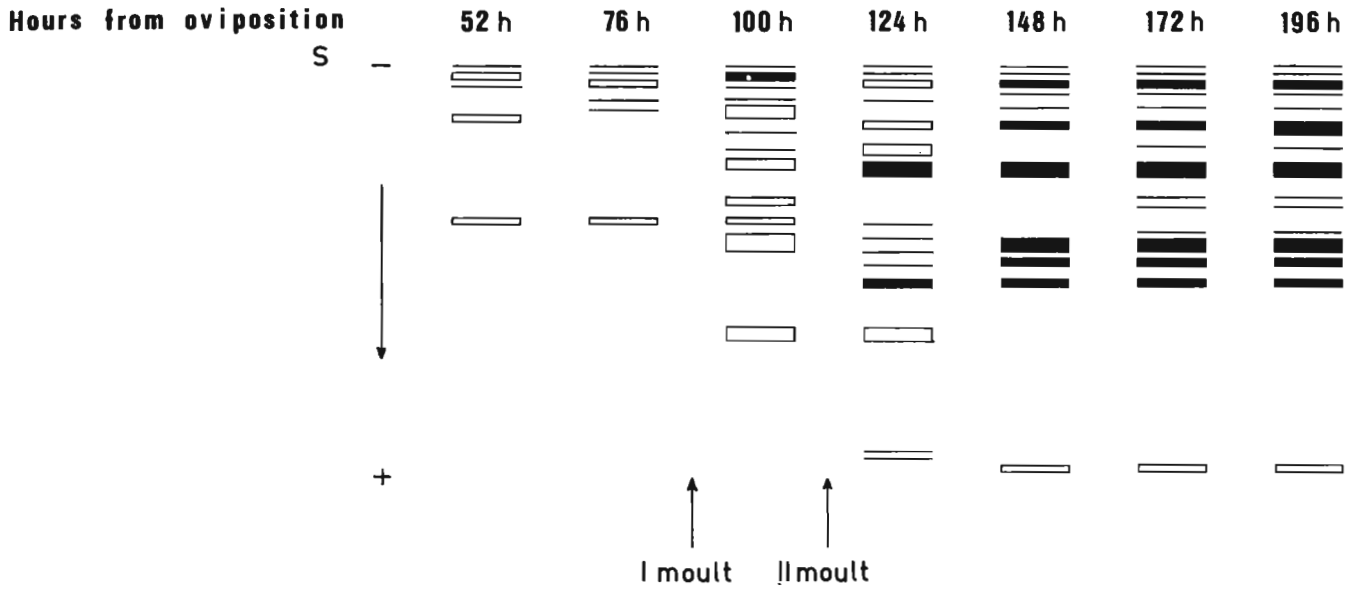


Plate 2

faster anodic bands seem to be somewhat depending upon environmental or experimental factors. They are always present but the relative concentration of their protein content may vary greatly.

References: Lowry, O.H. et al., 1951, Protein measurement with the folin phenol reagent. J. Biol. Chem. 193: 265-275. Raymond, S. and Weintrounb, L., 1959, Acrylamide gel as a supporting medium for zone electrophoresis. Science 130: 711.

Ortiz, E. Instituto de Genética y Antropología, Madrid, Spain. Drosophilids species in the Reserve of Doñana, Spain.

A first survey of Drosophilids species was performed in the Reserve of Doñana (recently established with the aid of the World Wildlife Fund) in the marismas and sand dunes near the mouth of the river Guadalquivir, in the south of Spain. Vegetation in the area is mainly constituted of shrub (Halimium, Ulex, Erica, Rubus), pine trees and cork oaks.

Flies were collected from May 13th to 16th in 1967, with 20 yeasted banana traps set up in six biotopes. Scaptomyza pallida was captured only by sweeping. The collected species were the following:

S. pallida	116	D. funebris	27
D. nitens	83	D. repleta	14
D. busckii	44	D. hydei	23
D. melanogaster	353	D. buzzatii	75
D. simulans	210	D. mercatorum	12
D. subobscura	547	D. immigrans	185
D. phalerata	59	D. cameraria	32

TOTAL 1780

Novitski, E. University of Oregon, Eugene. The concept of gamete dysfunction.

Recent interest in gamete dysfunction impels me to make some comments about the history of the term in Drosophila genetics. It first appeared in print, along with a detailed discussion of its possible importance, in a paper by Lindsley and Sandler (1957); this work, however, seems not to be generally appreciated, judging by the number of instances where reference to it would have been appropriate but was not made.

The initial observation of Iris Sandler, in her master's thesis, that the various segregation products from the Bar Stone translocation in the male were recovered with grossly disparate (but repeatable) frequencies, was moved from the level of a puzzling curiosity to an intriguing observation when it was realized, during a joint conference with Larry and Iris Sandler, that a remarkable mathematical relation obtained among the various classes. For the reader not familiar with this argument, a simple analogy will make the point clear. Take one set of alleles, A and A', which depress viability to a and a' respectively, and an independent set of alleles B and B', which depress viability to b and b', respectively. One would hardly expect in a cross where all four are present, and where the four combinations AB, AB', A'B and A'B' should be found equally frequently that they would appear in the arithmetical proportions ab, ab', a'b, a'b' with a precision to the fraction of a percent. For one thing, the synergistic interactions of viability effects should lead to gross departures from precise mathematical expectations. For another, there is no reason to postulate that viability effects are strictly multiplicative, as opposed to additive. A more rational guess might be that the net effect of two deleterious causes might depend on their interaction during development. Nevertheless, such mathematical agreement between the observed and calculated frequencies did exist, and has been later obtained repeatedly by Zimmering (1960), and Zimmering and Barbour (1961).

During discussions on this point during 1956 and 1957, I maintained that such precision must arise in some geometrical circumstance in gametogenesis, and was not likely to be caused by some biological malfunctioning, as inviability, infertility, dysfunctionality, unfertilizability, etc. In a paper that Iris Sandler and I published in 1957 we presented this argument and suggested further that if the available cytological evidence were correct, then the actual time of the effect would have to come after the spermatocyte divisions and during the spermatozoal stage. Another point of view (gamete dysfunction) proposed by Lindsley and Sandler during these discussions was that perhaps each sperm produced could be assigned a probability of functioning, p_1, determined by some one aspect of the chromosome complement, and that any other similar but independent aspect could be assigned another probability of functioning, p_2. The joint probability of survival would then be simply $p_1 p_2$. Considerations which seemed to me to argue against this proposal included the fact that in all cases where there was established an unequal recovery of two homologs that differed in size, it was the smaller that was more frequently recovered, independent of genetic constitution, and that when combinations of independent chromosomes were considered, the least frequently recovered were in some instances those that had the most balanced and complete, or normal, genomes. Irrespective of the specific point of view, however, perhaps the most significant feature of both ideas is that they unequivocally discarded the more trivial explanations based on zygote inviability, experimental error, etc., and pinpointed the basis of the phenomenon to the meiotic and prezygotic stages.

Some years later Peacock and Erickson (1965) concluded, from a comparison of the number of sperm stored and available for fertilization in a female with the actual number of progeny produced by sisters of such females, that only half as many progeny were produced as there were sperm present. This lead to the suggestion that half of the sperm were functional, and half non-functional, a positive answer to the question, "are all products of meiosis regularly functional?". This latter point has recently been questioned by Zimmering and Fowler (1968), and Fowler (in press), who find in experiments patterned after those of Peacock and Erickson that in some cases as many as 75% of the sperm present in one group of females may be represented by progeny from their sisters, and that, furthermore, the results from their tests appear to be subject to such great variability as to make any conclusions from such experiments suspect. In any case, the hypothesis of the regular non-functioning of a fraction of the products of meiosis has been consistent with the observations of Peacock that there were no gross cytological abnormalities at any stage in the meiosis of segregation distorter males, that the sc^4-sc^8 chromosome shows no meiotic loss, but is recovered with frequencies deviating from expectation, that sex-ratio in D. pseudoobscura does not exhibit any gross meiotic abnormality (like a precocious replication of the X

chromosome) as was previously thought, and that the distribution of "granules" (micro-organisms) is non-random with the segregation of the sc^4-sc^8 chromosomes amoung the secondary spermatocytes.

Within the past several years the question of dysfunction has been reopened by the work of Hartl, Hiraizumi and Crow (1968), in which they show that there is an initial decreased fertility of segregation distorter males, roughly proportional to the excess recovery of the SD chromosome over its normal homolog, and interpret this, as well as the decreased lifetime productivity of SD males, as manifestations of sperm dysfunction. In view of this, the reappraisal of the behavior of the Bar Stone translocation becomes of considerable interest.

The fertility of B^S males cannot readily be compared with their wild-type sibs because of the profound difference that might be based in the different phenotypes. Preliminary comparisons of wild type males and Bar males, both with B^S males, raised in complete darkness, except for the few minutes necessary for daily remating, indicated that the translocations males were quite infertile.

The mutational occurrence of a phenotypically normal eye in a Bar^S stock has made it possible to compare the fertility of translocation and non-translocation-bearing males independent of the usual phenotypic manifestation of Bar eyes. Translocation males were mated to Oregon-R females; F_1 females were mated again to Oregon-R males. Their progeny should consist of two types of males, translocation and non-translocation, phenotypically indistinguishable. These males were mated to six or seven cn bw ♀♀ each day for a total of 27 days, (subcultures after the first were assigned the letters of the alphabet, necessitating the termination of the experiment after twenty-six-plus-one days). To obviate any complications arising from hidden defects in the vision of the Bar Stone reverted males, all cultures were kept in complete darkness, except for the short period when the females were changed each day.

		Age of male in days					
		1-5	6-10	11-15	16-20	21-25	26-27
Total Progeny	+	26,942	20,875	7,469	2,403	1,554	742
	B^S	1,705	1,142	199	36	0	0
Fertile ♂ days	+	83	70	41	18	11	6
	B^S	54	37	6	4	0	0
Progeny/♂/day	+	325	298	182	133	141	124
	B^S	32	31	33	9	0	0

For ease of presentation the data are clumped into five day periods, except for the last two. Fertile ♂ days refers to the number of fertile ♂♂ times the number of days the males produced offspring during the five day period in question. The significant rows are, of course, the last two, which give the average number of progeny per fertile male per day.

The table shows a great difference between the fertility of the translocation male and its wild type sib, a difference much too great to be accounted for by the production of inviable aneuploid zygotes. While it cannot be denied categorically that the translocation males are less fertile because the translocation has accumulated sterility factors independent of the translocation itself, it seems much more likely that a phenomenon like sperm dysfunction is responsible for the low fertility. It should be noted, however, that the pattern of infertility is strikingly different from that of segregation distorter, since the latter appears to be of normal fertility during most of its fertile period.

References: 1. Hartl, D., Hiraizumi, Y., and Crow, J.F. 1967. Evidence for sperm dysfunction as the mechanism of segregation distortion in D.m. Proc. nat. Acad. Sci., Wash., 58: 2240-2245. 2. Lindsley, D.L., and Sandler, L. 1957. The meiotic behavior of grossly deleted X-chromosomes in D.m. Genetics, 43: 547-563. 3. Novitski, E., and Sandler, I. 1957. Are all products of spermatogenesis regularly functional? Proc. nat. Acad. Sci., Wash., 43: 318-324. 4. Peacock, W.J., and Erickson, J. 1965. Segregation-distortion and regularly nonfunctional products of spermatogenesis in D.m. Genetics, 51: 313-328. 5. Zimmering, S. 1960. Modification of abnormal gametic ratios in D. I. Evidence for an influence of Y chromosomes and major autosomes on gametic ratios from Bar-Stone translocation males. Genetics, 45: 1253-1268. 6. Zimmering, S., and Barbour, E. 1961. Modification of abnormal gametic ratios in D. II. Evidence for a marked shift in gametic ratios in early vs. later sperm batches from A-type Bar-Stone translocation males. Genetics, 46: 1253-1260.

David, J. and R. Ramousse. Laboratoire d'Entomologie Experimentale et de Genetique, Lyon, France. Quantitative evaluation of liquid food intake by Drosophila.

It is well known that adult Drosophila can be fed with a liquid food contained in a capillary tube. This method is often used for giving them rare or dangerous chemicals. A modification of this technique has been worked out in order to get daily measures of the volume ingested. A description of the method and some indications upon the first results are given here.

Method: The essential characteristics are indicated on the figures. Two calibrated capillaries, about 2 cm length (Drummond Company) are disposed vertically through the upper surface of a cage of plastic material (9 x 6 x 5 cm), as indicated in figure A. From preliminary studies, fragments from capillaries of a volume of 50 microlitres and 10 cm long proved to be the most convenient. Thus a length of 2 mm of liquid corresponds to one microlitre.

The capillaries are held in the cage holes by pieces of rubber tubes of appropriate size (figure B). One of them contains the liquid accessible to the flies. The opening of the other, which is used as a control for evaporation, is protected by a wire-gauze. In order to prevent the liquid from flowing out the capillary, its external surface is covered with grease, around the lower opening.

In such a device, evaporation has to be reduced to a minimum if accurate measures of liquid intake are needed. Therefore, 4 or 5 such cages are placed into a large box, the bottom of which is filled with a layer of water.

As the daily liquid intake of a fly is very small, it is better to have several in each cage. Groups of five flies were most often used.

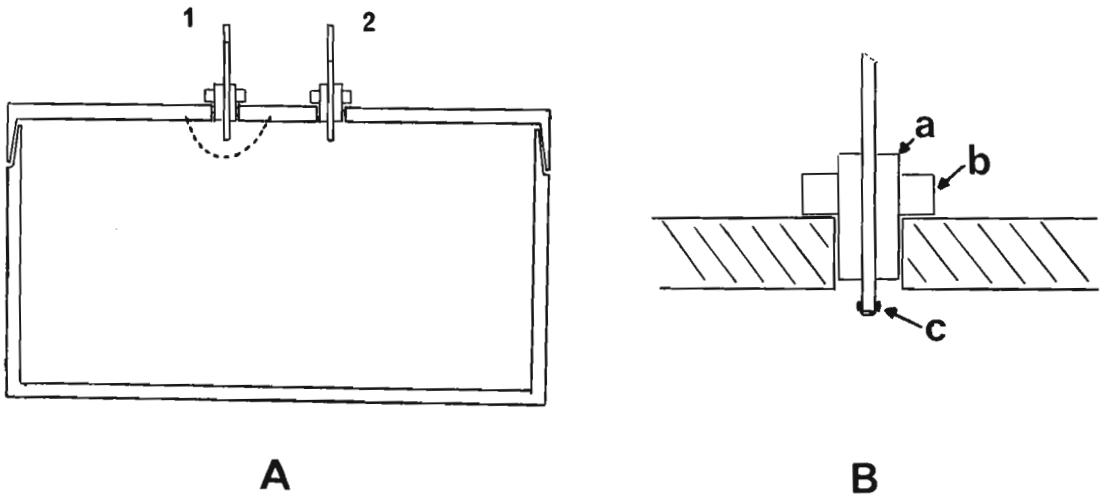


Figure A: Transversal section of an experimental cage (1: control capillary for evaporation; 2: capillary accessible to the flies).
Figure B: Detail of the insertion of a capillary (a and b: pieces of rubber tubes holding the capillary; c: grease around the lower opening).

Results: Sucrose solutions, at concentrations ranging from 4 to 14% were used as food, and 0.1% of nipagine was added to prevent bacterial development. 69 groups of flies (males or virgin females) were studied from emergence for at least 20 days.

The first striking observation is the very high variability of the results, either between successive days or between groups of flies. From a preliminary analysis, it appeared convenient to consider the average value obtained with each group of flies during 20 days as a single observation. From these data, the calculation of the mean evaporation in the control capillaries was 3.21 ± 0.18 µl and the extreme values were 0.8 and 6.95 µl.

The mean daily consumption, calculated after substracting the evaporation values and expressed as g of sucrose ingested by a fly in a day, is:

males	24.36 ± 2.26	n=25	(extreme values 5.6 and 43.6)
females	30.60 ± 2.30	n=44	(extreme values 1.6 and 75.8)
both sexes	28.34 ± 1.71	n=69	

Although the daily intake is a little higher in females than in males, the difference is is not significant, probably because of the enormous variability of the results.

It was supposed that the flies, ingesting for unknown reasons a very low quantity of sucrose, would take an insufficient amount of food and die prematurely. To check this hypothesis, the flies were divided into 3 groups, according to the amount of sucrose ingested, and the percentage of martality at 20th day was calculated as indicated below.

low consumption group	(from 1.6 to 20)	mortality percent : 29.9	n = 87
middle " "	(from 20.1 to 40)	" " : 24.2	n = 157
high " "	(from 40.1 to 75.8)	" " : 27.8	n = 54

From this it appears that there is no correlation between survival and quantity of sucrose ingested. In other experiments, where flies were fed in a usual way with a sugar-agar medium, the mortality at 20th day ranged from 12.50% to 47.50%. Thus feeding the flies with a solution in a capillary probably does not reduce longevity. However, if only water is given to flies, their mean survival is only 4 or 5 days.

Two other observations may be indicated here, although they are to be considered only as preliminary conclusions.

First, by pooling the whole data, a small, progressive decrease in sucrose ingestion was observed, from the beginning to the end of the experiment. It is therefore supposed that aging reduces food intake.

Second, the study of the influence of sucrose concentrations gave different results according to sex. In females, increased concentrations result in an increase in the quantity of sucrose ingested. In males, however, the mean daily consumption was quite stable, over the range of concentrations from 4 to 12%. Of course, such a stability corresponds to an important variation in the volume of ingested liquid.

These experiments are still in progress and various improvements are being tried in order to reduce evaporation and to improve the accessibility of the nutritive liquid to the flies.

Tsacas, L. C.N.R.S., Gif-sur-Yvette, France. Some data upon the morphology and biology of D. picta Zett.

The breeding of D. picta was carried out in 1962, from flies captured in Brittany (France). Since then, its morphology and biology have been studied in our laboratory; some of their particulars are given here.

The egg shows two pairs of filaments; the upper one is slightly shorter and more tapered at the extremity. The mature larva shows, on its terminal segment, six pairs of tubercules, plus an odd median anal tubercule: dorsals very small, dorsolaterals, ventrolaterals and ventrals very big, anals, plus one smaller median, siphonals. The anal plate (circumanalis) is narrow and elongated.

The pupa, ochraceous-yellow, is 3.4-3.7 mm long, respiratory horns not included. Horn-index is 5-8.4 mm (M = 6.1).

Wing-indexes: costal-index 3-3.53; 4th vein-index 1.23-1.61; 4c-index 0.61-0.94; 5x-index 0.87-1.14. Sterno-index 0.8-0.88. Testes almost colourless, big, with only one coil; ejaculatory sac with two diverticulae. Ovaries with 12-20 ovarioles. Spermathecae small, almost spherical. Ventral receptacle with 4-5 coils. Malpighian tubes joined in two pairs, common trunks short; the anteriors free, the posteriors united, with common lumen.

The length of the cycle, from egg-laying to hatching of the imago, is 20-29 days (M = 23) at a temperature of 20°C. It is thus decomposed: egg, 24-48 hours; larva, 7-15 days (M = 13); pupa, 5-8 days (M = 6). At a temperature of 25°C, the length of the cycle is reduced to 13-20 days.

Appropriate experiments allowed us to make the following observations: there is a very long lag between the hatching of the adult and the first egg-laying, a relatively short length of life, and a restricted fecundity (315 eggs layed during 68 days of life).

Chromosomes: metaphase plate shows 2n = 12. Those from the salivary glands show 4 long arms and 1 dot.

Schwalm, F.E., H.A. Bender and D. Klingele. University of Notre Dame, Indiana. Ultrastructural organization of the eggs of the female sterile mutant-lz^{61f} (D. melanogaster).

Ultrastructural organization of eggs from the lz^{61f} mutant one day after oviposition was studied. The distribution of components appears to be highly disorganized as compared to that in normal eggs (King 1960, Okada and Waddinton 1959). Mitochrondria, which are distributed evenly throughout the wild type eggs, are localized heterogeneously in lz^{61f}/lz^{61f} eggs. Similarly, the floccular beta spheres have merged and their contents form large pockets. These pockets are surrounded by a single layer of mitochondria (fig. 1). Alpha spheres are restricted to distinct regions of the egg where almost no other components are found. Large spaces are filled with endoplasmic reticulum which frequently assumes the form of annulate lamellae (fig. 1). Polar granules, similar to those found by Mahowald (1968) and in Coelopa eggs (currently under investigation in our laboratory), occur in different areas inside the egg, remote from the posterior pole and widely separated from each other (fig. 2).

These observations suggest that disorganization of the egg at termination of oogenesis could account for the lack of development. A more detailed study of oogenesis in this mutant has been initiated.

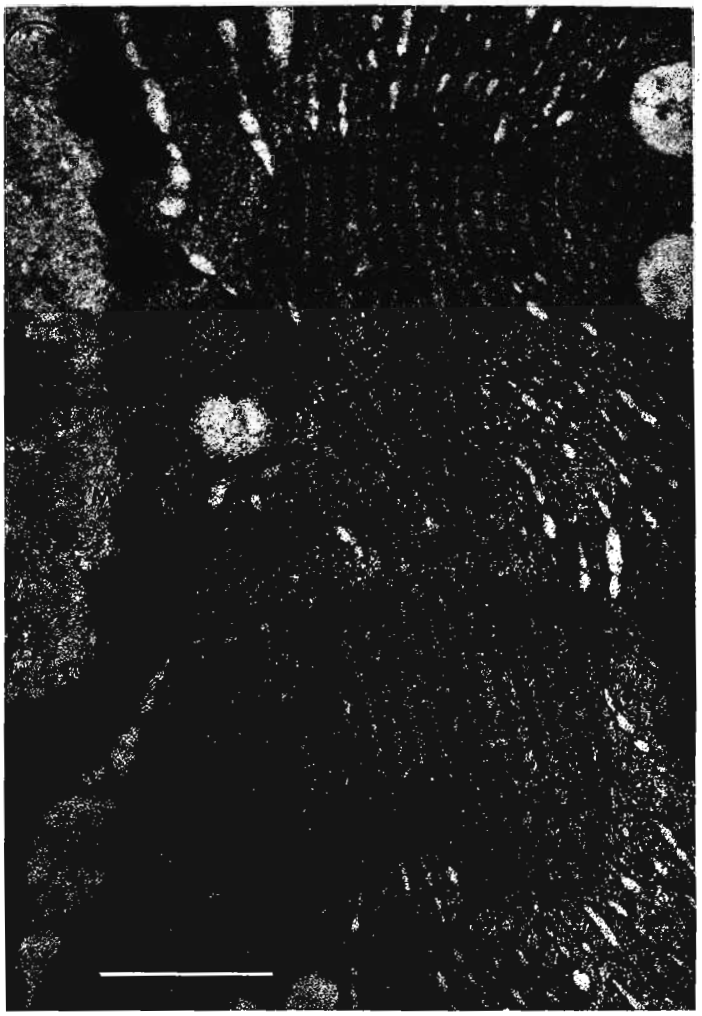


Fig. 1. Electronmicrograph from a longitudinal section of a lz^{61f} egg. Row of mitochondria (M) on margin of floccular space. Extensive cytoplasmic area, rich in endoplasmic reticulum (ER) continuous with annulate lamellae (AL).

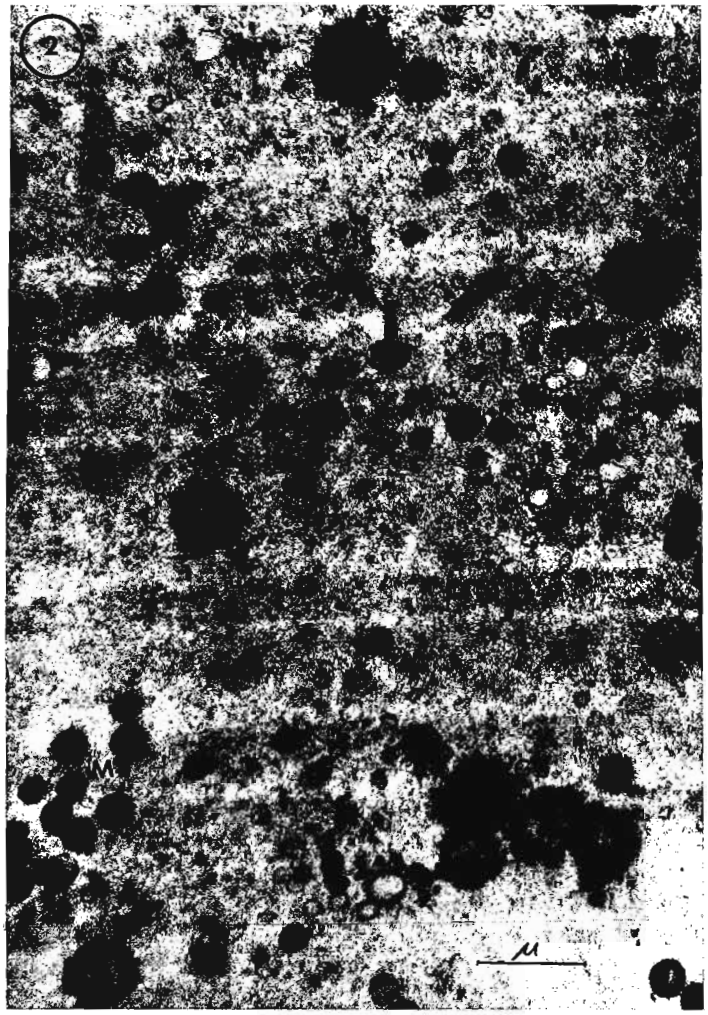


Fig. 2. Electromicrograph from central region of lz^{61f} egg. Granular bodies (Gb), presumably identical with polar granules in wild type eggs. Mitochondria (M).

References: King, R. 1960, Growth 24: 265. Okada, E. and Waddington, C.H. 1959, J. Embryol. Exp. Morphol. 7:583. Mahowald, T.P. 1968, J. Exp. Zool. 167: 237.
This is AEC Document No.COO-38-701.

Schwinck, I. University of Connecticut, Storrs, Connecticut. Phenogenetic inhibition and enhancement of drosopterin formation in various mutants of D. melanogaster.

Earlier it was found that the concentration dependent phenocopy effect of the xanthine dehydrogenase inhibitor 4-hydroxy-pyrazolo(3,4-d) pyrimidine (HPP) on drosopterins in cinnebar (cn) eyes decreases the amount of drosopterins slightly below the level of the rosy (ry) strain but never below 10% of the cn control value. We now asked the following: Is a low level drosopterin synthesis uncontrollable by the xanthine dehydrogenase metabolites? Or will these metabolites decrease the drosopterin synthesis further in other eye color mutants which normally have a rather small amount of drosopterins and a functional xanthine dehydrogenase? Therefore, HPP was fed to larvae of a control cn strain and of the following eye color mutants: claret (ca), orange (or^{66k}), pink-peach (p^{p}), raspberry (ras^{2}), and rosy (ry^{2}); all strains also contained cn in order to block the ommochrome synthesis and thus to facilitate the visual classification and the extraction of drosopterins in acidified ethanol. At various breeding temperatures (18°C, 22.5°C, 27°C), larvae were raised on control food and on HPP-food (0.005 M HPP), and drosopterins extracted from whole heads (1 head/.1 ml or for low values 2 heads/.1 ml), and the absorption at 485 mu determined in a Beckman microcuvette procedure. Our data demonstrate clearly that the HPP can further decrease the drosopterin formation in all mutant strains except ry, although to a different extent. Furthermore, HPP-feeding also causes the temperature dependent semi-lethality and delay in development which is so characteristic for the ry mutants and the HPP-caused phenocopy in cn;ry^{+} animals. The statistic evaluation of over 6 day old flies of the 22.5°C growth series shows highly significant differences (p=0.001) of the means of drosopterin quantities for control versus HPP-food for the genotypes cn;ca and cn,or^{66k} and cn;p^{p} and ras^{2};cn. However, there is always some residual drosopterin synthesis, although on different low levels for the different mutant strains.

Phenylalanine crystal implantation into pupae can increase the drosopterin synthesis in maroon-like and rosy eyes and in the HPP-caused rosy-like phenocopy of cn genotype, as published earlier. This suggested the following two working hypothesis: (A) Phenylalanine is involved in a control mechanism interacting with the xanthine dehydrogenase metabolites and, therefore, acts specific in the maroon-like and rosy mutant and the phenocopy. (B) Phenylalanine acts at a later, more general step in the drosopterin biosynthesis; in this case it should also increase the drosopterin formation in other eye color mutants which have an active xanthine dehydrogenase. The implantation of large phenylalanine crystals into abdomen of late pupae already forming drosopterins in their eyes or into 0-1 hr old flies resulted in a much better long-term survival compared to implantation in younger pupae. Obviously, a smaller increase is expected because 1/2 to 2/3 of the eye drosopterins are deposited before the onset of the experimental phenylalanine supply. Nevertheless, for cn;ca and cn,or^{66k} and cn;p^{p} an increased drosopterin synthesis to two - to three-fold amounts of the control value was found, which is almost as extensive as in the cn;ry^{2} flies used as a control in this experimental series. In contrast, the ras^{2};cn flies did not show a phenylalanine dependent increase of drosopterin synthesis, although in this mutant strain 3/4 of the normal drosopterin formation occurs after the eclosion of the flies and thus would be under the influence of the phenylalanine implant in the experimental series. These data suggest that phenylalanine interacts with some late step on the drosopterin pathway (hypothesis B), resulting in some mutants in a phenocopy distinctly different from the "normal" eye color phenotype.

These epigenetic metabolite control mechanisms thus drastically alter the eye color phenotype: (a) the inhibitor can decrease the drosopterin quantity to as low as 10% of "normal", and (b) the enhancer can cause a several-fold increase in drosopterin formation in various eye color mutants. These results are to be reported in detail elsewhere.

References: Schwinck, I., 1965, Zeitschrift f. Naturforschung 20b: 322-326. Schwinck, I., 1967, Zoolog. Anz. Suppl. 30: 382-390. Schwinck, I., 1968, Intern. Cong. of Genetics, Tokyo, Vol. I: 125. Schwinck, I., 1969, Genetics 61: s53. (Supported by USPHS Grant GM-10256 and a Grant from the University of Connecticut Research Foundation).

Fuller, C.W. and E.W. Hanly. University of Utah, Salt Lake City, Utah. Glutamine synthetase activity in D. melanogaster.

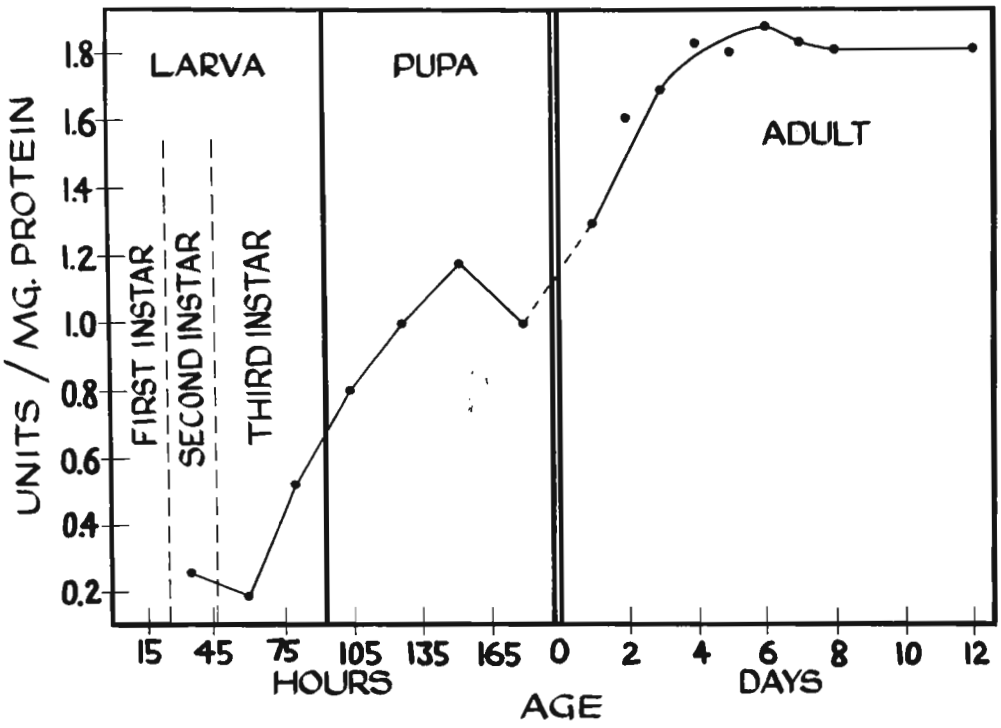
A colorimetric procedure for determining glutamine synthetase activity in various developmental stages of D. melanogaster was established. This involved the modification of a procedure originated by Lipmann and Tuttle (1945) which used a $FeCl_3$-hydroxamate color complex resulting from the following reaction:

$$\text{L-glutamate} + NH_2OH + ATP \xrightarrow{Mg^{++}} \text{L-GHA} + ADP + Pi$$

where GHA is γ-glutamyl hydroxamate which is then complexed with $FeCl_3$.

The assays were done on whole-fly extracts at various developmental stages where the enzyme source was a crude extract prepared in the following manner: 1) One gm frozen flies of various developmental ages was ground in a glass tissue grinder with 10 ml imidazole buffer, pH 7.3 or 10 ml distilled water. 2) Homogenate was centrifuged at 43,600 x g for 20 min. 3) Supernatant stirred with 0.5 gm Norite A for 10 min. 4) Mixture centrifuged at 43,600 x g for 20 min. 5) Supernatant used as enzyme source. All procedures were carried out at 4°C. Pellets of centrifugation steps had no activity.

The routine assay was done at 37°C for 20 min. in an incubation mixture of 2.25 ml containing 25 μmoles sodium ATP (freshly prepared daily), 250 μmoles sodium L-glutamate, 100 μmoles NH_2OH, 100 μmoles $MgSO_4$, 100 μmoles 2-mercaptoethanol, 375 μmoles imidazole buffer and 0.2 ml enzyme extract. These concentrations were determined to be optimum under the conditions used. All components were neutralized to pH 7.3. After the final addition of ATP, the reaction mixture was equilibrated to 37°C for 3 min. before the addition of enzyme extract. The reaction, after 20 min., was terminated by the addition of the $FeCl_3$ reagent (containing HCl and trichloroacetic acid). The tubes were thoroughly shaken and then spun for 15-20 min. in a clinical centrifuge to remove the precipitated protein. The supernatant was removed and absorbance measured at 500 mμ which was determined to be λ_{max}. Protein concentration was determined by the method of Lowry et al. (1951).



Results are reported as GSA (glutamine synthetase) specific activity (units/mg. protein, where one unit is equal to one μmole GHA formed per hour).

The temperature optimum for this reaction was found to be 41°C. The amount of GHA formed was linear with the amount of enzyme and the amount of each substrate at low concentrations of each. The K_m for L-glutamate was found to be about 1×10^{-2} M; for hydroxamate about 6×10^{-4} M.

Whole wild-type (Oregon-R) were used as source of enzyme extracts for larvae, pupae and adults of various ages. The figure shows that there is a slight dip in enzyme activity or concentration (measured on specific activity basis) at approximately 60 hours after hatching and again just prior to emergence from the puparium at 180 hours. These dips are reproducible. There is a slight break in continuity of the curve at emergence but only minor. The activity of the adult increases to a maximum at approximately 4-5 days of age. The slight drop in activity at about day 7 of the adult may not be real, although it occurs in every assay.

Tobari, I. and M. Murata. National Institute of Radiological Sciences, Chiba, Japan

Mutation rates at the loci controling esterase activity of D. virilis.

Recent studies on electrophoresis of single flies have made it possible to know the genetic variabilities of enzymes and their selection mechanisms in populations. Such studies were performed with some Drosophila species and indicated that there were considerable amounts of enzyme variations in most natural populations of Drosophila. No one knows, however, at the present time the exact nature of the mechanisms through which the genetic variations have been maintained in the populations. One of the possible mechanisms, suggested by Lewontin and Hubby (1966), is that selection tends to eliminate alternative alleles but mutation restores them. In order to accept this hypothesis it is necessary to assume the extraordinary high mutation rates or very, very weak selection on the average. The purpose of the present study is to estimate the X-ray induced mutation rates for esterase alleles of D. virilis and to see whether or not the mutation rates are much higher for the esterase alleles than for visible or dysgenic alleles, such as recessive lethals.

The present study consists of two experiments; in Experiment-I the mutation rate from "inactive" to "active" is estimated, while in Experiment-II the reverse is done.

In Experiment-I, male flies of D. virilis taken from "null" strain, which was homozygous for the silent allele at all loci concerned, and therefore had no esterase band, were irradiated with 2,000r of X-rays and thereafter mated with the homozygous females from the same "null" strain. The progenies emerging in the next generation were examined by thin layer agar electrophoresis.

In this experiment the total number of flies examined was 9,372; no mutation was observed at all esterase loci except for Est-2 locus. At this locus we detected 2 mutations from Est-2^O to Est-2^B, where Est-2^O was a silent gene producing no esterase band. This mutation rate was estimated to be 1.05×10^{-7}/r.

In Experiment-II, males homozygous for both the Est-2^B and Est-9 were exposed to 2,000r of X-rays. Immediately after irradiation they were crossed to the females taken from the "null" strain used in Experiment-I. In the next generation F_1 flies heterozygous for "null" and Est-2^B.9 were examined by the thin layer agar electrophoresis.

A total of 14,020 flies were examined in this experiment. At the Est-2 locus 2 mutations from Est-2^B to Est-2^O, and one from Est-2^B to Est-2^D were detected. At Est-9 locus, 7 mutations to "null" were found. The mutation rates were 0.72×10^{-7}/r, 0.36×10^{-7}/r and 2.50×10^{-7}/r, respectively, for Est-$2^B \rightarrow$Est-2^O, Est-$2^B \rightarrow$Est-2^D and Est-9 $\rightarrow$"null". No mutation from the Est-9 band to another esterase band was found. Furthermore, we found 27 cases showing that both of Est-2^B and Est-9 genes mutated together to "null" genes. In this case, it is not obvious that this event is responsible for either point mutation or chromosomal aberration.

Demerec (1934) has reported the mutation rate to be 5.2×10^{-8}/r on the average at 9 loci on the autosome of D. melanogaster. The same order of the mutation rate has been presented by Alexander (1954), i.e., 1.5×10^{-8}/r. At white locus which is located on the X-chromosome of D. melanogaster, Bonnier and Luning (1949) has estimated the mutation rate to be $0.8\sim1.2 \times 10^{-7}$/r. Girvin (1949) has estimated it to be 7.6×10^{-8}/r on the average at 7 visible loci on the sex-chromosome of D. virilis.

Comparing the results obtained in this study with those mentioned above, it seems very unlikely that the X-ray induced mutation rate at isozyme loci is considerably higher than those at visible loci. However, it cannot be determined from the present results that the genes controlling isozyme activity have either an extremely high mutation rate or a very low selective value because of the small number of chromosomes examined. Further studies should be done to accumulate data on this problem.

(The nomenclature of the esterase loci used in this study has been made by Ohba (1968), see Proc. XII Inter. Congr. Genet., Vol. II: 156.)

Novitski, E. and W.J. Peacock. University of Oregon, Eugene, Oregon; and C.S.I.R.O., Canberra, Australia. Results from the combination of B^S and SD in the male.

It is known that the components of the B^S translocation, and their homologs, are recovered from a male with unequal frequencies. This aberration has been combined with a segregation distorter chromosome (SD-72) to check for further interactions. The experimental set-up has been modified slightly by replacing the basal segment of the translocation, the piece of the X-chromosome extending from the centromere to the Bar region, capped by the tip of the fourth chromosome by a Y chromosome which has incorporated into it the basal segment just described, and the tip of the X chromosome carrying the normal allele of yellow. The translocation so constituted will be referred to as $T(1;Y;4)B^S$ and the modified Y chromosome as B^SYy^+.

Table 1. Progeny from mating ♂♂ of constitution $T(1;Y;4)B^S$, B^SYy^+/Y;SD-72/cn bw to ♀♀ of constitution y/y;cn bw (Line A) and to $y/y/B^SYy^+$;cn bw (Line B).

Type of ♀♀	B^S ♀	B^S cn bw ♀	y ♂	B^S ♂	y cn bw ♂	B^S cn bw ♂
A. y/y	44	21	126	0	0	0
B. $y/y/B^SYy^+$	27	7	40	62	1	3

It will be noted from both Lines A and B that the progeny that carry the simple Y chromosome from the father are always SD so that those cn bw progeny that do appear are B^S females, with only one exception. The large B^S ♂ class, 62, in Line B, is basically the y ♂ class which has received a B^SYy^+ chromosome, as well as an X chromosome, from the mother.

Table 2

	♂	♀	SD?	y♀	B^S♀	B^S♂
A	B^S	$\overline{XX}$	-	1092	595	608
B	B^S	$\overline{XX}/B^S$	-	914	372	519
C	B^S;SD	$\overline{XX}$	+	390	154	147
			-	91	90	110
D	B^S;SD	$\overline{XX}/B^S$	+	124	59	77
			-	36	28	42

Table 2 gives the distribution of progeny when males with the translocation and both without (A & B) and with (C & D) SD are mated to $\overline{XX}$, cn bw ♀♀ (A & C) and to $\overline{XX}/B^SYy^+$;cn bw ♀♀ (B & D).

Lines A and B, when analyzed jointly to determine the frequencies of the gametic types from the male, show the typical pattern. The larger of two homologs is least frequently recovered (X^D, .433 vs IV, .567 and B^SYy^+, .389 vs Y, .611) and when the expectations are arrived at by cross multiplying, the disagreement with the observed frequencies is approximately 3.5%. If the SD-bearing progeny from lines C and D are handled similarly, the results are approximately the same. (This may be seen by inspection by comparing the ratios of 1092, 592 and 608 with 390, 154 and 147, respectively, and the ratios of 914, 372 and 519 with 124, 59 and 77 respectively.) In other words, the conditions which lead to the unequal recovery of the translocation components characterize the SD cells. But the non-SD, the cn bw classes are more nearly equal (91, 90 and 110 in Line C and 36, 28 and 42 in Line D), and, if anything , with a slight increase in the B^S ♂ class, which is consistent with the results in table 1, that cn bw progeny are more apt to be B^S than not, although the effect here is not nearly so striking as in the first table. In fact, when the gamete types are considered individually in the standard way, the increase appears to arise from a greater recovery of the distal part of the translocation rather than the basal (B^SYy^+). A note also should be made of a clear difference between the results in Table 1 and those on lines C and D of Table 2. In the first case, all but one of the cn bw exceptions appearing received the X^D+B^SYy^+ gamete from the father. In the second, only a preponderance do. The essential difference between these experiments is that in the first case the females carried free X-chromosomes and the second attached X's. Whether this difference in female constitutions is responsible for the different results remains to be seen.

From this it can be seen that the recovery of the translocation components is different depending on whether or not the sperm also carries SD. When it does not, the homologs in the translocation approach a 50% recovery, with approximately 25% recovery of each of the four

products, whereas with SD, the recoveries are grossly disparate. This can be interpreted to mean that the same condition which leads to a preferential recovery of the SD chromosome also provides the basis for the preferential recovery of the translocation components. As in other experiments involving the B^S translocation, males are exceedingly infertile; non-function or dysfunction of the mature sperm seems a distinct possibility.

del Solar, E. Universidad de Chile, Santiago, Chile. Behavior in selected gregarious lines in D. pseudoobscura.

The manner in which Drosophila females distribute their eggs among the available sites for oviposition has been denominated aggregation. Two lines from a CH/CH population of D. pseudoobscura selected for high and low aggregation over twenty generations, were significantly different according to three statistics: a) the number of vials containing one or more eggs, b) the percentage of eggs in the vials with the largest number of eggs, and c) an aggregation index = $100\sqrt{s^2-\bar{x}}/\bar{x}$. These results suggested that this gregarious behavior is under genetic control.

In the aforementioned experiments the females from the line selected for low aggregation showed an increase in fecundity, which, measured in groups of 15 females in population cages containing 15 food cups over a ten day period, was of 1.7 to 1 eggs in the High respecting the low line.

The present experiments were designed to compare the fecundity of both lines under two conditions: a) 15 females in a population cage with 15 food cups, and b) 15 females in a 15 x 2.9 cm. vial containing a paper spoon with food medium. The food containers were renewed daily over 10 days in both cases.

The results summarized in Table 1 show that the females from the line selected for High

Table 1. Fecundity among flies selected for high and low aggregation in population cages and in vials.

Number of females	System	Number of replicates	Number of eggs per day High line	Low line	"t"	P
15	cages	3	323.2±62.3	543.8±46.5	2.107	0.05-0.02
15	vials	10	221.3± 7.9	187.6± 8.1	2.105	0.05-0.02
3	vials	10	77.2± 3.7	61.2± 3.1	2.352	0.02-0.01

aggregation lay more eggs under crowded conditions than the females from the Low line. This suggests that their fecundity is influenced by the space available for oviposition.

The behavior of females from the Low line maintained in cages for 12 or 24 hours was com-

Table 2. Average number of cups, eggs, and aggregation indices in lines selected for high and low aggregation.

Direction of selection	Time in hours	Number of replicates	Cups with eggs $\bar{x}$ ± S.E.	Eggs $\bar{x}$ ± S.E.	Index $\bar{x}$ ± S.E.
Low	12	6	8.2±1.0	196.1±29.2	153.8± 9.6
High	24	6	5.0±0.6	379.5±57.5	206.8±17.3
Low	24	6	13.8±0.4	517.0±58.0	111.8± 6.2
High	48	6	9.2±0.9	817.3±94.8	129.3±12.7

pared to that of females from the High line kept in other cages for 24 or 48 hours. The results summarized in Table 2 indicate that both lines behave independently of time, in the expected direction, i.e., while the average number of cups used, and the average total of eggs laid is always greater in the Low line, the aggregation index is lower than in the High line.

Polivanov, S. Catholic University of America, Washington, D.C. Double elimination (?) of X chromosomes in D. melanogaster.

An abnormal male was found in a lz^{63i}/M-5 culture. This male had one typically lozenge eye, while the other eye was Bar. (B flies are sometimes produced in lz/M-5 culture when M-5 chromosome is broken due to crossing-over.) This male was isolated and 8 virgin M-5 females were added to the culture. Four days later this male died. No offspring were produced.

This male had normal external morphology except the eyes. The most probable explanation for the production of such a male was suggested by S. Pipkin. Apparently this individual was started as a lz/B female, and then due to some or other reasons one of the X chromosomes was either inactivated or eliminated in each cell. It is unusual, however, that different X chromosomes were lost in different parts of the body. It is unlikely also that parts of the body still contained both X chromosomes, since the eye was very narrow as in B males and no morphologically female structures were found. (Sex combs were present on both front legs and abdomen had typically male shape.)

Wattiaux, J.M. and A. Elens. Facultés Universitaires N.D. de la Paix, Namur, Belgium. Variation in the sexual behaviour of Drosophila.

The purpose of this paper is to call attention to special kinds of fluctuations which may give some misleading results and to suggest a rationale to avoid this pitfall. The variability we are referring to, concerns the apparent heterogeneity in sexual behaviour depending upon the time of observation.

The results to be described here have been obtained by means of a technique introduced by Elens and described by Elens and Wattiaux (1964). Two kinds of virgin females and two kinds of virgin males are introduced into a small wooden box with a checkered canvas floor and a glass cover which enables the scorer to record the different sorts of copulation and their occurrence according to time of observation.

TABLE I. VARIATION OF THE RATIO OF HETEROGAMIC TO HOMOGAMIC COPULATIONS (D. melanogaster)

Time of observ. in minutes	cross +/+ x e/e			cross b/b x e/e			cross vg/vg x e/e, vg/vg		
	actual values		ratio of	actual values		ratio of	actual values		ratio of
	hetero	homo	cum. val.	hetero	homo	cum. val.	hetero	homo	cum. val.
20	184	268	.69	10	56	.18	24	42	.57
40	94	60	.85	26	22	.46	16	28	.57
60	108	162	.79	14	18	.52	10	2	.69
80	53	36	.83	4	8	.52	12	6	.79
100	11	6	.85	2	4	.52	12	0	.95
120	2	0	.85	0	0	.52	4	0	1.00
Heterogeneity chi-square	7.55*			27.6**			31.5**		

TABLE II. VARIATION OF THE RATIO OF HETEROGAMIC TO HOMOGAMIC COPULATIONS (D. subobscura)

Time of observation in minutes	cross: Meerdael x Jerusalem		
	actual values		ratio hetero/homo
	hetero	homo	cumulated values
30	114	158	.72
90	135	176	.75
180	99	95	.81
360	80	96	.82
Heterogeneity chi-square	4.23 N.S.		

We will refer to some results obtained in D. melanogaster and D. subobscura.

Table I and Table II record the actual number of heterogamic vs. homogamic copulations observed during a given time interval (f.i., from 0' to 20', from 21' to 40' ....) and the ratio of heterogamic to homogamic copulations (sexual isolation index) calculated from cumulated values. The heterogeneity chi-squares are calculated from non-cumulated values.

It appears that the coefficients of sexual isolation do not fluctuate randomly around some average value but show a significant increase, according to the time of observation. In other words, since females are only inseminated once, active females, i.e., those copulating in the beginning of the experiments, are also more selective.

Reference: Elens, A. and Wattiaux, J.M., 1964, Direct observation of sexual isolation DIS 39: 118-119.

Tokunaga, C. Lawrence Radiation Laboratory, University of California, Berkeley, California. The effect on somatic crossing over of an ey^D insertion into chromosome 3.

In an earlier study Stern and Tokunaga (1967) described a striking example of nonautonomy in the differentiation of the multiple sex comb pattern of ey^D males. The evidence was based on the appearance of sex comb differentiation in genetic mosaics consisting of ey^D and not-ey^D areas. The genetic constitution of the zygotes was $y;T(1;3;4)sc^{J4}ey^D/+$ where the X chromosome carried y and one of the third chromosomes carried y^+ of the sc^{J4} translocation at its left tip and ey^D inserted in region 70C of the salivary map. The other third chromosome was normal. Somatic crossing over to the right of ey^D, i.e. between ey^D and the kinetochore can result in a y;not-ey^D constitution and crossing over to the left of ey^D in $y;ey^D$. Both constitution may be recognized as yellow spots on a y^+ background. They could be distinguished from each other provided not-ey^D behaved autonomously on the ey^D background. As, however, the great majority of yellow spots formed multiple sex combs it was concluded that y;not-ey^D spots behaved nonautonomously so that they could not be distinguished from $y;ey^D$ spots. An estimate of the relative frequency of the two kinds of y spots was based, in an independent experiment, on the relative frequency of somatic crossing over to the right and left of h, (3-26.5, salivary map unit 66D), this gene having been substituted for ey^D. Hairy (h) behaves autonomously in mosaics and the occurrence of h spots was studied on the scutellum of $y;T(1;3)\ sc^{J4}/h$ males. Sixty five spots that included at least one macrocheata were clearly recognizable as yellow. Of these, 45 were hairy and 20 were not-hairy giving a ratio of crossing over to the right and the left of h as 45:20 or more than 2:1.

It has been suggested that the ratio of right to left crossovers in the preceding experiment with h may not be a reliable index for the ratio in the main experiment which involved the insertion of ey^D. This was tested by a new experiment in which both ey^D and h were present. Among 3329 males of the genotype $y;T(1;3;4)sc^{J4}ey^D$, 36 had mosaic scutella exhibiting a yellow spot which included at least one macrochaeta. Of these spots, 28 were hairy and 8 were not-hairy, giving a ratio of crossing over to the right and the left of h as 28:8 or more than 3:1. This ratio does not differ significantly from that found in flies without the ey^D insertion. It is concluded that the estimate of the somatic crossing over ratio to the right and the left of ey^D that forms the basis of the interpretation of nonautonomy of the not-ey^D effect in sex comb mosaics is a valid one.

Adamkewicz, S. Laura. and R. Milkman. The University of Iowa, Iowa City. Apparent heterosis in the second chromosome of D.m.

From two wild Amherst isofemale strains, pure slo and pure fast α-glycerophosphate dehydrogenase lines, respectively, were obtained. These were crossed, and the F_1's were backcrossed to each parent line in each direction. Uncrowded (25-50 eggs per vial) and crowded (400 eggs per vial) backcross progeny were examined. All flies emerging from each vial were counted.

Table 1
Numbers of each genotype among flies tested and survival

Cross	Genotype	N		Adults/Eggs*	
		Crowded	Uncrowded	Crowded	Uncrowded
Slow x F_1	FS	575	270	0.31	0.72
	SS	481	260		
F_1 x Slow	FS	561	283	0.42	0.79
	SS	505	272		
Fast x F_1	FS	563	289	0.34	0.83
	FF	258	271		
F_1 x Fast	FS	534	303	0.27	0.88
	FF	337	309		

* In all crosses, about 94% of the eggs hatched (137-142 of 150)

The data in Table 1 suggest density-dependent heterosis. Moreover the heterozygotes emerged much earlier than fast homozygotes in crowded, but not in uncrowded, vials. We have no evidence that the α-glycerophosphate dehydrogenase locus is itself involved in the apparent heterosis: indeed comparison of reciprocal cross result in Table 1 tends to suggest a contribution some distance away, since the excess of heterozygotes is not so great when the F_1 parent permitted crossing over.

The multiple applicator (see Technical Notes, this issue) was used.

Mukai, T. North Carolina State University, Raleigh, North Carolina. Spontaneous mutation rates of isozyme genes in D. melanogaster.

Using three second chromosomes collected from a Madison, Wisconsin population, an experiment is being conducted to accumulate mutant genes, after they have been replicated to 150. The mating scheme is Cy/Pm (5 females) x $Pm/+_i$ (1 male) where i indicates line number. All chromosomes carried F alleles at the alcohol dehydrogenase (ADH) locus. A second experiment was initiated using two lethal carrying second chromosomes that originated from an Erie, Pa. population. One of them carried the F allele and the other the S allele at the ADH locus. These chromosomes were each replicated to 500 chromosomes (Total=1000) and 1000 lines were established in the mating scheme: Cy/F 1 x Cy/F 1 and Cy/S 1 x Cy/S 1 [Cy-chromosomes (SM1 chromosomes) carry F gene at the ADH locus]. These lines were maintained by single pair brother-sister matings. In generation 85 for the first group and from generation 37 to generation 39 for the second group, these lines were examined for the ADH alleles and, in addition for the malic dehydrogenase-1 (MDH-1) - Madison only - and α-glycerophosphate dehydrogenase-1 (αGPDH-1) alleles, the loci of which are also located in the second chromosome. The results are described in the following table. Several somatic mutations, which show mutant characters that are not transmissible, were discovered but they were not counted as mutants.

Material	Erie, Pa.			Madison, Wis.	
Enzyme	ADH	α-GPDH-1	MDH-1	ADH	α-GPDH-1
Number of mutants	0	0	0	1*	0
Total number of chromosome generations	$\sim 7.4 \times 10^4$	$\sim 7.8 \times 10^4$	$\sim 7.4 \times 10^4$	$\sim 1.2 \times 10^4$	$\sim 1.2 \times 10^4$

*mutations from F to S

So far, a pooled estimate for isozyme mutation rate is 0.4×10^{-5}/locus/generation, so it would appear that isozyme mutation rates are not higher than recessive lethal mutation rates on a per locus basis. Accumulation of enzyme mutations at these loci is being continued.

Bahn, E. University of Copenhagen, Denmark. Restoration of fertility of the female sterile mutant rudimentary on pyrimidine enriched culture medium.

Nørby discovered (Hereditas: in press) that rudimentary mutants show a pyrimidine requirement for development on a special minimal culture medium. It was, therefore, investigated whether homozygous r♀♀ mated to r♂♂ would respond to enrichment of the culture medium with pyrimidines by showing higher fecundity. Striking results were obtained when pure preparations of cytidine were added to the medium. On a routine basis homozygous rudimentary stocks are now kept without difficulty on a sugar yeast medium with 1% RNA added (Sigma, Ribonucleic acid from Torula yeast, Grade VI). In Table 1 the results are compiled in absolute numbers from the cross r^{39k}/r^{39k} x r^{39k} made on the standard sugar yeast medium with different additaments. In sets of 12 vials, 3 pairs per vial were allowed to lay eggs for 7 days.

Table 1

Additament	Number of offspring produced: males	females
Control (sugar yeast)	38	61
½% Orotic acid	39	40
1% DNA	30	38
1% RNA	287	461
½% Cytidine	1279	1072

Despite the vast surplus of RNA no effect on the wing phenotype has been observed. These results show, as Counce (DIS 44: 101) concluded from her studies on deep orange, the necessity of clearly defining and carefully controlling the conditions under which studies of female sterility mutants are carried out.

Brosseau, G.E., Jr. University of Iowa, Iowa City, Iowa. V-type position effects for e^+ and ro^+ in Drosophila.

An attempt to recover induced V-type position effects for the loci ro^+ and e^+ yielded quite different results for each of these two genes. Oregon-R males were irradiated with 4000 r of X-rays and mated either to ro females, to e females or to Xa/Ubx^{130}, e females depending upon the locus being tested. The F_1 progeny were examined for any expression of the recessive phenotype. All putative mutants were confirmed by a retest and salivary chromosomes were checked where possible. The experiments were designed to only recover the desired position effects and no counts of F_1 were made with one exception. The e experiments involved about 4 times as many F_1 progeny as the ro tests.

The matings to ro were carried out first. A large number of progeny with some roughness of the eye were recovered. Many of these were sterile and others proved to be dominant changes at loci other than ro. Among the fertile rough eye F_1 progeny were 3 ro mutants and 2 V-type position effects of ro^+. The mutants proved to have normal salivary chromosomes while both the position effects had rearrangements that brought region 97D, in one case, and 97E, in the other, next to the chromocenter. Both of these latter rearrangements were associated with marked variegation of the eye.

The experiments with ebony yielded 8 e mutants but no position effects. This was a surprising result because of the contrasting ease with which ro^+ position effects were found and because current thinking of the mechanism of position effect does not take into account the possibility that a particular locus might be immune to position effect. One trivial explanation is that e might be non-autonomous in action. This is not the case because 2 of the 8 e mutants were mosaic mutants. The last of the three tests carried out with e was conducted at 19°C in the hope that the temperature enhancement might maximize the likelihood of recognition of the ebony phenotype. This run yielded 1 ebony whole body mutant and 1 mosaic mutant but no position effects among 3700 F_1 flies.

There is presently no hypothesis that would permit reconciliation of the discrepancy between these results with ro vs e. Either the appropriate rearrangements are not recoverable in the case of breaks near e^+ or some property of this locus confirms upon it an immunity to the gene-repressing effect of heterochromatin. The nature of the respondent locus must also be taken into account in formulating hypotheses to explain V-type position effects. (Supported by NIH Grant GM06508-10)

Petit, C. Faculté des Sciences, Paris, France. Is D. melanogaster a domestic species?

The genetic structure of various populations of D. melanogaster has been examined, the character chosen as reference being the number of ovariolae. The coefficient of right - left correlation (ρ) was used to estimate the genetic homogeneity (see Reeve and Robertson, 1954).

The investigation has revealed an important genetic heterogeneity both in the sparse populations encountered in the beginning of summer and in the large populations found at vintage time. A start in differentiation has been noted in a basement where constant conditions allowed important populations to develop all the year round, but in the case of a "wild" population the characteristics seem to be maintained from one year to another.

		Wild populations		Cellar populations	
		m ± e	ρ	m ± e	ρ
Populations encountered in July	1964	21.85 ± 0.18	0.38*	22.90 ± 0.14	0.43*
	1965	21.31 ± 0.16	0.35*		
Populations found at vintage time	1964			22.78 ± o.18	0.43*
	1965	21.58 ± 0.16	0.39*		

* significant

These results tend to prove that the species D. melanogaster is less domestic than is generally believed.

Novitski, E. and Dan L. Dews. University of Oregon, Eugene, Oregon.
Comparison of mating ability of diploid and triploid females.

Triploid individuals are no larger than diploids despite the fact that 3N cells are typically larger than 2N cells. This is so because there are fewer cells in the adult 3N female than in the 2N female. If this is true of the nervous system, one might wonder if the functioning of the triploid adult is affected.

In a preliminary attempt to explore this question a comparison was made of the mating behaviors of the diploid female and triploid female when placed in competition with each other. The detailed results are given below; they demonstrate quite conclusively that the triploid females in our experiments were at a disadvantage compared with the diploids. While these results are consistent with the initial supposition that triploids would be at a disadvantage with respect to diploids because of a probable reduced number of cells in the nervous system, it remains to be shown that this in fact is the case, since there must be a number of other differences between 2N and 3N females that could have the same end result. It is of interest that this result is similar to one, pointed out to the authors by K.C. Atwood, obtained by Fankhauser et al (Science 122, 692) in tests of 2N and 3N salamanders.

	3N		2N		
	not mated	mated	not mated	mated	χ^2 1 d.f.
Exp. I	24	13	16	22	3.90*
Exp. II	45	24	11	56	33.42**
totals	69	37	27	78	32.99**

Experimental procedure and results: Two different triploid lines were each backcrossed to Oregon R males for several generations to produce two lines giving 2N and 3N females of wild phenotype. Wing cell and ommatidium size were used to distinguish 2N from 3N females. A small number, from three to ten, of virgin 3N females were matched with an equal number of virgin diploid females from the same culture bottle, and with an equal number of previously unmated Oregon R males. The females were 5-6 days old and the males 3-4. To avoid interfering with mating behavior, all flies were transferred unanesthetized to quarter pint milk bottles. After 2 hours the flies were etherized, 2N females separated from 3N females, males discarded, and all females cultured individually. It was assumed that lack of progeny production by a female indicated she had not mated. As a partial check on this assumption, 22 2N and 3N females which did not produce progeny were examined 4-5 days after mating to see if their spermathecae and ventral receptacle contained sperm. Sperm was found in only one of the 22 females; this female had laid no eggs. In 4 out of 17 runs, unintentional deviations from exact equality of 3N females and 2N females occurred. These deviations from equality did not exceed one individual per run.

Although a much greater proportion of the 2N females mated in Exp. II than in Exp. I, 3N females in both Exp. I and II mated significantly less than did 2N females. It is not known whether this difference is due to rejection of courting males by 3N females or to less courtship by males of 3N females than of 2N females. Both experiments I and II included runs using the two different 3N stocks. No significant difference between the two 3N stocks was found.

Taira, T. and F. Uda. Waseda University, Tokyo, Japan.
Deamination of adenosine 2',3'-cyclic phosphate in D. melanogaster.

Four nucleoside cyclic phosphates were isolated from the hot ethanol extracts of the 3rd instar larvae of D.m. and identified as follows: cytidine 2',3'-cyclic phosphate (Cp!), uridine 2',3'-cyclic phosphate (Up!), guanosine 2',3'-cyclic phosphate (Gp!) and inosine 2',3'-cyclic phosphate (Ip!).

The occurrence of Ip! instead of Ap! in the larvae suggests the presence of a deaminase which catalyzes the conversion of Ap! to Ip!. Such an enzyme has indeed been shown to be present in Drosophila larvae.

The purification and characterization of the deaminase in D. were carried out by means of the separation of 50 to 70 percent saturation of ammonium salfate and the fractionation of gel-filter column. The present results suggest that: (1) the deaminase from D. larvae would be one sort of molecular weight, about 200,000, and (2) this enzyme could catalyze the conversion of Ap! to Ip! as well as that of adenosine to inosine.

Novitski, E., and E. Ehrlich, University of Oregon, Eugene, Oregon. Suppression of SD by Y;autosome translocations.

Modification of the k value of SD by rearrangements is well known. In order to see if there is any relationship between the position of the breakpoint of a translocation and its degree of modification, we have, over the past half dozen years, induced a number of translocations specifically for this purpose. Four of the translocations involve an SD-72 second chromosome; four others are Y-3 translocations. The series T(Y;2) A, B, C and E are included in this list although they are of ancient origin, having been induced by Dobzhansky in 1929 and are therefore of unspecified behavior with respect to SD. The six marked by an asterisk were induced in lines carefully selected for high k value.

Positions of breakpoints of Y-autosome translocations and the k values given by SD in combination with them

Translocation	Y-Chromosome	Breakpoint	Relative Position	k Value
T(Y;2),SD,EM106*	$y^{+}YB^{S}$	31D	middle of 2L	.555
T(Y;2),11-11N	$sc^{8}.Y$	34A	middle of 2L	.176
T(Y;2),12-4A	$sc^{8}.Y$	34A	middle of 2L	.116
T(Y;2),E	Normal	36D	near centromere 2L	.980
T(Y;2),11-26A	$sc^{8}.Y$	36F	near centromere 2L	.237
T(Y;2),SD,j-4*	$y^{+}YB^{S}$	37B	near centromere 2L	.337
T(Y;2),A	Normal	41A	near centromere 2R	.966
T(Y;2)B	Normal	41A	near centromere 2R	.337
T(Y;2)C	Normal	41A	near centromere 2R	.895
T(Y;2),SD,EM-135*	$y^{+}YB^{S}$	42A	near centromere 2R	.132
T(Y;2),SD, CB-1c*	Normal	44D	near centromere 2R	.456
T(Y;2),1	Normal	56E	end of 2R	.491
T(Y;2),7	Normal	57D	end of 2R	.583
T(Y;2),16	Normal	59F	end of 2R	.488
T(Y;3),12-4B	$sc^{8}.Y$	78F	near centromere 3L	.533
T(Y;3),12-26M	$sc^{8}.Y$	83D	near centromere 3R	.504
T(Y;3),j-3*	$y^{+}YB^{S}$	91A	middle of 3R	.881
T(Y;3),j-6*	$y^{+}YB^{S}$	91C	middle of 3R	.672

Several points seem clear from the table. In this sample, there appears to be no relationship between the degree of modification of the k value and the position of the breakpoint; this is emphasized by the fact that three of the four Y;3 translocations also suppress SD markedly. It would appear that a more general effect than simple pairing of homologs must be invoked.

Hughes, M. and M.P. Kambysellis. Harvard University, Cambridge, Massachusetts. Effects of ecdysone on RNA synthesis.

When salivary glands from middle third instar larvae of D. hydei are incubated in vitro with α-ecdysone, a series of changes in the chromosomal puffing pattern are set in motion. These changes are identical to those that occur in normal development during the six hours before puparium formation (Berendes, H.D. 1967, Chromosome 22: 274-293). Using animals which had been raised sterilly (Doane, W.W. 1967, Methods in Developmental Biology ed. Wilt, F.H. and Wessells, N.K. pub. Thomas Y. Crowell Co. pp. 219-245), we examined the effect of α-ecdysone on RNA synthesis in these glands by pulse labeling with H^{3}-uridine and analyzed the RNA on sucrose gradiants. We found that glands incubated in Schneider's medium (Schneider, I. 1964, J. Exp. Zool. 156: 91-104) containing 4µg/ml of α-ecdysone showed a rapid and specific decline in the rate of ribosomal RNA synthesis as compared to glands incubated in Schneider's medium alone.

This work was supported by the NSF grants GB-7963 to C.M. Williams, GB-8762 to F.C. Kafatos and by a PHS training grant No. 2 TO1 GM00036-12 to the Department of Biology.

Gehring, W. Yale University, New Haven Connecticut. A recessive lethal (l(4)29) with a homeotic effect in D. melanogaster.

During the examination of a group of fourth chromosome lethals kindly provided by B. Hochman, lethal pupae homozygous for l(4)29^{b} were dissected and studied morphologically. Since some of those pupae die at a late stage when imaginal structures are already formed, it is possible to analyze the visible effects of the mutation on imaginal structures. Lethal flies show two kinds of homeotic transformations: 1) First and second antennal segments are transformed into leg structures; 2) second and third legs are partially transformed into first legs. The effects on the antenna can be summarized as follows: First and second antennal segments are reduced and replaced by a coxa and trochanter. Only a small portion of the second antennal segment forming the joint with the third segment is present. The third antennal segment appears normal but the arista is absent or reduced to a tiny undifferentiated vesicle. In addition, the palpus which is also formed by the antennal disk shows a disorderly arrangement of bristles and sensilla. Leg differentiation is affected in various ways. All three pairs of legs are distorted, segments are swollen and claws are missing. In the male all legs bear sex combs, those of the middle and hind legs often being incomplete. In both sexes the tibia as well as the basitarsus of all legs show transverse rows of bristles, which indicates a partial transformation of the second and third legs into forelegs. Whether the lethal effect and the described morphological alterations belong to the pleiotropic pattern of a single gene, or whether the mutation affects several genes, cannot be decided on the basis of the present genetic evidence. There are three mutants of this complementation group, l(4)29, 29^{a}, 29^{b}, which occurred spontaneously in wild populations. A brief examination of l(4)29 showed that this mutant had the same phenotype as l(4)29^{b}. The fact that recessive homeotic mutations are associated with a lethal effect is of interest with regard to studies on transdetermination[1,2] in cultures of imaginal discs. It might explain why homeotic mutations corresponding to several of the known transdeterminations have not yet been found.

The effect of various mutants known to produce a homeotic effect on the eye-antennal disk are summarized in Table 1. The table clearly indicates that the homeotic transformations are not random. Distal antennal structures are transformed into distal leg segments, while proximal antennal structures are replaced by proximal leg segments. The absence of both the arista and claws in l(4)29 provides further evidence for this proximo-distal correspondence. This may reflect an evolutionary homology of antenna and leg. However, other homeotic mutations like eyeless-ophthalmoptera, which induces the eye disk to form wing structures, apparently involve non-homologous organs.

Table 1. Homeotic transformations in various mutants affecting the eye-antennal disk.

	Homeotic Mutants				
Leg Structures Formed	ss[a]	l(4)29	Antp	Ns	Head Structures Replaced
sternopleura				+	prefrons + vibrissae
coxa		+	(+)	+	1st antennal segment
trochanter		+	(+)	+	2nd antennal segment
femur			+	+	3rd antennal segment
tibia			+	+	3rd antennal segment
tarsus	+		+	+	arista

\+ indicates leg structures formed and head structures replaced, respectively

1) Hadorn, E. 1966. Major Problems in Developmental Biology. ed. M. Locke, Academic Press, N.Y. 2) Gehring, W. 1968. Results and Problems in Cell Differentiation. Vol. 1, The Stability of the Differentiated State. ed. H. Ursprung, Springer, Berlin.

Allen, Archie C. Texas Tech University, Lubbock, Texas. Lethal frequencies in laboratory populations of D. melanogaster.

Eighteen different third chromosome lethals were used in equal frequencies to start 4 populations at different temperatures and sizes (relatively large populations in cages at 18° and 25°C, designated L-18 and L-25; and small populations in vials at 18° and 25°C, designated S-18 and S-25). All third chromosomes carried one of the 18 lethals. The populations were maintained for about a year. After this, lethal frequencies were determined for each population. The two lethals in highest frequencies were selected from each population, without determining if the two different lethals in each population were among those selected for any other population, to initiate new populations with a level of 25% for each lethal and 50% quasi-normal third chromosomes from the natural population (American Samoa). Samples were taken at intervals (generation time varies with population size and temperature, about 24 days for L-25 and S-18, 11 days for S-25 and 42 days for L-18) for over 2 years. Frequencies were estimated for each lethal by crossing single males carrying a third chromosome in balanced condition with a marker to appropriate balanced lethal females from stock cultures. If no wild type appeared in the offspring of this cross the sampled chromosome contained the lethal in question.

Frequencies of two lethals in four experimental populations of D. melanogaster

	Population L-25				Population L-18				Population S-25				Population S-18			
S#*	#	L-25-17	#	L-25-30	#	L-18-2	#	L-18-21	#	S-25-1	#	S-25-16	#	S-18-2	#	S-18-27
0		0.25		0.25		0.25		0.25		0.25		0.25		0.25		0.25
1	45	0.18	43	0.16	20	0.05	18	0.17	25	0.08	24	0.13	34	0.06	25	0.32
2	21	0.10	32	0.09	81	0.07	70	0.29	20	0.10	28	0.20	26	0.19	13	0.31
3	100	0.11	100	0.18	82	0.07	88	0.14	41	0.15	42	0.19	35	0.09	38	0.13
4	45	0.20	55	0.07	40	0.0	40	0.20	37	0.14	49	0.14	82	0.23	98	0.07
5	94	0.05	90	0.08	97	0.02	80	0.11	71	0.27	54	0.22	40	0.20	62	0.03
6					53	0.04	62	0.07	87	0.07	84	0.21				
7									79	0.02	89	0.23				

*S# = Sample Number

The table shows the frequencies of each lethal through a number of generations (estimated 20 for L-25, 11 for L-18, 42 for S-25, and 23 for S-18). They were all maintained above the expected level for lethal heterozygotes selected one half of the time, $\Delta q_n = -sq_{n-1}(1-q_{n-1})/1-2sq_{n-1}$ where s = 0.5. With the exception of one lethal, L-18-2, the total frequency values are higher than would be expected for a neutral effect in the heterozygote, $\Delta q_n = -q^2_{n-1}/1+q_{n-1}$, at the 0.001 level of significance. The 95% confidence limits shows overlap for a neutral effect of all lethals with no overlap with expected values when s = 0.5. These observations indicate a heterotic effect for the 8 lethals with the possible exception of L-18-2.

When the lethals were crossed with 9 remaining balanced stocks that survived from the 18 original stocks, 3 lethals were found in 5 different populations (one in three populations L-18-21, L-25-17, and S-25-1; one in two populations L-18-21 and L-25-30; and one in one population S-18-2). L-18-21 carried two of the original lethals that must have arisen through crossing over and recombination. One of these two lethals is allelic with L-25-30, the other with L-25-17 and S-25-1. The persistence of some lethals under different environmental conditions indicates a heterotic effect independent of the environment in which they are found. To bear this out, one lethal persisted in 3 different populations, as to size and temperature, and was found in linkage with a second lethal (indicating epistatic interaction between lethals) that was found in two different populations.

It may be concluded that chromosomes with genes or gene complexes that are lethal for homozygotes can be selected for their heterotic effects, as indicated by their persistence in different experimental populations. The level at which lethal genes are heterotic in natural populations may have been underestimated with data on fitness values of particular lethals and tests for allelism. And, while these results are not absolute, there are indications (6 of 18 lethals recovered in frequencies at or above the level expected for a neutral effect in heterozygotes) that a very high percent of lethals found in natural populations benefit the heterozygote. This work was supported by Grant GM 12222 from the National Institutes of Health.

Krimbas, C.B., E. Diamantopoulou and M. Loukas. Agricultural College of Athens, Greece. Evidence on the absence of selective neutrality in isozyme alleles of Lap and Est loci in D. subobscura.

It has been repeatedly claimed that isozyme genetic polymorphisms, so commonly found in natural populations, are selectively neutral.

We have constructed several cages from many individuals originating from natural collections. These cages have been maintained from one to five years. Each cage contains several thousands of flies, so drift is not an explanation for changes in gene frequencies. Every cage is characterized by the place of origin of flies and the year of collection.

Some of these cages have been sampled twice, once in 1968 and once in 1969 for two enzyme systems, leucineaminopeptidases and an esterase. Both these enzymes display polymorphisms which are controlled by two different genes located in chromosome O of D. subobscura. Table 1 shows the changes in gene frequencies within a year.

Table 1

Lap

		1968					1969				
		A	B	C	D	N	A	B	C	D	N
Parnes	'65	.26	.65	.06	.03	65	.04	.96	-	-	84***
Crete	'62	.45	.25	.30	-	65	.27	.60	.13	-	78***
Holland	'63	.48	.51	.01	-	71	.18	.78	.01	.03	94***
Pindos	'68	.17	.80	.01	.02	84	.12	.86	.01	.01	86

Est

		A	B	C	D	E	F	N	A	B	C	D	E	F	N
Parnes	'65	.32	.21	.40	.03	.04	-	63	.67	.14	.09	.07	.01	.02	88***
Crete	'62	.58	.25	.12	-	.05	-	24	.74	.11	.07	-	.08	-	82
Holland	'63	.26	.02	.72	-	-	-	46	.43	.10	.43	-	.04	-	98**
Pindos	'68	.34	.18	.29	.01	.18	-	62	.40	.09	.40	-	.09	.02	90**

For the Lap locus allele B increased in frequency in all cases, in three of them the difference is highly significant statistically. It should be noted that every population is different from the others as far as gene arrangements of chromosome O are concerned.

The same pattern, although not so dramatic, is observed for the Esterase gene sampled: all cages show an increase of allele A.

The only plausible explanation of these results is that under cage environment allele B of Lap gene and allele A of Est gene are selected for. Experiments are under way to understand the exact mechanism of selection.

Asterisks indicate statistically significant differences between 1968 and 1969 samples. One asterisk indicates significance at the .05 level, two at the .01 level, three at the .001 level.

Götz, K.G. Max-Planck-Institut für biologische Kybernetik, Tübingen, Germany. Fractionation of Drosophila populations according to optomotor traits.

The optomotor control of orientation and locomotion in the fruit fly D. melanogaster requires the conveyance of information from distinct movement detectors in the visual system to distinct movement effectors in the motor system. Abnormalities of the optomotor control system have been found occasionally in Drosophila. The abnormal flies can be isolated from population samples by appropriate fractionation according to the magnitude and the sign of the optomotor responses. A cyclically operating machine was used to fractionate two inbred strains, the wild stock "Berlin" and the mutant $In(1)sc^{8}w^{a}$. Movements of an artificial visual environment elicit similar orientation control responses, but antagonistic locomotion control responses in the two strains. The responses depend on various parameters and may even change with habituation to the stimulus. However, the application of selection pressure through eight generations has little if any effect on the different optomotor behaviour of the inbred strains.

Rae, P.M.M.* and M.M. Green. University of California, Davis, California. Synaptonemal complexes in some D. melanogaster mutants.

On the assumption that the presence of synaptonemal complexes is correlated with the occurrence of meiotic crossing over we undertook the demonstration of complexes in meta(super)-females and ix^2 intersexes. Synaptonemal complexes morphologically inseparable from those of wild type females were found in both metafemales and ix^2 intersexes by using standard EM methods. This suggests that so far as crossing over is concerned, metafemales and ix^2 intersexes are female-like rather than male-like. Normal synaptonemal complexes were also found in ca^{nd} females lending additional support to the view that ca^{nd} is a desynapsis gene.

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Singh, A. Panjab University, Chandigarh, India. Drosophilidae of South Andamans, India.

The study of the collection from the South Andamans revealed ten species. Out of these three are novo and the remaining seven are reported for the first time from this area. The frequency distribution of the species at five sites of collection is given in Table I.

TABLE I. FREQUENCY DISTRIBUTION OF THE VARIOUS SPECIES OF THE DROSOPHILIDAE IN THE S. ANDAMANS

Species No. and name	Frequency distribution at: Port Blair M	Port Blair F	Baratang M	Baratang F	Wright Myo M	Wright Myo F	Humphrey ganj M	Humphrey ganj F	Cowria Ghat M	Cowria Ghat F	Total flies M	Total flies F
Microdrosophila												
1 Microdrosophila sp	-	-	1	-	-	-	-	-	-	-	1	-
Pholadoris												
2 D. sp. novo.	2	5	8	6	5	-	2	-	-	-	17	11
Tanygestrella												
3 D. gracilis	25	18	90	115	118	192	5	-	3	-	241	325
Sophophora												
4 D. bipectinata	93	179	262	442	207	256	65	-	52	-	679	877
5 D. malerkotliana	4	} 23	13	} 16	6	} 34	1	-	1	-	25	} 73
6 D. ananassae	3		-		36		56	-	15	-	110	
7 D. sp. novo.	14	2	-	-	40	12	6	-	6	-	66	14
8 D. truncata	3	-	25	1	-	-	11	-	-	-	39	1
9 D sp. novo.	-	1	-	2	8	3	-	-	-	-	8	6
Drosophila												
10 D. nasuta	4	3	4	1	-	-	1	-	-	-	9	4
Grand Total	148	231	403	583	420	497	147	-	77	-	1195	1311
Period of Collection	29.2.64 to 3.3.64		12.3.64 to 22.3.64		25.3.64 to 3.4.64		4.3.64 to 8.3.64		8.4.64 to 16.4.64			
Temperature Maximum	31.1-32.4		32.7-33.5		32.2-33.9		31.4-34.9		33.4-34.6			
Temperature Minimum	24.5-20.5		25.7-21		26.5-21.2		23.2-21.1		25.3-23.2			
Relative Humidity	63-74%		67-75%		59-69%		40-72%		60-67%			
Rainfall	Nil		Nil		Trace		Nil		Nil			

Note: M = male, F = female; } = females both of D. ananassae and D. malerkotliana

Rose, R., S. Shafer and R. Hillman. Temple University, Philadelphia, Pennsylvania. Reciprocal in vitro transfer RNA aminoacylation between Escherichia coli and D. melanogaster.

Comparative in vitro studies of the species specificity of the interaction of transfer RNA (tRNA) and aminoacyl tRNA synthetases from yeast, E. coli, and rat liver, have indicated that the extent of the charging of tRNA depends not only on the source of tRNA and the source of the enzymes, but also on the particular amino acid involved in the reaction (Benzer and Weisblum, PNAS 47: 1149, 1961). We have observed similar phenomena in charging experiments using tRNA and enzyme preparations from E. coli and the Oregon-R strain of D. melanogaster.

The in vitro aminoacylation of the E. coli and D. melanogaster tRNA by the partially purified post-microsomal supernatant fractions prepared from E. coli or D. melanogaster was carried out according to the procedure of Rose and Hillman (Biochem. Biophys. Res. Comm. 35: 197, 1969).

The two classes of results are shown in Table 1. With glutamic acid and proline, only homologous charging was observed: D. melanogaster enzymes charge D. melanogaster tRNA and E. coli enzymes charge E. coli tRNA, with little or no heterologous activity. However, with leucine, phenylalanine, valine, and lysine, not only homologous but also heterologous aminoacylation was observed. Of the two heterologous systems studied, the activity is much higher using D. melanogaster enzymes and E. coli tRNA. In the case of lysine, this reaction is three times greater than the corresponding E. coli homologous reaction.

Table 1. Results of Reciprocal Aminoacylation Experiments

	E. coli tRNA		D. melanogaster tRNA	
C^{14} Amino Acid	E. coli Supernatant Fraction	D. melanogaster Supernatant Fraction	E. coli Supernatant Fraction	D. melanogaster Supernatant Fraction
	CPM/mg tRNA x 10^{-3}			
Glutamic Acid	621.3	58.8	13.4	243.8
Proline	487.3	7.9	10.9	84.3
Leucine	336.9	585.7	8.3	191.6
Phenylalanine	266.0	526.9	39.7	253.6
Valine	357.6	418.5	33.1	245.7
Lysine	349.1	1368.3	34.1	228.2

The results indicate that the extent of heterologous tRNA aminoacylation between E. coli and D. melanogaster is affected not only by the source of the material, but also by the particular amino acid tested. (Supported in part by an Institutional Grant IN 88 from the American Cancer Society to Temple University and in part by Grant 1T1-HD 138 from the U.S. Public Health Service.)

Russell, M.A. and F.W. Robertson. Department of Genetics, University of Edinburgh, Scotland. The comparison of growth differences in D.melanogaster in terms of DNA and protein content.

Fluorimetric methods of measuring DNA have been modified to allow estimations on individual adults of D.melanogaster. Extensive comparisons of both DNA and protein content per fly have been carried out for different genotypes, which include inbred lines and crosses between them, selected lines etc., while the effects of different environmental treatments have also been examined. Both genetic and environmental differences may lead to substantial differences in the protein/DNA ratio so that equivalent proportional changes in adult body size may be arrived at in different ways. Such differences apparently derive from the properties of regulation and the rules which determine how a given change in adult size will be effected in terms of cell size and number. Comparisons between the biochemical evidence and estimates of cell size and number changes in the wing, as well as the comparison of heritability of protein and DNA content, support this view.

Lakhotia, S.C. and A.S. Mukherjee. University of Calcutta, India. Activation of a specific puff by benzamide in D. melanogaster.

Effect on the salivary glands of D. melanogaster of in vitro incubation in Benzamide (BM) has been studied. From each mature late third instar larva one of the paired salivary glands was incubated in control ringer (i.e., without BM) while the contralateral gland was incubated in BM-ringer (1.3mg. BM/ml. ringer; pH - 6.7) for 10 minutes and then transferred to control ringer or BM-ringer respectively, both containing H^3-uridine (100 uCi/ml.) and incubated for another 10 minutes after which they were fixed, squashed and autoradiographed with Kodak AR 10 stripping film. It has been observed that in comparison with the control gland the chromosomal RNA synthesis in BM-incubated gland is drastically reduced while the nucleolar RNA is not much affected. RNA synthesis in all but one puff (93D on 3R) is in majority of nuclei completely inhibited in the BM-treated gland. The puff at 93D on the contrary is very highly activated after BM treatment. This puff is either completely absent or very slightly active in the control gland, but in the BM-treated gland this puff is 5-6 times more activated than the control (fig. 1). This specific stimulation of the activity of a single puff under conditions which in general inhibit all chromosomal RNA synthesis is very interesting. All the treatments employed so far to induce puffing in Dipteran salivary glands have resulted in stimulation of a number of puffs. Benzamide has been shown to be an inhibitor of chromosomal RNA synthesis in preference to nucleolar RNA (Jacob, et al., 1964). In view of the fact that in the present study also nucleolar RNA is much less affected while the puff at 93D is super-activated, it is tempting to speculate whether this particular puff at 93D has some functional relation with the nucleolus. Further studies are in progress.

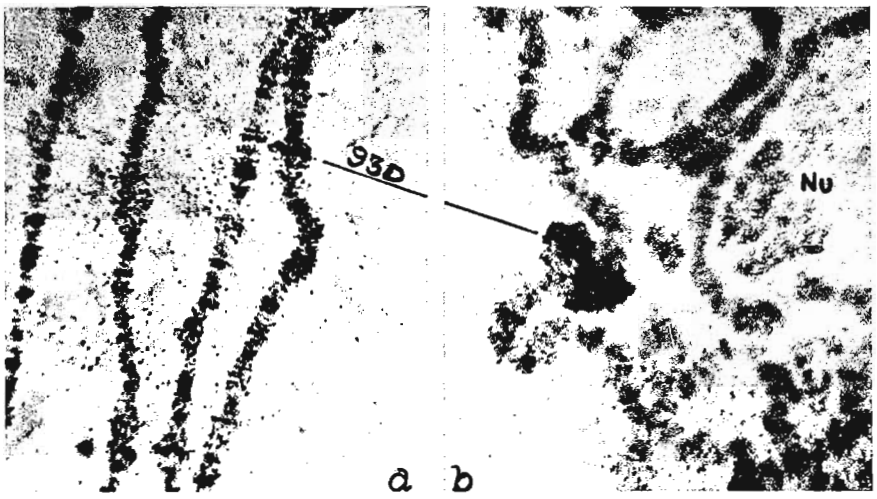


Fig. 1. H^3-uridine labelling in (a) CONTROL gland and (b) in BM-treated gland from the same larva. Note the labelling on the puff at 93D. In (b) Nu indicates the nucleolus.

Reference: Jacob, J., Birnsteil, M.L. and Sirlin, J.L., 1964, "Nucleic acids - structure, biosynthesis and function", Proc. Symp., Hyderabad (India) pp. 197-209.

Mittler, S. Northern Illinois University, DeKalb, Illinois. Controls in tests for chemical protection against radiation induced dominant lethals.

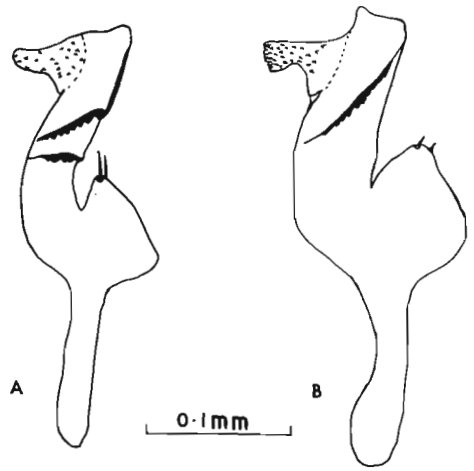
In the past five years various chemicals have been injected into young adult male D. melanogaster in an attempt to reduce the number of radiation induced chromosomal aberrations. In most experiments 0.85% NaCl was used as a control. In the dominant lethal tests, the male was injected with approximately 0.1 µl of the chemical solution to be tested and then exposed to 1600 R of X-rays. These males were mated daily for twelve days, the females were isolated in plastic tubes and permitted to deposit eggs thru a nylon mesh onto darkened media in a petri dish (this method was suggested by Abrahamson). M.M. Walsh in our laboratory found that if the males were not injected and were irradiated at the same time with males injected with 0.85% NaCl that in 8 out of the 12 broods, the males injected with saline solution had significantly less dominant lethals as determined by the failure of larva to emerge in 24 hours. This seemed unusual for a "wet" fly to have less radiation injury than a "dry" fly. However, several years later K. Balkin also in our laboratory working with dominant lethals also found that uninjected males when irradiated produced significantly more dominant lethals in 7 out of the 12 broods than those males injected with 0.85% NaCl.

Angus, D.S. University of Queensland, Brisbane, Australia. D. quadrilineata from Mt. Maquiling, Luzon, Philippines.

The distribution of D. quadrilineata has been discussed previously (Angus 1967) from the literature without access to material - living or dead.

One of the localities from which D. quadrilineata has been recorded is Mt. Maquiling, Luzon, Philippines by Sturtevant in 1927. In February 1969 an extensive sample of living Drosophilas was obtained from Mt. Maquiling, Luzon, Philippines (Mather 1970). From this collection a culture was established from 2 females inseminated in the wild. This culture has allowed the testing of the sexual isolation between this species and the closely related D. tetrachaeta (Bisianumu, New Guinea strain) and D. pseudotetrachaeta (Cairns, Australia strain). It turned out that only rarely will D. quadrilineata cross with the other two species and that an F_1 is never produced. Thus the biological reality of D. quadrilineata is established.

The existence of extensive material from culture has allowed a detailed morphological examination leading to the conclusion that the best way to distinguish D. quadrilineata from D. tetrachaeta and D. pseudotetrachaeta is by the presence on the aedaegus of D. quadrilineata of a second transverse row of scleritized teeth not present in the other two species.



AEDAEGUS
D. quadrilineata (A) D. tetrachaeta (B)

SEXUAL ISOLATION TESTS

Females	Males	Dissected Females	Inseminated Females
D. tet.	D. quad.	104	0
D. pseudo.	D. quad.	102	6
D. quad.	D. tet.	101	2
D. quad.	D. pseudo.	107	5

Acknowledgement: This work was carried out as part of the Research Project "Evolution in the Genus Drosophila" directed by Dr. Wharton B. Mather, Head of the Genetics Laboratory, Zoology Department, University of Queensland.

References: Angus, D. 1967. Additions to the Drosophila fauna of New Guinea. Pap. Dep. Zool. Univ. Qd, 3(3): 31-42. Mather, W.B. 1970. The genus Drosophila at Mt. Maquiling, Luzon. DIS 45: 111.

Nakashima-Tanaka, E. and M. Ogaki. University of Osaka Prefecture, Japan. Chromosomal analysis of jumping behavior to light in D. melanogaster.

It was observed that the adult flies of two mutant strains (bw;st ss and bw;st;sv^n) showed an anomalous response to light (jumping up or dropping down) at the moment the light was intercepted or turned off. This response showed a negative correlation with the age of flies. On the other hand, there was no response to light in the Hikone-H wild strain. This peculiar behavior was able to discriminate very clearly between bw;st ss and Hikone-H strains. The F_1 progenies of reciprocal crosses between bw;st ss and Hikone-H strains did not show any response to light as well as Hikone-H strain. Therefore, it seems that such behavior to light is completely recessive. In order to analyze which chromosome is responsible for the jumping behavior, six special synthetic strains were made: for instance (1) 1-Hikone-H;bw;st ss, (2) 1,2-Hikone-H;st ss and (3) 1,3-Hikone-H;bw (that is, having the Hikone-H first and third chromosomes and the bw second chromosome) etc. The behavior to light of these special synthetic strains and the original bw;st ss and Hikone-H strains were tested. Only 1-Hikone-H;bw;st ss and the original bw;st ss strains responded to light but the others did not. From the above experiments the conclusion may be drawn that the jumping behavior to light in Drosophila is controlled by recessive genes. Furthermore, the multiplicative effects of the genes located on the second and third chromosomes are necessary for the positive response to light, but at least the first chromosome has no relation with the behavior.

Elens, A.A. and J.M. Wattiaux. Facultes Universitaires N.D. de la Paix, Namur, Belgium. Influence of light intensity on mating propensity of D. ambigua and D. subobscura.

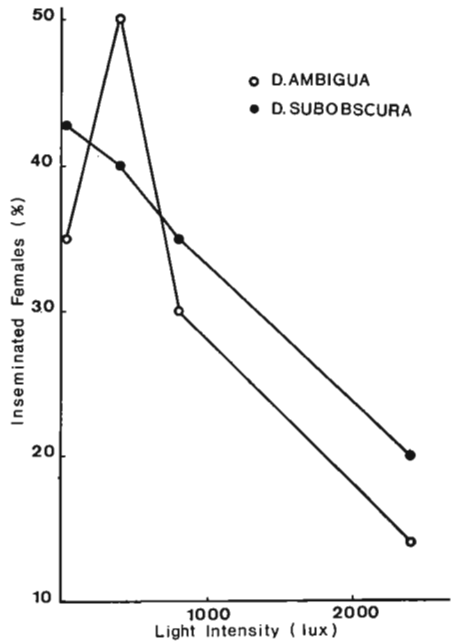


Fig. 1: Effects of light intensity on mating propensity.

Preliminary experiments have shown a strong influence of light intensity on the mating propensity of D. subobscura and D. ambigua. This dependence, however, is not identical.

This relation came out in tests carried on with a different purpose. In each test 26 males and 26 virgin females, 6 to 7 days old, were put together in the "observation chamber" previously described, and the number of matings accomplished during six hours was recorded (from 8 A.M.). The light intensity was recorded with a "Luxmeter" LAND. The mating tests (10 for each of the 4 light intensities studied) were performed in a thermo-regulated room, at 20°C. The individuals were also developed at 20°C. All the experiments were run from January to April.

In D. subobscura, the mating propensity is negatively correlated with light intensity; mating propensity is lower in D. ambigua, but shows the same general trend, with a conspicuous difference at 400 LUX, where it is higher. In Fig. 1, the differences in percentage of inseminated females are always significant, as shown by the Tuckey test at 0.05 probability level, for the same light intensities.

We regret that an accidental loss of the stocks did not allow us to repeat similar experiments in other seasons.

Gvozdev, V.A. and V.T. Kakpakov. Kurchatov's Institute of Atomic Energy, Moscow, U.S.S.R. Establishment of female embryonic cell sublines of D. melanogaster in vitro.

The growth of diploid cells of the Oregon R-C wild stock was maintained in C15 medium with 15% bovine fetal serum during 50 days and more (Genetika, Russ. No. 2: 129, 1968). The rates of cell growth in primary culture were greatly enhanced in the presence of heated at 60° pupae extract (1 g of wet weight of pupae per 3 ml of C15 saline solution). The presence of pupae extract and of bovine fetal serum is required for the growth of cell lines which were obtained from the primary culture. Pupae extract contains both thermolabile (100°C) and thermostable factors enhancing cell growth.

The cells are transferred at 4-7 day intervals and passed over 250 generations. The population doubling time at 28°C is 24 h.

Two sublines with female karyotype were obtained: a diploid subline-67j25D and a tetraploid subline-67j25T. Variations in the number of IVth autosome pair were not taken into account. Both sublines have been originated from the line containing, at the 19th passage, 50% diploids, 12% tetraploids and 38% aneuploids. The majority of the latter were characterized by four X-chromosomes and seven large autosomes.

Determination of the activity of the sex-linked 6-phosphogluconate dehydrogenase structural gene has shown that in the diploid and tetraploid sublines all X-chromosomes were active.

O'Brien, P.E. and J.H. Potter. University of Maryland, College Park, Maryland.
Courtship behavior in two strains of D. melanogaster.

A comparative analysis was made of the courtship behavior of two strains of wild type D. melanogaster. One strain was obtained from the Genetics Research Unit, Carnegie Institution, Cold Spring Harbor, New York. The other strain was derived from flies taken in the wild near Chester, Vermont. Both strains were carried by mass subculturing for nine months before the study was initiated.

Between 17 and 28 single pair matings of 3-5 day old virgins of the same strain were observed. The data were quantified on the basis of the following parameters: (1) time before courtship, time between placement of the pair in the mating chamber and the first display by the male; (2) duration of courtship, time between the initial display and the mount; (3) wing vibration time, cumulative time spent in wing display by the male; (4) duration of mount, time spent by male in mounted position.

The Wilcoxon two sample test was used to analyze the statistical significance of the differences observed between the two populations. Statistically significant differences between the populations were demonstrated for 3 parameters: time before courtship, $p<.001$; duration of courtship, $p<.02$; total wing vibration time, $p<.05$. Differences in the duration of the mount between the two populations were not statistically significant.

Mather, Wharton B. University of Queensland, Australia.
The genus Drosophila at Mt. Maquiling, Luzon, Philippines.

In February 1969 the genus Drosophila was sampled from fermenting banana baits. The baits were placed in lush vegetation on the slopes of the mountain.

The primary sorting of the flies yielded the results shown in Table I, and a sample of females from the melanogaster group when individually bred out, gave the results in Table II determined from males.

TABLE I
Primary Sorting

Species	Number	% of total
D. setifemur	134	13.7
D. pararubida	49	5.0
D. quadrilineata	3	0.3
melanogaster group	789	80.6
	975	

Table II
melanogaster group sample

Species	No.	% of mel. gr.	% of total
D. malerkotliana	49	14.3	11.6
pseudoananassae	14	4.1	3.3
D. bipectinata	2	0.6	0.5
D. takahashii	54	15.8	12.7
takahashii-like	5	1.5	1.2
D. gracilis	36	10.6	8.6
melanogaster group sp.	1	0.3	0.2
D. montium	2	0.6	0.5
montium subgroup sp. I	3	0.9	0.7
montium subgroup sp. II	4	1.2	1.0
D. truncata	32	9.4	7.6
rufa-like	140	40.1	32.3
	342		

It will be noted that the melanogaster group is very dominant. The immigrans group is represented by D. pararubida and D. setifemur. This is a similar situation to that in Sabah (Mather, 1968).

Cultures of the species from this collection have been preserved and are being studied in relation to cultures of the species from Sabah, New Guinea and Australia as regards chromosomal variation and reproductive isolation.

Reference: Mather, Wharton B. The genus Drosophila in Sabah. D.I.S. 43: 100.

Basden, E.B. Institute of Animal Genetics, Edinburgh, Scotland.

Drosophila mycethophila Goureau and D. testacea Goureau.

In 1865 Colonel C.C. Goureau published a second supplement to his "Les Insectes Nuisibles aux Arbres Fruitiers aux Plantes Potagères ....", a Paris pamphlet of 147 pages. On page 120 he described Drosophila mycethophila (spelt mycetophila on p. 141) from toadstools (champignons), it differing from D. transversa Meigen (sic), i.e. transversa Fln., by having only two, not four black marks on each abdominal segment. This could be D. histrio Mg. (1830), or even D. limbata v. Ros (1840), or D. kuntzei Duda (1924); and less likely to be D. phalerata Mg. (1830), which frequently (♀) has four-spotted segments.

On page 119 he describes D. testacea Goureau, also from champignons, it differing from transversa by the black third-antennal joint and clear transverse veins. This species is most probably D. cameraria Hal. (1833), and not D. testacea v. Ros (1840).

Goureau's specimens need to be examined to confirm their identity but they probably no longer exist and it is unlikely that his two toadstool species had not already been described, as suggested above. Goureau's name mycethophila should be made known, however, as D.E. Hardy has described D. mycetophila from Oahu (1965, Insects of Hawaii, 12: 376), which should now be given a new name. An apposite one would commemorate the 100 years between the two.

Falk, R. The Hebrew University, Jerusalem, Israel.

Evidence against the one-to-one correspondence between bands of the salivary gland chromosomes and genes.

The cytological location of a series of lethals that were induced in the proximal segment of the X-chromosome of D. melanogaster by Lifschytz & Falk (Mutation Res. 8: 147-155; 1969) was determined in an experiment in which various proximal segments of the X chromosome, of known cytological length, were tested for their capability to cover lethal effects. The segments of the X-chromosome were obtained from X-Y translocations produced by Nicoleti and Lindsley (Genetics 45: 1705-1760).

Females heterozygous for lethals that were mapped in the proximal segment were mated to males with the X-Y translocations. The recovery of hyperploid sons with the lethal chromosome and the X^P element of the translocation indicated that the lethal was located proximally to the known breakage point of the translocation. Of three translocations T(1;Y)14, T(1;Y)132, and T(1;Y)151 that all have their breakage point in 19F, the first two did not produce viable hyperploid males with even the most proximal lethals Q464 and P19. T(1;Y)151, on the other h hand, covered lethals Q463, P19, 3DES and Q464. It did not cover E54, Q414, w2, R-9-29 or AA33. This, its breakage point was at the "hot spot" at section 18 of the complementation map of Lifschytz & Falk (1969).

Since T(1;Y)14 and T(1;Y)132 give fertile males only in the presence of an additional free Y, the possibility had to be considered that T(1;Y)/Y males produced only gametes that carried either both elements of the translocation or only the free Y, i.e., that the translocation elements did not segregate.

To test this possibility the reciprocal mating FM4/T(1;Y)132 x $\underline{1}$/Y·mal$^+$ was tried with different lethals. In the mating with $\underline{1}^{Q463}$ 11 y B females were obtained among a total of 174 daughters. These y B females obtained from their mother the FM4 chromosome together with the X^D element of the translocation, i.e., they were due to non-disjunction of the translocation elements. They survived as they obtained from their father the Y·mal$^+$ chromosome. No hyperploid males with the lethal chromosomes were obtained in these matings. This proves that the absence of $\underline{1}^{Q463}/X^P.Y^S$ was indeed due to their lethality.

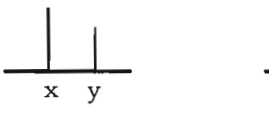
Since section 19F of the salivary gland chromosomes has at most six visible bands and since from the complementation map T(1;Y)151 and T(1;Y)132 are separated by at least 20 functional units (two more units were identified in this segment since the publication of the map) the minimum estimate of genes per band in this segment is three. These results exclude the possibility of a one-to-one correspondence between salivary gland chromosome bands and cistrons.

The work is part of a co-ordinate programme of research under the sponsorship of the International Atomic Energy Agency 752/CF.

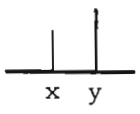
Dews, D. University of Oregon, Eugene, Oregon. A model for frequency-dependent mating success.

Frequency-dependent mating success has been observed in D. melanogaster by Petit (1958), in D. pseudoobscura by Ehrman (1966, 1967, 1968) and by Spiess (1968), in the D. willistoni species group by Ehrman and Petit (1968) and in D. persimilis by Spiess and Spiess (1969). These workers have found that when the ratio of two competing types is varied, the minority male type is often more successful than the majority male type. The types may differ by a mutation, by a chromosome inversion, by geographical region of collection or by development in different environments. Spiess (1968) has suggested a mechanism based on male-male interference to explain minority male advantage in which either male type has an advantage when rare and one type has an advantage when the two types are in equal numbers (case I). Spiess and Spiess (1969) note that this sort of mechanism does not explain the case in which both types have an advantage when rare and mating is random when the two types are present in equal numbers (case II). They suggest sense organ "adaptation" as a possible mechanism. Recently Ehrman and Spiess (1969) report that minority male advantage is eliminated in the top of double chambers if either rare type pairs or rare type males are in the bottom chamber. They conclude that male-male interactions seem to be ruled out in favor of the females' "recognition" of the relative frequency of the two male types. Ehrman (1969) has shown that mating success can be changed by air-borne stimuli.

This note presents a model which seems to be able to accommodate the various types of frequency-dependent mating advantage reported in the literature. The courtship behavior of the male must have a number of components which stimulate the female. Contact stimuli (chemical and mechanical) and distance stimuli (visual, air-borne chemical and air-borne mechanical) have been suggested by Spieth (1968) and others. It seems reasonable to expect two types differing by a mutation, by a chromosome inversion, by geographical region of collection, or by morphological or physiological features arising from development in different environments, to be quantitatively different with respect to at least several of the components of courtship. The first postulate of the model is that at least two components (x and y) of the male's courtship are present at different levels in each of the two competing types (A and B). The second postulate is that the female's level of excitation is increased by the male's continued delivery of the courtship components; when the level of excitation of both components reaches some threshold, she accepts the male courting her at that time. The third postulate is that the maximum component of the two lines must not be the same.

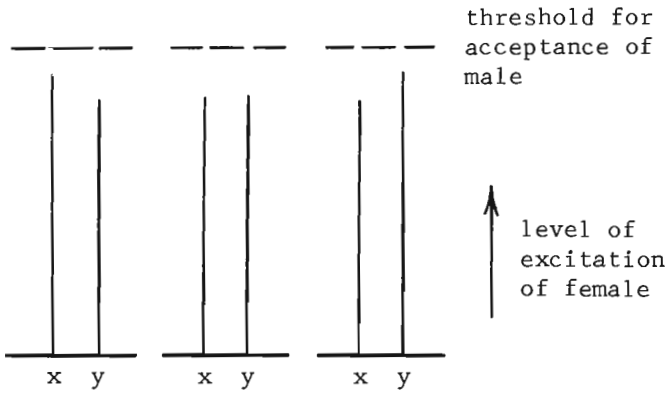


Type A males



Type B males

Fig. 1a. Postulated levels of two female-stimulating components (x & y) of male courtship (case II).



Type B male rare | Type A & Type B males equally frequent | Type A males rare

Fig. 1b. Level of excitation of female after several courtships.

If type A males have higher x but lower y components than type B males and if the difference between the x components of types A and B is about equal to the difference between the y components of types A and B (see Fig. 1a), then the model predicts case II. When type A males are common and type B males are rare, a common type A male is more likely to court a given female than is a rare type B. After some time type A males will have furnished threshold or near-threshold amounts of stimulation in component x, but component y will not yet by near threshold. At this time (see Fig. 1b) the rare type B male is more capable than the common type A male of raising component y to the threshold level and so is more likely to be accepted by the female. If we change the frequency of the two types of males such that type B is common and type A is rare, we get (after some courtship activity has occurred) the y component near threshold and the x component not yet near threshold. At this time a rare type A male is more capable than a common type B male of raising component x to the threshold level and so is more likely to be accepted by the female. When the two male types are equally frequent, the levels of a female's x and y components will rise towards threshold at an equal rate. Neither male type will have an advantage. An example of case II was reported by Ehrman (1966) with the mutant delta in competition with wild type.

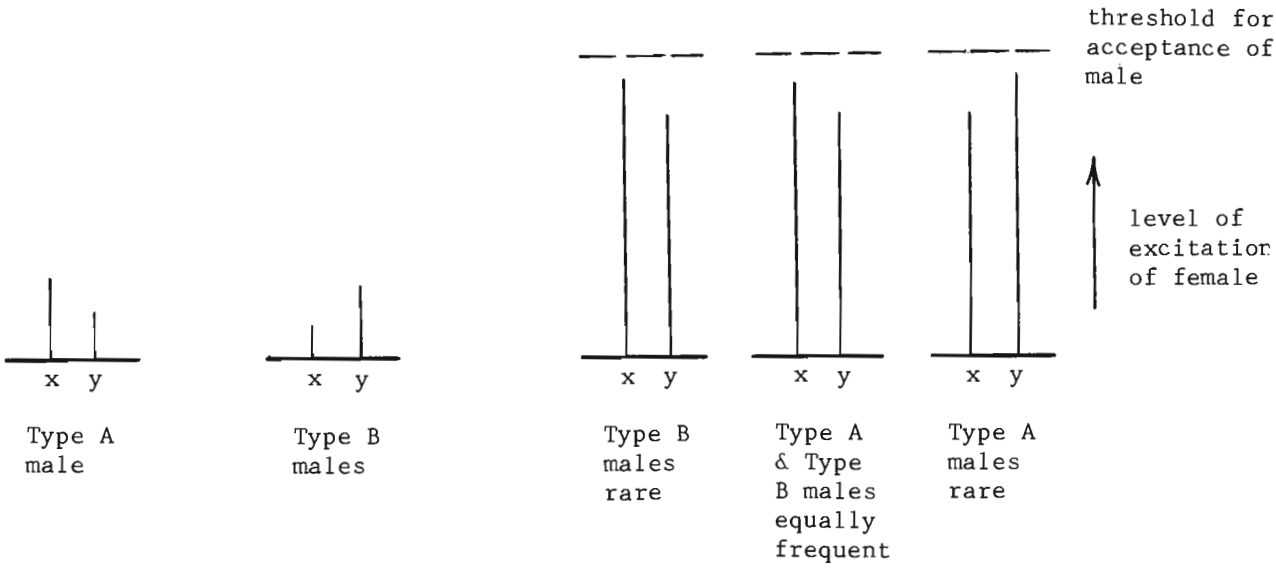


Fig. 2a. Postulated levels of two female-stimulating components (x & y) of male courtship (case I).

Fig. 2b. Level of excitation of female after several courtships.

If type A males have higher x and lower y than type B males (as before) but the total amount of stimulation provided by type A males is somewhat greater than that provided by type B males, then case I is expected. An example was reported by Ehrman (1966) using AR/AR raised at 16° and 25°C.

A third case giving two possible outcomes seems possible if type A and B males have equal y but unequal x components (case III). As before the maximum component of the two lines must not be the same. The prediction in this case is that when type A males are rare they have an advantage and that when type B males are rare, mating is random. When the two male types are present in equal numbers, there are two possible predictions: (1) If the total x of types A and B is greater than the total y of types A and B, then mating is random (see Figs 3a & 3b). (2) If the total of the x of types A and B is less than or equal to the total y of types A and B, then A males are expected to have an advantage (see Figs 4a & 4b). An example of case III(2) was reported by Ehrman (1966) using the mutant orange and wild type.

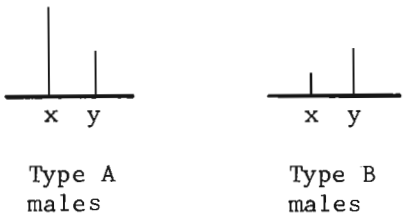


Fig. 3a. Postulated levels of two female-stimulating components (x and y) of male courtship (case III (1)).

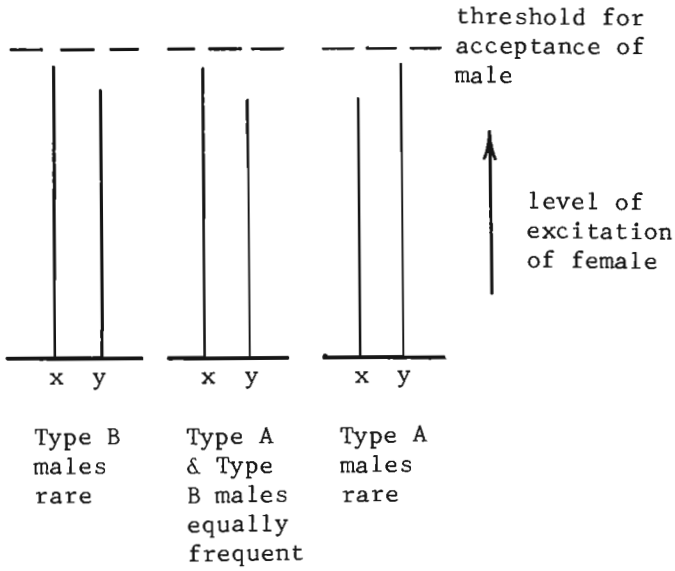


Fig. 3b. Level of excitation of female after several courtships.

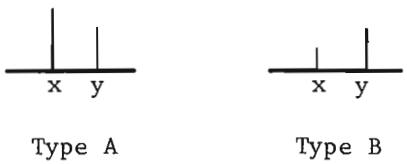


Fig. 4a. Postulated levels of two female-stimulating components (x & y) of male courtship (case III (2)).

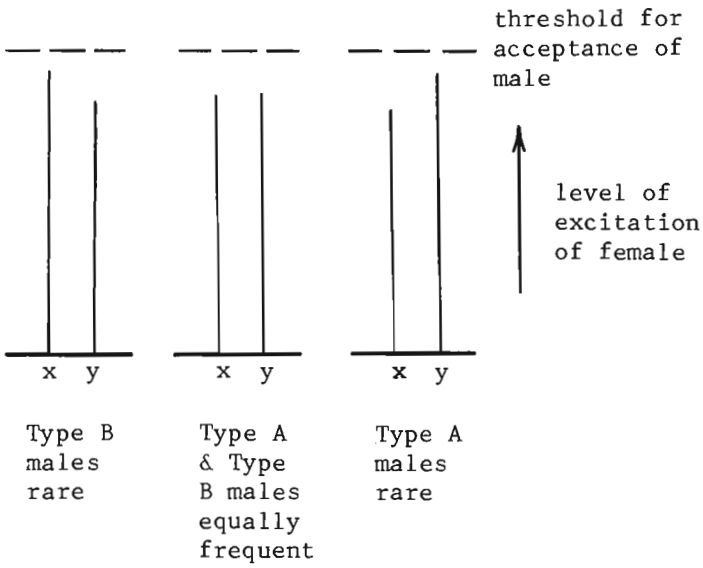


Fig. 4b. Level of excitation of female after several courtships.

References: Ehrman, L. 1966, Anim. Behav., 14: 332-339. Ehrman, L. 1967, Amer. Natur., 101: 415-425. Ehrman, L. 1968, Genet. Res., 11: 135-140. Ehrman, L. 1969, Evolution, 23: 59-64. Ehrman, L., and C. Petit, 1968, Evolution, 22: 649-658. Ehrman, L., and E.B. Spiess (1969) in press. Petit, C. Bull. Biol. 92: 248-329. Spiess, E.B. 1968, Amer. Natur. 102: 363-379. Spiess, L.D. and E.B. Spiess, 1969, Amer. Natur. 103: 155-172. Spieth, H.T. 1968, Evolu. Biol., 2: 157-193.

Barthelmess, I.B. and F.W. Robertson. Department of Genetics, University of Edinburgh, Scotland. Quantitative relations between variation in red eye pigment and related pteridine compounds in D. melanogaster.

Lines selected for high and low red eye pigment, various lines derived by chromosome exchange, inbred lines etc., have been used for measurements of quantitative differences in fluorescing pteridines, which have been separated by chromatography. The genetic situation was different in high lines, when compared with lines selected for low pigment or inbred without selection. The high lines remained heterozygous and showed dominance and epistasis in crosses to the unselected stock while, in low lines, fixation for genes which reduce pigment content had taken place. Increase in pigment content led to increase of all the observed precursors while the low and inbred lines showed accumulation of certain pteridines and reduction of others. The genetic behaviour of the fluorescing compounds parallels that of the red pigment. The reduced pigment content in both low and inbred lines could be accounted for by reduced enzyme activity in later stages of the pathway leading to red pigment, while the positive relation between precursors and pigment content in high lines could be due to an increase in early precursors. Many of the general features of the inheritance of differences in pigment content recall those shown by body size.

Banerjee, M. and A.S. Mukherjee. University of Calcutta, India. Effect of split-dose X-irradiation on fractional mutations in D. melanogaster: preliminary results.

In order to examine the effect of split X-ray dose on fractional mutations in D. melanogaster (Oregon R+) four sets of experiments were performed: (i) 3 KR given to 48 hrs. old adult males, (ii) 1 KR given to a batch of white pupae and again the males which eclosed from these white pupae were given 2 KR when 48 hrs. old, (iii) 1 KR given to white pupae and the males emerging from them were utilized and (iv) 1 KR given to 48 hrs. old males (the dose rate varying from 36-40 R/5 sec., with Picker's X-ray machine operated at 110 KV, 4mA, using 0.25mm Al filter). The males were crossed to "maxy" ♀♀ (for 4 days) and the F_1 females were examined for visible mutants for the 15 loci. The results of the preliminary series of experiments are presented in Table 1. It is clear that as expected the frequency of whole-body mutations is higher with 3 KR than with 1 KR (singles), but the proportion of fractionals

TABLE I. RESULTS OF THE FOUR EXPERIMENTS SHOWING CHANGES IN FRACTIONAL MUTATION FREQUENCY IN DIFFERENT CONDITIONS

Expt. No.	Total Dose	Stages of Irradiation	Total No. of F_1 ♀♀ Examined	Whole Body Mutants No.	Whole Body Mutants %	Fractional Mutants No.	Fractional Mutants %	Whole Body/ Fractional Ratio	% of Visibles
1	1 KR	48 hr adult ♂♂	3080	1	0.032	2	0.065	0.5	0.097
2	1 KR	white pupae	8708	10	0.114	8	0.092	1.25	0.206
3	3 KR	48 hr adult ♂♂	4159	15	0.36	8	0.19	1.87	0.553
4	3 KR (1 KR + 2 KR)	white pupae & 48 hr adult ♂♂	5551	14	0.25	7	0.125	2.0	0.377

among the total mutants is higher with 1 KR than with 3 KR (singles). In addition, proportion of fractionals as compared to whole-body tends to be higher in samples of sperms (Expt. 1) than in samples of spermatids and spermatocytes (Expt. 2). When 3 KR is fractionated and given in two stages (Expt. 4), the total mutation frequency is decreased as compared to that in 3 KR single dose series (Expt. 3), but the net frequency of fractionals (whole-body to fractional ratio) appears to have been more in the former than that expected for spermatids or sperms alone with 3 KR. However, it appears that this effect of split-dose depends upon the specific stage of irradiation. The details of these works are in progress.

(Work supported by a Fellowship from Lady Tata Memorial Trust to the first Author).

Nozawa, K. Nagoya University, Japan.

Estimation of the effective size in D. experimental populations.

In a previous experiment the author (1963) estimated the effective population size by measuring the random fluctuation of frequencies of a mutant gene in competition with its wild type allele in D. melanogaster, obtaining the ratio of effective (N) to apparent size (N') (N/N' ratio) 35-62% when N'<10 and 22-30% when N'>10. These values seemed to be too small in comparison with the experimental results of Kerr and Wright (1954a and b), Wright and Kerr (1954), Crow and Morton (1955) and Buri (1956), all of which gave the N/N' ratio 56-83%. A new experiment was carried out in order to obtain a more accurate estimate of effective size in relation to change in parental population density. In this experiment the effective numbers of female and male parents were estimated separately by using sex-linked marker genes.

Females of Muller-5 stock (w^a B/w^a B) were mated with the Oregon wild type males (+/Y). Ten F_1 females (w^a B/+) were mated with the mixture of 5 F_1 males (w^a B/Y) and 5 wild type males (+/Y). In the next generation (F_2) three kinds of female genotype, w^a B/w^a B, w^a B/+ and +/+, and two kinds of male genotype, w^a B/Y and +/Y, appeared and were counted, so that the frequencies of w^a B chromosomes in the female (q_f) and male (q_m) flies were calculated. One, 4, 10, 20 or 50 pairs of females and males which were sampled randomly from the F_2 population were allowed to breed in a culture bottle of 3 cm. diameter which contained corn-meal agar media added with 0.2 cc. of 5% suspension of dry yeast (manufactured by Oriental Co.). The F_3 flies emerged from the culture bottle were counted and the frequencies of w^a B chromosomes in the female (q'_f) and male (q'_m) flies were obtained. All the F_2 and F_3 flies emerged were counted in each culture bottle.

From the above chromosome frequencies, the values of variables $\delta Q_f = [(q'_m-q_f)-\overline{(q'_m-q_f)}]/\sqrt{q_f(1-q_f)/2N'_f}$ and $\delta Q_m = [(2q'_f-q_f-q_m)-\overline{(2q'_f-q_f-q_m)}]/\sqrt{q_m(1-q_m)/N'_m}$ were calculated, where N'_f and N'_m were the numbers of female and male F_2 flies, respectively, used in the matings for obtaining F_3 populations. Being fixed the values of N'_f and N'_m, the groups of values of δQ_f and of δQ_m were expected to form approximate normal distribution with mean 0 and standard deviation $\sigma_{\delta Q_f}$ and $\sigma_{\delta Q_m}$, respectively. Then, the average effective numbers of female ($\overline{N}_f$) and male ($\overline{N}_m$) parents were estimated as $N'_f/\sigma^2_{\delta Q_f}$ and $N'_m/\sigma^2_{\delta Q_m}$, respectively.

Table 1 shows the results of experiments. From this table it can be seen that the effective numbers of female and male parents enlarge along with the increase in parental popu-

Table 1. Estimations of effective numbers of female (A) and male (B) parents.

(A) No. of female F_2 flies (N'_f)	No. of culture bottles (n)	$\overline{\delta Q}_f$	$\sigma_{\delta Q_f}$	Effective no. of female parents ($\overline{N}_f = N'_f/\sigma^2_{\delta Q_f}$)	$\overline{N}_f/N'_f$ (%)
1	215	-.018±.130*	.965±.092*	1.073(.895 ~ 1.312)**	107
4	335	-.013±.110	1.008±.076	3.937(3.404 ~ 4.605)	98
10	109	+.011±.212	1.117±.150	8.015(6.229 ~10.695)	80
20	73	+.004±.410	1.756±.290	6.486(4.777 ~ 9.306)	32
50	325	-.020±.290	2.626±.206	7.247(6.234 ~ 8.537)	14

(B) No. of male F_2 flies (N'_m)	No. of culture bottles (n)	$\overline{\delta Q}_m$	$\sigma_{\delta Q_m}$	Effective no. of male parents ($\overline{N}_m = N'_m/\sigma^2_{\delta Q_m}$)	$\overline{N}_m/N'_m$ (%)
1	215	-.005±.158*	1.164±.112*	.738(.614 ~ .903)**	73
4	335	-.007±.128	1.172±.090	2.912(2.396 ~ 3.416)	72
10	109	+.017±.224	1.173±.158	7.267(5.644 ~ 9.704)	72
20	73	+.038±.380	1.672±.276	7.154(5.270 ~10.262)	35
50	325	+.097±.274	2.470±.192	8.189(7.055 ~ 9.635)	16

* 95% confidence limit.

** In parenthesis a range of effective number corresponding to the 95% confidence limit of $\sigma_{\delta Q}$ is given.

lation density when the number of parental pairs is less than 10, but that the effective numbers are almost constantly between 7 and 8 irrespective of the parental density when the number of parental pairs is more than 10. Therefore, it is suggested that in the Drosophila mating population kept in a closed culture bottle the effective population size has a certain maximum level which would be determined by the volume of bottle, area of culture media and/or amount of food for larvae. This result means also that the N/N' ratio can be reduced infinitely by increasing in the parental population density. Any statistically significant difference could not be observed between the effective numbers of female and male parents allowed to breed in a culture bottle.

References: Nozawa, K. 1963, Japan. Jour. Genet. 38: 6; Kerr, W.E. and Wright, S. 1954a, Evolution 8: 172; Kerr, W.E. and Wright, S. 1954b, Evolution 8: 293; Wright, S. and Kerr, W.E. 1954, Evolution 8: 225; Crow, J.F. and Morton, N.E. 1955, Evolution 9: 202; Buri, P. 1956, Evolution 10: 267.

Bos, M. University of Groningen, Genetics Institute, Haren (Gn.), The Netherlands.

The influence of disruptive selection on body size in D. melanogaster.

In a previous report (DIS 44: 105, 1969) it was shown that stabilizing selection (S) on thorax length in D. melanogaster did not have an effect on the phenotypic variance, calculated as squared coefficients of variation ($c.v^2.$). In both S-lines the mean thorax length decreased about 6% below the control level (C). In the two D⁻-lines (disruptive selection with compulsory mating of opposite extremes) $c.v^2.$ increased considerably. In D⁻2 no change of mean size occurred, in D⁻1 there was only a slight increase after G 23 (Table 1).

Table 1. The effects of stabilizing and disruptive selection on phenotypic variance and mean.

	$c.v^2.$							mean size females (1/100 mm.)			
	G 0	5	10	15	20	25	30	G 0	10	20	30
C 1	6.50	6.10	9.24	7.78	12.18	7.78	6.15	108.7	109.1	110.7	111.3
C 2	6.50	5.15	5.95	9.36	7.51	----	8.41	108.7	108.6	105.3	108.4
S 1	6.50	4.12	5.38	11.56	8.64	10.56	----	108.7	106.9	102.2	-----
S 2	6.50	3.76	17.30	10.43	13.40	5.43	----	108.7	107.1	105.3	-----
D⁻1	6.50	8.82	15.84	13.40	28.62	31.47	18.32	108.7	107.9	108.6	112.6
D⁻2	6.50	7.78	12.39	20.79	20.70	19.98	24.31	108.7	108.2	109.4	108.1

Progeny tests (table 2) show that the increase in the phenotypic variance in D⁻1 is a consequence of an increase in the residual variance (environmental variance and/or genetic interaction). The increase of the phenotypic variance in D⁻2 is a consequence of an increase in additive genetic variance.

Table 2. Heritabilities and the composition of the phenotypic variances ($c.v^2.$) in the base population (B), the control lines (C) and in the stabilizing (S) and disruptive (D⁻) selection lines.

	B	C 1		C 2		S 1	S 2	D⁻1	D⁻2
	G 0	G 19	G 30	G 19	G 30	G 19	G 19	G 30	G 30
Phenotypic variance	6.50	12.46	6.15	7.08	8.41	8.01	7.02	18.32	24.30
Heritability	0.53	0.34	0.24	0.25	0.18	0.32	0.31	0.19	0.81
Additive genetic variance	3.45	4.24	1.48	1.77	1.51	2.56	2.18	3.48	19.68
Residual variance	3.05	8.22	4.67	5.31	6.90	5.45	4.84	14.84	4.62

The difference between the two D⁻-lines is corroborated by the result of divergent directional selection started from G 32. After four generations divergence (♀♀) between the high and the low line is 18.9 units in D⁻2 and only 8.1 units in D⁻1 (1 unit = 1/100 mm).

Policansky, D. University of Oregon, Eugene, Oregon. Three sperm sizes in D. pseudoobscura and D. persimilis.

All males examined from several laboratory stocks and wild populations of D. pseudoobscura and D. persimilis were found to have three distinct sizes of sperm (fig. 1). Two of the stocks of D. pseudoobscura came from San Diego, two of the stocks of D. persimilis came from Texas and Chicago and the wild populations were from California, Oregon and Washington. The sperm were measured by tracing the lengths on photographs; individual sperm of the three lengths were 0.31mm, 0.10mm, a and 0.05mm long. Yanders and Perras (DIS 34:112) reported lengths ranging from 0.295 to 0.304mm in D. pseudoobscura. Dobzhansky (1934) reported lengths of 0.4-0.5mm for the same species.

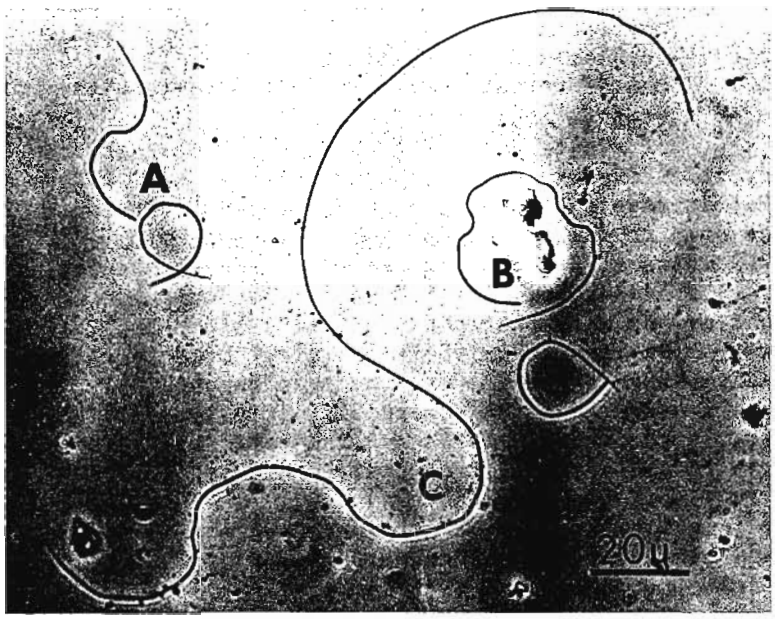


Fig. 1. Sperm of D. pseudoobscura (sh;or, Eugene).

A whole testis of D. pseudoobscura (sh;or, Eugene) was stained in Feulgen reagent and squashed. Sperm bundles of the three different sizes were seen and measured as described above. The sperm bundles were about 0.30mm, 0.10mm and 0.05mm long. These lengths correspond with the lengths of the free sperm. All the sperm in any bundle appear to be the same size, indicating that different size sperm are the product of different meiotic cells.

Under dark field illumination all three sizes show distinct pink-stained areas presumed to be DNA. These appear light in photographic prints (fig. 2). In the two shorter sizes (A and B) the stained area is at the tip of the sperm. In the longest size (C) the stained area is somewhat removed from the tip and less clearly defined.

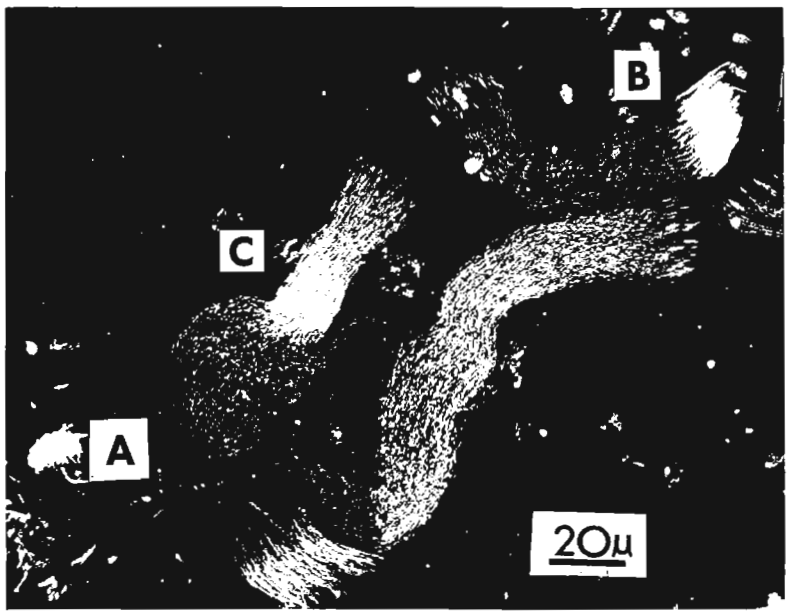


Fig. 2. Stained sperm bundles of D. pseudoobscura (sh;or, Eugene).

The fact that the long sperm stain differently from the shorter ones argues against the possibility that the shorter ones are merely fragments; also all three sizes are motile and can be found in the storage organs of females.

I gratefully acknowledge the assistance of John Ellison.

Reference: Dobzhansky, Th., 1934. Studies on hybrid sterility. I. Spermatogenesis in pure and hybrid D. pseudoobscura. Z. Zellforsch. und mikroskop. Anat. 21: 169-223.

Footnote: Correspondence with Dr. R.A. Beatty, Institute of Animal Genetics, Edinburgh,

subsequent to submission of this article, indicates that polymorphism in the sperm of the obscura group was observed in his laboratory by N.S. Sidhu (Ph.D. Thesis, Edinburgh, 1963) and that these observations will appear in a paper to be published shortly in the Proceedings of the Royal Society of Edinburgh.

Šrám, R.J. Department of Genetics, University of Edinburgh, Scotland.
The influence of storage on the viability of zygotes carrying chromosomal aberrations.

When spermatozoa from D. melanogaster treated with some chemical mutagens are stored in untreated females, the frequency of structural chromosomal changes increases considerably. One of the possible explanations of this storage effect may be a change in the viability of zygotes carrying chromosomal aberrations.

To answer this question, two reconstruction experiments were carried out.

In the first experiment, the effect of storage on the viability of zygotes carrying a translocation was tested. Translocations used in this experiment were induced by EI and involved 2nd and 3rd chromosome. About 10 males (T/bw;st) from each translocation culture were mated for three days to bw;st virgins and discarded; fertilized females were transferred to fresh vials every three days until eight broods were obtained. In each brood the ratio of homozygous bw;st and T/bw;st was scored. Thirteen translocations were tested in this way. The ratio was not affected by storage and remained approximately the same through all 8 broods.

In the second experiment 15 EI induced sex-linked lethals were similarly tested. Females of the constitution 1/M-5 were individually mated with M-5 males and the ratio of M-5/M-5 and 1/M-5 scored before and after storage. As in the previous case storage did not affect this ratio.

Since the viability of structural or lethal heterozygotes does not change with storing, it can be concluded that storage effect and viability are not causally related.

(This work was supported by a Grant from the University of Edinburgh.)

Gearhart, J. Cornell University, Ithaca, New York.
Quantitation of drosopterins in Lobed^2 eyes of D.m.

It has been reported by Taira and Nawa (D.I.S. 33:167) that red pigments in the mutants BB, bar-3, L^2, and Dp, decrease in direct relation to eye size. Using the technique of cellulose acetate electrophoresis with single eyes (Gearhart and MacIntyre, in press), I have found that within L^2 this direct relationship does not exist. Ten eyes were chosen at random from an L^2 stock. A visual estimation of eye size was obtained by drawing the eyes and then cutting out and weighing the paper (mg). With the electrophoretic technique, results are expressed as mm^2 (area under the absorption curve at 520 nm).

Eye No.	Weight (mg)	% Wild Type*	Densitometric Reading (mm^2)	% Wild Type**
1	235.6	98	367.0	98
2	185.0	77	332.0	89
3	184.3	77	363.5	96
4	117.0	49	244.5	65
5	188.5	79	376.0	100
6	175.0	73	329.0	88
7	136.3	57	267.5	71
8	96.6	40	294.0	78
9	176.9	74	291.0	78
10	217.0	90	307.0	82

* Wild type 240 mg (average of 4 eyes) ** Wild type 375 mm^2 (average of 4 eyes)
r = (0.69)

It is evident from this data that no direct relationship exists between eye size and amount of red pigment within the L^2 mutant.

Work supported by PHS Training Grant No. GM-01035.

Félix, R., J. Ramírez, V.M. Salceda and A. de Garay Arellano. Comisión Nacional de Energía Nuclear, Mexico City, Mexico.

Effect of butylated hydroxytoluene on the mean life span of D. melanogaster.

A theory has been advanced (Harman, 1956) on the deleterious side effects of free radicals on ageing. Such free radicals arising from enzymatic and non-enzymatic sources would be expected to produce a multiplicity of harmful changes throughout a biological system (Harman 1968). Mutation, cancer and ageing are three related processes which arise spontaneously in nature and are also induced by irradiation (Hempelmann and Hoffman, 1953). The universality of the ageing phenomenon suggests that the reactions which cause it are basically the same in all living things (Harman, 1956). It is believed that one mechanism of irradiation effect is through liberation of OH and HO_2 radicals (Stein and Weiss, 1948). The effects produced by endogenously formed free radicals would not be expected to be identical in all respects to those resulting from similar radicals arising by irradiation, because of differences in concentration and distribution of radicals, and of local availability and concentration of substances capable of inhibiting their effects.

Thus, radiation-induced free radicals are concentrated along paths randomly distributed throughout the entire cell, whereas those of endogenous origin would be expected for the most part to arise in and be concentrated around relatively localized areas such as the mitochondria (Harman, 1962).

Free radicals, spontaneously produced and accumulating with time, may give rise to damage in proportion to their concentration. Spontaneous accumulations of free radicals from autooxidation processes in organic fats, oils or other easily oxidizable compounds are known examples of radical accumulation with time (Dimmich et al., 1961; Lion et al., 1961; Miyagawa et al., 1958).

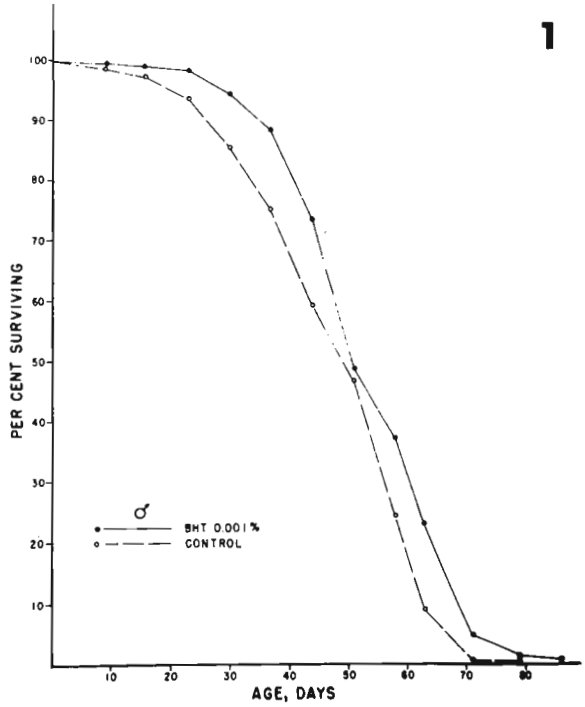
The free radical reaction inhibitor butylated hydroxytoluene (2,6-di-tert-butyl-p-cresol) has been shown to increase significantly the normal life span of male LAF_1 mice when added to the daily diet (Harman, 1968). These data lend further support to the possibility that endogenous free radical reactions contribute significantly to ageing.

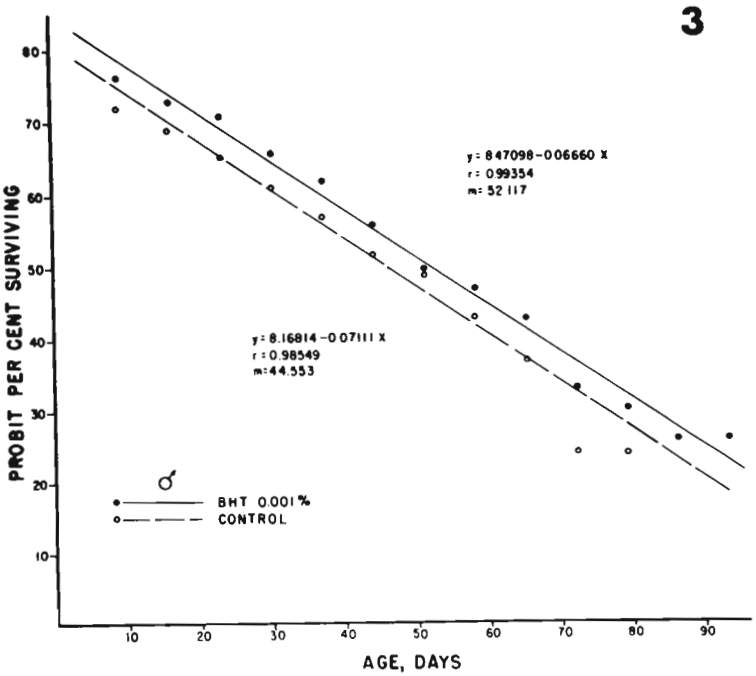
Drosophila is an especially adequate experimental organism for investigating the problems of radiation-induced life shortening and natural ageing. It is not yet well known to what extent the causes underlying the modification of the life span in insects and mammals are the same, but the similarity of experiments and results in both groups justify the hopes that research on Drosophila may throw some light on the processes concerning ageing and induced modifications of life span in mammals.

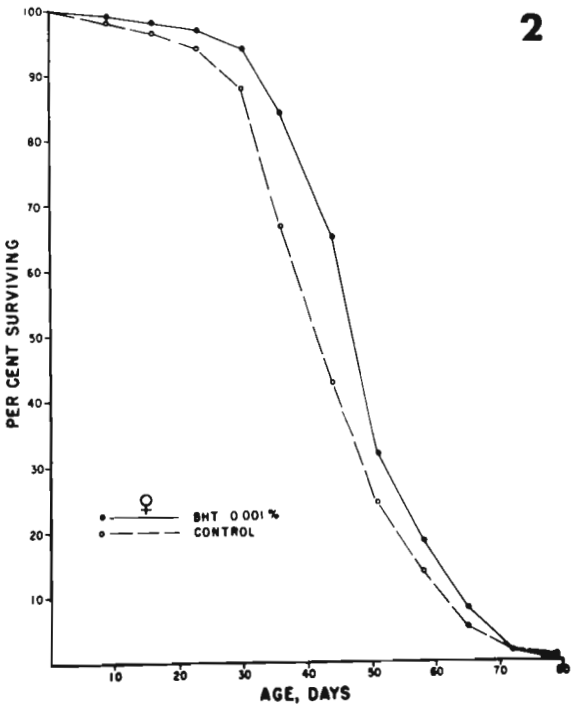
The purpose of this study was to determine whether BHT has an effect on Drosophila similar to the lengthening of the normal life span observed in mice. The stock y/sc^8Y was used in order to carry on separated records of mortality of males and females, as the females appear yellow in contrast to the males, which have non-yellow bodies since they carry the normal dominant allelomorph of yellow in the sc^8 inversion of their Y chromosome. All the cultures were kept at a temperature of 25°±1°C. Groups of 50 males and 50 females collected from 0 to 24 hours after emergence from the pupal stage were shaken into bottles with fresh medium. Counting of dead flies was done every other day after the living flies were transferred to new cultures. In this way the spoiling of the medium was avoided. All transfers were made without etherization to avoid possible interference with viability, as well as the sticking of the flies to the new culture medium. The counting was carried on until the last fly's death was recorded.

For our purpose the experiment was divided into three groups, with six bottles per group. As was stated before, every bottle contained 100 newly emerged adults at the beginning of the experiment: Group I, adults treated with a concentration of 0.01% BHT; Group II adults treated with a concentration of 0.001% BHT; and Group III, adults not treated but otherwise handled as the treated flies. BHT was dissolved in 96% ethanol before being added to the regular agar cornmeal medium regularly employed in the laboratory. The adult flies of Group I and Group II were maintained throughout life in the medium containing BHT.

After adding the data obtained from each of the six series or bottles of each group, the mean life span for the treated groups and its control was determined in the following manner: a sigmoid graph was obtained by plotting per cent surviving against time (Figs. 1 and 2). Using the probit transformation a second graph was drawn to situate in the time scale the point (mean life span) corresponding to the mid point in the scale of per cent surviving (Figs. 2 and 3). The values of the mean life span for each of the groups are shown in Table I. BHT (0.001%) incorporated into the food medium of Drosophila prolonged the mean life span of males from 44.55 to 52.12 days, and the mean life span of females from 43.12 to 47.42 days







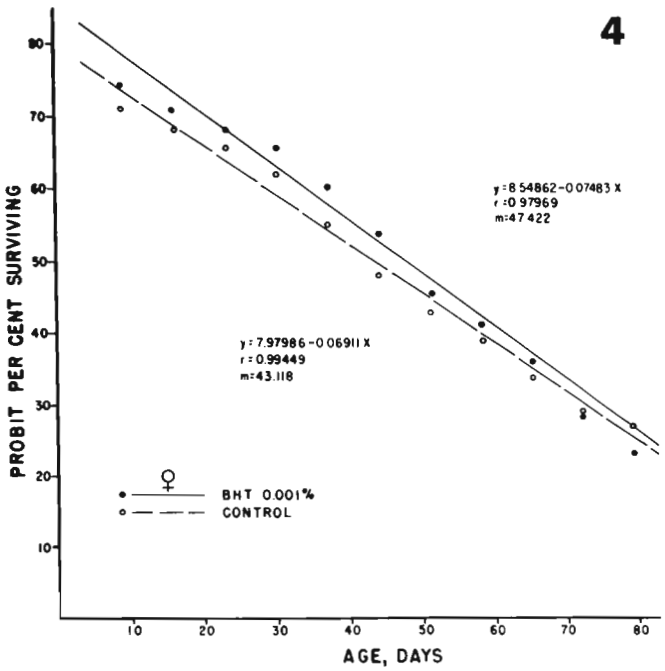


Fig. 1. Mortality of Drosophila males: effect of butylated hydroxytoluene (0.001%).
Fig. 2. Mortality of Drosophila females: effect of butylated hydrozytoluene (0.001%).
Fig. 3. Probit analysis of the effect of butylated hydroxytoluene (0.001%) on the mortality of Drosophila males.
Fig. 4. Probit analysis of the effect of butylated hydroxytoluene (0.001%) on the mortality of Drosophila females.

an increase of 0.17% and 0.10% respectively.

TABLE I

Effect of butylated hydroxytoluene (BHT) on the mean life span of Drosophila melanogaster.

	males		females	
	m.l.s.	p.c.i.	m.l.s.	p.c.i.
Control	44.55		43.12	
BHT (0.01%)	46.97	0.05	43.33	0.00
BHT (0.001%)	52.12	0.17	47.42	0.10

m.l.s.: mean life span in days p.c.i.: per cent increase

References: Dimmich, R.D., Hollis, D.P., and Heckley, J., 1961, Nature 192: 776-777. Harman, D., 1956, J. Gerontol. 11: 298-300. Harman, D., 1962, Rad. Res. 16: 753-763. Harman, D., 1968, J. Gerontol. 23: 476-482. Hempelmann, L.H. and Hoffmann, J.G., 1953, Ann. Rev. Nuclear Sci. 3: 369-389. Lion, M.B., Kirby-Smith, J. and Randolph, M.L., 1961, Nature 192: 34-36. Miyagawa, I., Gordy, W., Watabe, N. and Wilbur, K., 1958, Proc. Natl. Acad. Sci. U.S.A., 44: 613-617. Stein, G. and Weiss, J., 1948, Nature 161: 650.

Barnett, B.M. and E.R. Muñoz. Comisión Nacional de Energía Atómica, Buenos Aires, Argentina. Effect of low temperature on inseminated females.

In the course of an investigation on the effect of radioprotectors at the genetical level and the influence of low temperatures, some data was collected on the viability of sperm in inseminated females exposed to 0°C during various periods of time. The general procedure was as follows: four day old Canton S wild type males and females were mass mated for two days; the flies were then put in cold storage without etherizing. After the treatment the males were discarded and the females put in vials in groups of ten. Nine daily broods were made and when the progeny were counted, males and females were scored separately. The length of exposure to 0°C varied between 1 hour and 16 hours. When similar results were obtained, the data of the successive treatments was pooled, thus group I includes the controls and 1, 1.5 and 2 hr treatments. Group II includes treatments from 2.5 to 10 hr and group III includes treatments from 12 to 16 hr. The reduction in the number of offspring in the successive broods can be seen in Table I, where broods 1, 5 and 9 were taken as representative of the general pattern.

When the total progeny is considered, the reduction in number of offspring as a function of length of exposure to 0°C leads to a somewhat different grouping, as can be seen in Table II.

TABLE I

	offspring/female (average)		
Brood	Group I	Group II	Group III
1st	8.5	2.4	1.7
5th	1.6	1.4	0.7
9th	1.0	0.8	0.6

TABLE II

Treatment	Average Offspring/female	Total progeny	No. treated females
Controls & 1 hr	31.3	5794	189
1.5 to 5.5 hr	20.3	21052	1107
6 to 9 hr	14.4	6540	493
10 to 16 hr	9.0	4457	551

The viability of the females was quite unaffected by the cold storage and no alterations were found in the male/female proportions of the progeny in any of the treatments.

Hunter, A.S. Universidad de la Región Centro-Occidental, Barquisimeto, Venezuela.
Drosophila of Venezuela.

Collections of Drosophila have been made in various parts of Venezuela, and of those positively identified, several are not included in the list of 34 species published by Cova García and Suárez (1962).

In the state of Lara at a region known as Hato Arriba (elevation 1,900 meters) three species of the mesophragmatica group have been found. These are D. viracochi, D. mesophragmatica and D. gasici. Virgins of iso-female lines of each species were crossed with known Colombian lines for identification, and gave fertile F_1. This is the most north-easterly extension of the geographic range of these Drosophila which are endemic species restricted to the Andes. D. dreyfusi and D. araicas were also collected in this site and were identified by comparison with the drawings of genitalia of Breuer and Pavan (1954) and Pavan and Nacrur (1950).

A representative of the sub-genus Sordophila has been found in the Henry Pittier National Park at Rancho Grande, and also in the vicinity of Barquisimeto. This D. acanthoptera is a strikingly different little fly with very broad cheeks, small dark eyes and unusual wings just as described and pictured by Wheeler (1949). The following species were also found in Rancho Grande: D. griseolineata, D. guarumunu, D. setula, D. krugi and D. sucinea. The last two are easily identified by the genitalia which are illustrated in the work of Breuer and Pavan (1954) and Malogolowkin (1952). It is interesting that among iso-female lines of the D. sucinea 20% produced all female offspring and can only be maintained by adding males from other lines.

In the vicinity of Barquisimeto, D. moju, D. fulvimacula, D. paranaesis, D. cardini, D. canalinea and D. campestris have frequently been collected. Dr. Dobzhansky and his collaborators have found the sibling species, D. equinoxialis, D. tropicalis and D. willistoni as well as the D. paulistorum listed by Cova García and Suárez.

References: Breuer, M. and Pavan, C., 1954, Rev. Brasil Biol. 14: 465. Cova García, P. and Suárez, O., 1962, Revista Venezolana de Sanidad y Asistencia Social XXVII: 317. Malogolowkin, C., 1952, Rev. Brasil Biol. 14: 465. Pavan, C. and Nacrur, J., 1950, Dusenia I: 263. Wheeler, M., 1957, U.T.P. 5721: 79.

Postlethwait, J. H. and H. A. Schneiderman. Case Western Reserve University, Cleveland, Ohio.
Effects of an ecdysone on growth and cuticle formation of D. imaginal discs cultured in vivo.

When the imaginal discs of D. melanogaster are implanted into larvae, they metamorphose when the larvae metamorphose. When they are implanted into adult abdomens, the discs may grow, but they do not metamorphose. The present experiments were designed to see whether injection of an ecdysone into an adult fly will cause implanted imaginal discs to metamorphose.

Whole leg discs from mature third instar larvae were injected into fertilized adult females. The hosts then received single or repeated injections of ecdysterone (=20-hydroxyecdysone) in 10% ethanol in Ringer. In a typical experiment, one group of flies bearing implants received either 7.2 or 720 micrograms of ecdysterone/gm fly weight in a single dose, or in six equal installments over a period of eleven days. In none of the singly injected flies did the discs grow significantly or metamorphose. In contrast, multiply treated implants increased in size more than threefold, and most secreted some cuticle, but failed to metamorphose.

To cause metamorphosis, repeated doses of higher concentrations of ecdysterone were necessary. Thus 3600 micrograms/gm given over an eleven day period in three injections caused metamorphosis in thirteen of thirteen implants. These metamorphosed implants were completely covered with cuticle, and formed bristles, claws, tibial sensory organs, sex combs, and sensilla trichodea.

These results indicate the following: 1) lack of ecdysone in uninjected adult flies accounts for the absence of metamorphosis in implanted discs. 2) Ecdysterone is inactivated rapidly in the adult. 3) To cause either growth or metamorphosis, ecdysone is needed as a sustained stimulant, not merely as a trigger (hence the effectiveness of repeated doses). 4) Low concentrations of ecdysone stimulate the enlargement of discs, whereas, 5) high concentrations stimulate cuticle secretion and metamorphosis.

The discovery of a simple chemical method of regulating the growth and metamorphosis of imaginal discs promises to simplify developmental studies with Drosophila.

Blaney, W. M. Birkbeck College, London, England. Some observations on the sperm tail of D. melanogaster.

This investigation was prompted by the observation (Oster, Duffy and Binnard, 1966) that the sperm tail of D. melanogaster consisted of two distinct, and in some circumstances separable, filamentous units. It was felt that electron microscopic study ought to clarify this suggestion.

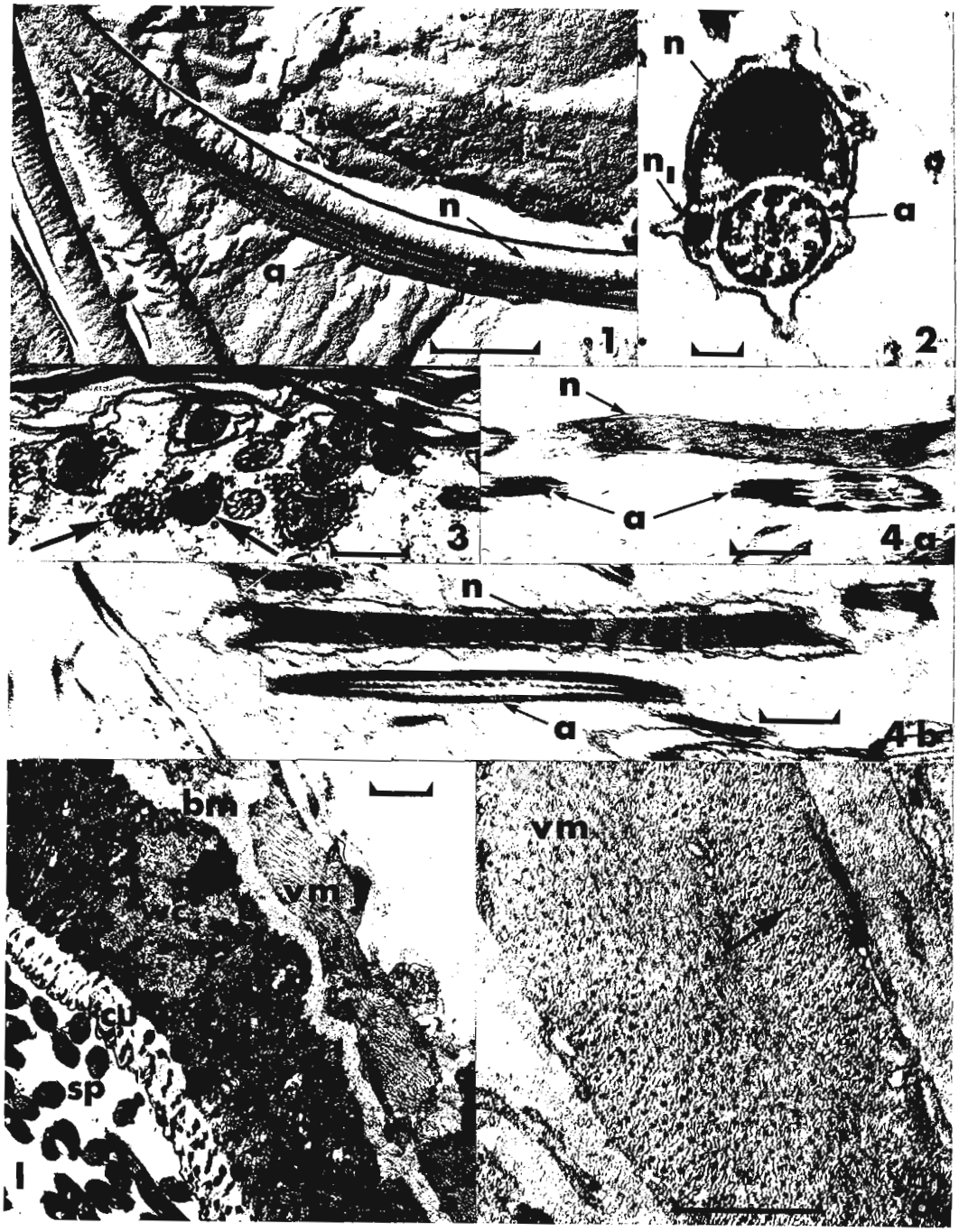
D. melanogaster was used and old females which had been with males were chosen so as to increase the likelihood of sperm being present in the reproductive system.

Observations were made on shadowed carbon replicas of individual sperm tails and on ultrathin sections of ventral receptacle and spermatheca using a Zeiss E.M.9 electron microscope. For carbon replication the ventral receptacle was dissected out on a glass slide in a drop of Drosophila Ringer's solution, with slide and instruments coated with 'Silicone Repelcote' to minimise surface tension problems. The organ was then transferred to a fresh micro-drop of Ringer's solution and gently teased apart to release the contained sperm. Fixation was effected by bringing a drop of 1% osmium tetroxide solution as close as possible to the drop of Ringer's solution containing the sperm and holding it there for one minute. The sperm were then picked up on copper grids coated with a formvar film, and allowed to air-dry. The grids were then washed carefully in distilled water to remove the precipitated salts of the Ringer's solution and any proteinaceous material derived from the rupture of the ventral receptacle. Carbon replicas were then made (Pease 1964) and shadowed with gold/paladium alloy. For preparation of thin sections, the ventral receptacle and spermatheca were fixed in buffered 2.5% glutaraldehyde and post fixed in buffered 1% osmium tetroxide, both adjusted with sucrose to give 0.25 M solution. The tissues were dehydrated in an ethanol series and were embedded in araldite via propylene oxide, with a three day penetration period. Sections were cut on a Porter-Blum hand operated microtome and stained for 45 minutes in a saturated solution of uranyl acetate in 50% ethanol, and for 20 minutes in lead citrate (Reynolds 1963).

Study of the surface view of the sperm tail shows it to consist of two longitudinally orientated portions, one of which shows strands of substructure also orientated longitudinally (fig 1). The sperm tail cut in transverse section also shows two principal elements (fig 2), one of which is more or less homogeneous in appearance while the other shows radially arranged substructure. These two regions are respectively the nebenkern and the axial region (Baccetti and Bairati, 1965). The mitochondria in the spermatid become re-organized into a single body which becomes the nebenkern (Yasuzumi, Fujimura and Ishida 1958). In some species, e.g. Dacus (Baccetti and Bairati 1965), this forms two nebenkern bodies of equal size, but in D. melanogaster there is one large nebenkern and one small one (fig. 2). The axial filament has a flagellar organization (Baccetti and Bairati 1965). Thus it is clear that the sperm tail consists of two principal elements: the large nebenkern and the axial region. It was also noted that these two may become separated (fig. 3).

It has been suggested (Oster et al 1966) that sperm tails split into two longitudinal strands by acetic acid treatment may be fragmented into the two regions here described. Of this there can be little doubt and it seems likely that in the present case the separation is due to the action of the fixative. Oster et al describe a number of reagents which cause the separation and a number which do not, but the principal criterion appears to be the osmotic potential of the reagent. It has been shown (Ballowitz 1890) that treatment with hypertonic solutions of osmic acid split beetle sperm tails into a number of separate fibers. Oster et al state that one of the two fibers is spiralized and is thicker than the other at the tail end of the sperm. It has been shown (Baccetti and Bairati 1965) that the nebenkern is much reduced in diameter towards the tail of the sperm while the axial region is less so. It would therefore seem likely that the 'spiralized fiber' of Oster et al is in fact the axial region and the 'straight fiber' is the nebenkern. The separation described and figured by Oster et al (see their figure 1B) would occur if the nebenkern decreased in size, particularly in the long axis, due to osmotic stress. If osmotic stress by a hypertonic bathing solution did exist then the nebenkern would be shortened longitudinally and the axial region, with its many internal struts, might resist this. The differential stress set up between the nebenkern and the axial region would cause them to separate at numerous points along their length, as described by Oster et al, and the subsequent shortening of the nebenkern would leave the axial region thrown into lateral folds. This seems a reasonable interpretation of figure 1B of Oster et al and is supported by observations of thin sections of sperm (figs. 4a and 4b) which show lengths of nebenkern cut longitudinally and axial region apparently passing in and out of the plane of the section.

Incidental to the study of sperm tail structure it was noted that the wall of individual coils of the ventral receptacle has an outer muscle coat (fig. 5). This muscle tissue is



Electron micrographs of sperm and the ventral receptacle

Fig. 1. Shadowed replica of part of sperm tail showing region of nebenkern (n) and axial region (a). Scale line = 1.0u.

Fig. 2. T.S. of sperm tail from spermatheca showing the axial region (a), the large nebenkern (n) and the small nebenkern (n_1). Scale line = 0.1u.

Fig. 3. Part of section of spermatheca with sperm tails in lumen cut in T.S. In most cases the two regions of the sperm tail have become separated (⟶). Scale line 0.5u.

Figs. 4a and 4b. L.S. of sperm tail showing axial region and nebenkern. The axial region passes in and out of the plane of the section. Scale line = 0.5u.

Fig. 5. T.S. of ventral receptacle showing sperm (sp) in lumen (l) of receptacle, cuticular lining (cu), cells of receptacle wall (wc), basement membrane (bm) and external to this a layer of visceral muscle (vm). Scale line = 1.0u.

Fig. 6. Part of the ventral receptacle wall showing visceral muscle (vm) cut mainly in T.S. Each thick filament is surrounded by 12 thin filaments (⟶). Scale line = 0.5u.

seen to show (fig. 6) the thick and thin filaments of myosin and actin, respectively, typical of muscle. Close examination of the muscle cut in transverse section shows that each thick filament is surrounded by twelve thin filaments as shown in insect visceral muscle (Smith, Gupta and Smith 1966), as opposed to six thin filaments found in insect flight muscle and other 'skeletal' muscle (Smith 1961). It has been stated (Demerec 1950) that the coiled ventral receptacle lacks muscle fibers, but this investigation has shown that throughout its length there is a narrow but well developed layer of visceral muscle surrounding the tube. This may assist in the emission of stored sperm, and may allow temporal control of this process.

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Kuhn, D.T. Arizona State University, Tempe, Arizona. Another case of mass mutation.

A case of mass mutation was encountered in D. melanogaster at Arizona State University between September 1966 and October 1967. The mutations appeared in an Urbana laboratory strain heterozygous for In(3L)P, st. Many germ line and somatic mutations were observed during this one year period. Whole body mutations such as ebony, Minute, yellow^2 and white were encountered more than once. White eyed males were observed on four different occasions.

The mass mutation phenomenon disappeared just as rapidly as it had appeared. Spencer (1935) noted a similar disappearance of visible mutations during his eight year study in D. funebris and D. hydei. He found two mutating periods that were separated by a three year interval during which time not a single visible mutation was observed.

Three months prior to the disappearance of the mass mutation phenomenon, an attempt was made to gather quantitative data on the frequency of spontaneous sex-linked lethals produced in the strain showing the mass mutation. Samples were taken in July, August and September of 1967. A frequency of 0.51 percent (393 X-chromosomes tested) lethals was observed in July. During August the frequency was 0.48 percent (1032 X-chromosomes tested), while in September it dropped to 0.21 percent (935 X-chromosomes tested). The sudden decrease in frequency of sex-linked lethals from August to September paralleled the disappearance of all visible mutations. From September 1967 to the present no more visible mutations have been observed in this strain.

Even though the sample of X-chromosomes tested was small, it is very possible that the simultaneous disappearance of visible mutations and decrease in the frequency of sex-linked lethals were not coincidental. An inactivation or alteration of a gene by a virus-like particle (Mampell, 1946) could result in either a visible mutation (germ line or somatic) or a mutation that would be lethal to the organism. Therefore, it is suggested that this strain became infected with a virus-like particle that was responsible for high frequencies of visible and sex-linked lethal mutations. In September the postulated virus-like particle abandoned the strain and the mutation rates reverted to frequencies characteristic for the strain.

This investigation was supported by Public Health Service Research Grant GM 1235 and Public Health Service Training Grant GM 01433 to Arizona State University from the National Institute of General Medical Sciences.

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Cook, R.M. Sheffield University, England.

Control of fecundity in D. melanogaster.

Data pertaining to the control of fecundity in a 'Pacific' wild-type strain of D. melanogaster have been collected. Daily egg production for individual females was measured after the following treatments: 1. mated with fertile males on day 3; 2. mated with sterile males on day 3; 3. mated with sterile males on days 3, 5 and 8; 4. maintained as virgins. Males used in the sterile matings were obtained from a stock, kindly supplied by Dr. A. Manning, which when outcrossed yields males lacking a 'Y' chromosome, with consequent sterility.

Young virgins lay almost no eggs, whilst mated individuals of the same age may have high fecundity. The differences in egg output brought about by the treatments may therefore be described in two ways:

a. Comparisons of numbers of individuals laying within each group, i.e. dichotomising layers v.s. non-layers. Fig. 1 shows this treatment, the percentage which layed in each group being plotted for each day. Fisher exact probability tests indicate that the increase in the proportion of individuals laying which follows sterile matings is significant, in comparison to the virgins, until day 8.

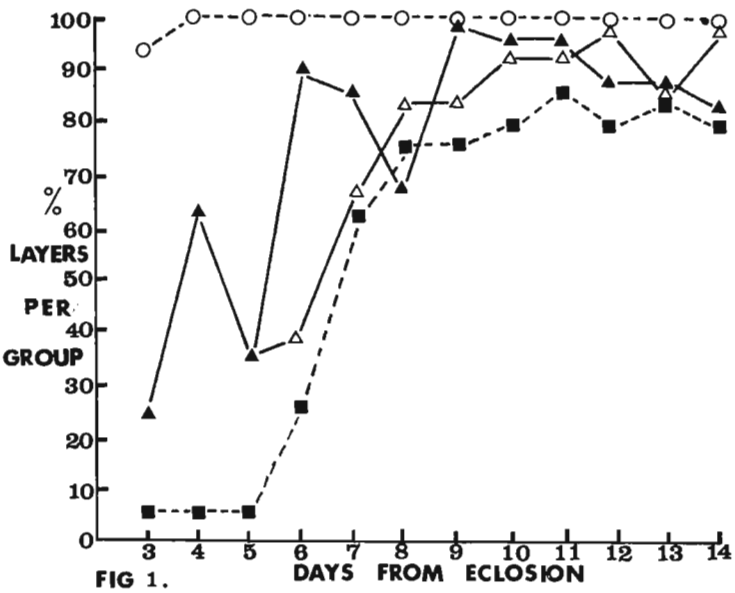

FIG 1.

b. Comparisons on the basis of absolute egg production, after excluding the individuals which do not lay. Fig. 2 shows mean number of eggs produced per group per day, with non-laying individuals excluded. Sterile mating results in a marked increase in egg laying measured 24 hours after the mating. By 72 hours post-mating, however, the level has dropped precisely to that of the virgin females.

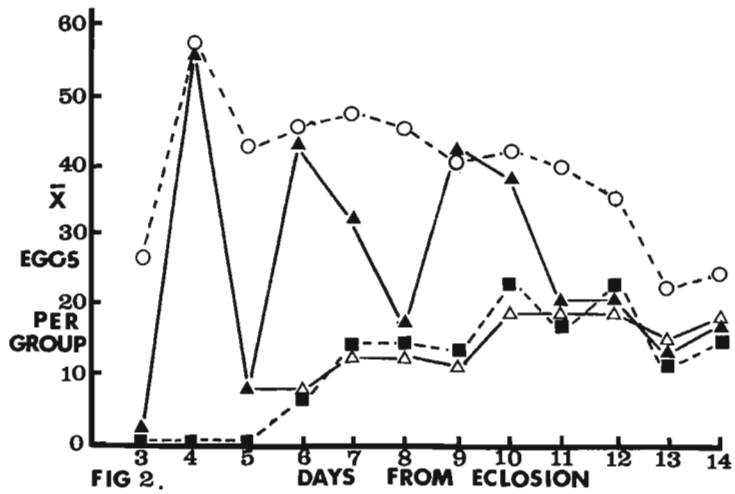

FIG 2.

○---○ females fertilised day 3. N=16.
▲——▲ females sterile mated days 3,5&8. N=22.
△——△ females sterile mated day 3. N=18.
■- - -■ virgin females. N=19.

Conclusions: 1. Fertile mating results in a massive increase in egg production, as many other workers have found.

2. Sterile mating results in variable 'activation' of increased egg production, many individuals being apparently unaffected.

3. Individuals which are activated by sterile mating lay at a level comparable to that of fertilised females, but the increase is extremely transient, returning rapidly to the virgin level.

4. Initially egg production by virgins is at an extremely low level but from day 5 onwards there is a drift upwards. This appears to represent a real, shifting baseline to which previously activated females return.

5. These results are in some disagreement with David (1963) who finds only a slight increase in egg output following sterile mating. David did not measure the egg production of individual animals and in consequence he was unable to determine the proportion of flies activated by this treatment.

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Guzmán, J., R. Félix and J. Ramírez, Comisión Nacional de Energía Nuclear, México City, Mexico. Effects of pre-treatment with Serotonin-Creatinine Sulfate Complex on the radiation-induced frequencies of X chromosome loss, recessive lethals and II-III translocations in D. melanogaster males.

The radiation protective effect of Serotonin (5-hydroxytriptamine) in animals was pointed out by Gray et al. (1952), Bacq and Herve (1952) and Langendorff and Kock (1957). Langendorff et al. (1958) tested the radioprotective effect of 5-HT given before radiation on the mortality rate of white mice within 30 days after the treatment. The results of the experiment showed that 5-HT is a very effective radiation-protective substance if given before irradiation. The decrease of oxygen tension inside the tissues as a result of the vasoconstrictive properties of Serotonin-Creatinine Sulfate Complex (S-CS) in mammals was the indirect mechanism proposed by Gray et al. (1952), Rothe et al. (1963), van den Brenk and Jamieson (1962), and van den Brenk and Moore (1959).

Laguarda-Figueras and Villalobos-Pietrini (1967) presented data demonstrating that S-CS protects planaria (Dugesia tigrina) against the lethal effects of X-rays. In planaria the radioprotection can not be explained by the lowering of the oxygen tension by vasoconstriction as such genera has no circulatory system. Villalobos-Pietrini and Laguarda-Figueras (1967) reported the radioprotective action of S-CS in Vicia faba seedlings pre-treated with S-CS, measuring the survival rate of roots after irradiation.

Alexander et al. (1955), Langendorff and Melching (1959), Dukor (1962), and Lohmann et al. (1966) proposed several hypothesis to account for the radioprotection afforded by S-CS.

In this experiment the effect of S-CS on chromosome X or Y loss, sex-linked recessive lethals, and II-III translocations was measured in the progeny of pre-treated and irradiated "Oster males" by means of the genetic scheme designed by Oster (1958). Male flies from a stock containing a marked sc^8 Y chromosome and the closed X, X^{c2} marked with the mutants yellow (y) and Bar (B) in the males, and yellow, forked (f), attached X chromosomes in the female (yf:=) were aged for 72 hours before the treatment with S-CS, irradiation, or both, and mated to virgin "Oster females" with markers in the I, II and III chromosomes (y sc^{S1} In-49 sc^8;bw;st pP). The use of the markers B and y, to identify the treated sex chromosomes of the males, makes the detection of sex-linked recessive lethals fairly easy. F_1 males and females are allowed to mate with each other for at least two days before being separated into vials. The F_1 fertilized females are tested for lethals by examining the F_2 offspring for the absence of Bar males which would indicate that a lethal has been induced in the paternal X chromosome. The frequency of exceptional (X/O) males among the F_1 flies is determined by counting the yellow males, which represent cases of loss of the whole or part of the X or Y chromosomes. Normal males have non-yellow bodies, since they carry the normal dominant allelomorph of yellow in the sc^8 insertion of their Y chromosome. The scoring of translocations is simplified by using the markers brown (bw) located on chromosome II, scarlet (st), and the peach allelomorph of pink (pP), which are both located on chromosome III. The eyes of the flies heterozygous for these markers appear brick-red in color while the eyes of those homozygous for brown and scarlet are white. The translocation tests can be carried out by mating one F_1 non-yellow male with one virgin female similar to his mother per vial. Such females are obtained from the "Sterilizer" stock (Y^{Lc}/XY^S;bw;st pP). The cross of males from this stock with "Oster females" produces automatically virgin F_1 females to be mated with the F_1 non-yellow males.

Several concentrations of the S-CS complex (Hycel, Houston, Texas) dissolved in 0.7N NaCl solution were administered by injection in the gonadal area of aged "Oster males" in order to determine the concentration to be used without interfering with their viability or fertility. Since Carlson and Oster (1962) have shown that the amount of liquid expelled after injection varies from fly to fly, estimates of the amount of solution injected into each fly were not attempted. A 100% concentration of S-CS killed all the injected males. The death of 25% of the injected males within five days of the treatment with the S-CS solution (50%), indicates that S-CS is being absorbed by the cells and interfering with cellular physiology to such an extent that death follows. A concentration of 25% was used; at this level no mortality was recorded among the injected adults within fifteen days. A physiological 0.7N NaCl solution was injected into male controls, instead of using distilled water which obviates the problem of induced sterility and possible cell selection by osmotic shock.

The source of radiation was an X-ray Stabilipan Siemens instrument operating at 220 kV and 15 mA, with an exposure rate of 85 R/min. The distance from the window was 30 cm. and a ThII filter was used.

"Oster males" collected within 72 hours of eclosion were injected with the S-CS solution or with the saline solution. Within an hour of injection half of the males were treated with 2,500 R, and mated to one "Oster female" per vial. Males and females were allowed to mate for two days before being separated. After 17 days the emerged F_1 flies from each vial were counted separately in order to count the X/O males per vial (Table I). No premeiotic events were included, as all the exceptional males were found in different vials. To detect sex-linked lethals, F_1 females and males were shaken over into fresh vials and the F_2 males from each vial were examined (Table II). The F_1 males were tested for translocations between autosomes II and III by being backcrossed to virgin females obtained from the mating of "Oster females" with "Sterilizer males" (Table III).

The chi-square data below each table show that no significant differences are found when the groups treated with S-CS are compared with the non-treated groups.

TABLE I. FREQUENCIES OF X/O MALES FOUND AMONG F_1 PROGENIES

Group	X/O Males	F_1 Females	F_1 Males	Total	%
A) Control	16	5039	4505	9560	0.3552
B) NaCl solution	5	2769	2265	5039	0.2207
C) S-CS solution	4	1708	1438	3150	0.2782
D) Control + 2,500 R	30	4008	3029	7067	0.9904
E) NaCl sol. + 2,500 R	25	3457	2673	6155	0.9353
F) S-CS sol. + 2,500 R	17	2170	1789	3976	0.9503

Chi-square values from the comparison of groups

A-B	A-C	B-C	D-E	D-F	E-F	A-D	B-E	C-F
1.066	0.246	0.135	0.026	0.001	0.027	9.738	9.767	5.404

TABLE II. SEX-LINKED RECESSIVE LETHALS FOUND AMONG F_2 PROGENIES

Group	Sex-linked lethals	Number of chromosomes tested	%
A) Control	4	1045	0.3828
B) NaCl solution	4	1064	0.3759
C) S-CS solution	4	1060	0.3774
D) Control + 2,500 R	43	942	4.5648
E) NaCl sol. + 2,500 R	35	1082	3.2348
F) S-CS sol. + 2,500 R	40	947	4.2239

Chi-square values from the comparison of groups

A-B	A-C	B-C	D-E	D-F	E-F	A-D	B-E	C-F
0.001	0.001	0.001	2.404	0.131	1.388	35.517	24.572	34.511

TABLE III. TRANSLOCATION FREQUENCIES AMONG F_2 PROGENIES

Group	Translocations	Number of chromosomes tested	%
A) Control	0	1555	0
B) NaCl solution	5	1030	0.4854
C) S-CS solution	2	818	0.2445
D) Control + 2,500 R	44	965	4.5596
E) NaCl sol. + 2,500 R	36	1024	3.5156
F) S-CS sol. + 2,500 R	36	793	4.5397

Chi-square values from the comparison of groups

A-B	A-C	B-C	D-E	D-F	E-F	A-D	B-E	C-F
7.563	3.810	0.701	1.403	0.001	1.232	72.161	24.102	32.253

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Gerdes, R.A. Texas Woman's University, Denton, Texas. Sex-linked recessive lethal test with hydrogen fluoride treated D. melanogaster.

There are many reports in the open literature relating to the mutagenicity of radiation and radiomimetic chemical mutagens. Recognizing this, we are evaluating potential air contaminents for mutagenic effects.

The experimental procedure was to place unetherized samples of Oregon-R into fumigation chambers, at various concentrations of HF contamination, for a 24 hour period. Then the males were mated to "Basc" females in a sequence of 3-3day broods. The standard sex-linked recessive lethal test was made on the F_1 females. The results of these tests are in the following table.

Treatment Level	Brood A	Brood B	Brood C	Pooled
0 HF Control	1/1809 = .00055	1/1720 = .00058	0/1702 =	2/5231 = .00032
1.3 ppm HF	0/1889 =	1/1871 = .00053	2/1873 = .0011	3/5633 = .00053
2.9 ppm HF	4/1719 = .0023	3/1742 = .0017	4/1720 = .0023	11/5181 = .00212
4.3 ppm HF	3/1907 = .0016	9/1832 = .0049	4/1775 1/ .0023	16/5514 = .0029

lethal/# chromosomes tested

Additional data is being collected to determine if brood differences are present, or if, as this data indicates, there are no brood differences.

Savontaus, M.-L. University of Turku, Finland. Tetrad analysis of control and X-ray irradiated females of D. melanogaster.

Females of the genotype y sc cv ct f car/y were collected within 6 hr of eclosion. They were divided into two groups one of which was irradiated with 3000 r at the rate of about 632 r/min at a focal distance of 10 cm and the other served as an untreated control. Immediately after irradiation both the treated and control females were mated individually with 2-3 males of the genotype sc cv ct f car/Y and transferred daily with their mates to fresh bottles for 9 days. Totally, 11387 flies were counted in the irradiated group and 3586 in the control. Crossing-over was scored in the regions: sc-cv, cv-ct, ct-f and f-car. Tetrad analysis of the cross-over data was as follows:

	control	irradiated
non exchange tetrads	4.8%	30.3%
single exchange tetrads	76.4%	56.7%
double exchange tetrads	17.5%	12.2%
triple exchange tetrads	1.3%	0.8%

Compared with the control results, the frequency of non-exchange tetrads in the treated group was greatly increased. This suggests that the net reduction in crossing-over observed by Chandley (1968, Mutation Res. 5) after X-irradiation and heat-treatment is probably due to an increased desynapsis or asynapsis of the treated chromosome.

Strangio, V. A. University of Melbourne, Australia. Sub-metacentric recombinant chromosomes recovered from irradiated males bearing a ring-X and a doubly-marked Y.

In one experiment, male prepupae (6±3 hours after puparium formation) carrying X^{c2}, v f and $B^{S}Y\ y^{+}$ chromosomes received 800r X-ray treatment. After emerging, these males were mated every 24 hours to four virgin "yellow, apricot" females over the next 4 days. Male gametes utilised during this period were irradiated as spermatocytes and late spermatogonia (see DIS 41: 170).

An exceptional "Bar" daughter appears if the contributing male gamete carries: (a) intact, separate X and Y through non-disjunction or (b) an intact X and a separate B^{S}-carrying Y-fragment translocated to an autosome or (c) a single non-ring chromosome with paternal X and Y components.

Three different examples of this last type of aberration were recovered. Examination of giant neuroblast cells shows the three chromosomes to be sub-metacentric. Detailed study of one (17C) suggests that the shorter arm has the heterochromatic block structure of Y^{L} (Cooper; Chromosoma, 10: 535, 1959). Unlike the other two recombinants, 17C exhibits regular recombination when made heterozygous to a normal test X. Recombination data involving B^{S} also support the idea that this marked part of Y^{L} is located in or close to the short arm. More rigorous genetic tests are in progress. The longer arm of 17C (presumably carrying most or all of the X euchromatin in normal sequence) also appears to be heterochromatic at its distal end.

When spermatocytes are irradiated, induced exchange between rod-X and Y heterochromatic regions results in readily recoverable recombinants (DIS 41: 176). Where the X is in ring form, the absolute rate of X-Y exchange is probably unchanged but the frequency of recovered recombinants is very much reduced (same report). Exchange causes the formation of a dicentric chromatid in which the X material is sandwiched between two telomeric Y-fragments. If the inter-centromeric bridge is broken in a euchromatic region at anaphase I, subsequent breakage-fusion-bridge cycles undoubtedly reduced the chance that a recombinant chromosome will be recovered. But a break in heterochromatin may be capable of healing to yield a "non-telomeric" stabilized chromosome end. Khush and Rick (Chromosoma, 23: 452, 1968) have shown that breaks in the heterochromatin of tomato chromosomes do heal in this fashion.

In a second experiment with X^{c2}, y B and B^{S} Y y^{+}, monocentric recombinants carrying either one of the two Y markers were generated. These are also being analyzed.

del Solar, E. Universidad de Chile, Santiago, Chile. Choice of oviposition sites by D. pseudoobscura females among areas with different numbers of eggs.

Groups of 15 D. pseudoobscura females were placed in population cages containing 15 food cups, each with 14 ml of Ohba's culture medium. The cups were removed every 24 hours, the eggs in each were recorded, and they were then replaced in the same position. The experiment was replicated 12 times. If only the cups containing one or more eggs are considered, the average number of eggs laid after 24 hours can be analyzed. Table 1 shows that the females do not discriminate among sites which contain different numbers of eggs.

Number of eggs	$\bar{X}$ ± S.E.	N
1 - 10	37.8± 5.7	34
11 - 20	38.6± 8.0	23
21 - 30	37.6± 7.0	27
31 - 40	34.5± 6.6	21
41 - 50	42.5± 9.8	15
51 - 60	46.4± 9.7	12
61 - 80	29.6± 9.6	15
81 - 100	27.7±10.0	11
101 - 150	40.2± 9.0	13
151 - 200	22.3±12.9	7

A comparison was then made between the oviposition cups containing previously laid eggs and clean ones. The results show that the females tend to lay more eggs in clean cups than in those previously occupied. 76 previously occupied cups containing 2905 eggs and 52 clean ones with 2271 eggs were recorded between 24 and 48 hours. From 48 to 72 hours 101 occupied cups with 3635 eggs and 22 clean ones with 980 eggs, were found.

The mean number of eggs was 26.3 for the occupied category, and 44.0 for the clean one, with a chi-square of 4.438. (P = 0.02-0.05).

Esposito, V.M. and V. Ulrich. West Virginia University, Morgantown, West Virginia. Characterization of D. melanogaster acid phosphatase.

Non-specific acid phosphatases have been found in many tissues and in many organisms (Schmidt, 1961). Although evidence for their existence has been accumulating since 1907, real interest did not develop in them until the discovery of bone enzyme by Robinson in 1923. Walker, Lemon, Davison and Schwartz (1954) concluded on the basis of an extensive literature survey, that acid phosphatase consisted of an inclusive group of enzymes with different substrate specificities. Electrophoretic variants or isoenzymes of acid phosphatase, their hybrid nature and their genetics have been presented by MacIntyre (1966).

Adult Drosophila
↓
Macerate in 0.45 sucrose
Centrifuge at 650 X g for 5 min.
↓
Pellet — Supernatant
Treat with 40% $(NH_4)_2SO_4$ saturation
↓
Stir at 4°C for 1 hour
Centrifuge at 12,000 X g for 20 min.
↓
Pellet — Supernatant
Add $(NH_4)_2SO_4$ to 70% saturation
↓
Stir at 4°C for 1 hour
Centrifuge at 12,000 X g for 20 min.
↓
Pellet — Supernatant (discard)
↓
Wash with 70% $(NH_4)_2SO_4$ - Centrifuge at 12,000 X g
Repeat 3 times for 20 min.
↓
Pellet
↓
Put into solution with 0.45 M sucrose
Dialyze at 4°C for 8 hours
↓
Protein Solution
↓
Acid Phosphatase preparation
Disc electrophoresis
Section containing acid phosphatase activity cut out and eluted with 0.45 M sucrose
Centrifuged at 12.000 X g for 20 min.
↓
Electrophoretically purified acid phosphatase

Fig. 1 Isolation and purification of acid phosphatase.

Cultures of D. melanogaster were reared and maintained on a standard cornmeal-agar medium that had been seeded with live yeast. Crude enzyme preparations were obtained in two ways. Individual larva were macerated in 0.05 ml of 0.45 M sucrose buffer with a micro-mortar and pestle and centrifuged at 650 X g for 2 minutes at 4°C. These were used for all experimental assays. Larger quantities of material were prepared by macerating 10 grams of adult flies in 20 ml of 0.45 sucrose buffer containing 0.5 grams Norit A. This mixture was then centrifuged at 650 X g for 5 minutes. The resulting sediment was discarded, and the supernatant, after filtering through glass wool, was used either directly as a crude preparation for the assays or further purified by $(NH_4)_2SO_4$ precipitation and electrophoresis (Figure 1). The enzyme was assayed by a modified method originally described by Lawrence, Melnick, and Weiner (1960).

Polyacrylamide gel columns, as described by Ornstein and Davis (1964), were used for electrophoretic purification of enzyme. Glass cylinders 65 mm in length and 7 mm inner diameter were used. To avoid heat inactivation of the enzyme electrophoresis was performed in a cold room (4°+1°C) with 3 milliampers per column. The running time was 90 minutes.

The principal criteria employed in separating and classifying phosphatases is their specificity to substrates, pH optimum and activity response to divalent cations (Schmidt, 1961). The pH optimum was determined on sodium alpha-napthyl acid phosphate. The buffers used to cover the pH range from 3.8 to 10.0 were acetate (pH 3.8-5.8), phosphate (pH 5.6-8.0)

and tris (pH 7.0-10.0). A pH of 5.0 was found to be optimum for this system.

Since various phosphatases require metal ions for activity, assays were conducted to determine the necessity of these as cofactors. Mg^{++} (magnesium chloride), Mn^{++} (manganous chloride) and Zn^{++} (zinc chloride) were introduced separately and in various combinations to the reaction mixture in concentrations of 1 to 10 mM. The divalent cations neither activated nor inhibited this system when either crude or purified enzyme preparations were used.

The substrate specificity of both crude and electrophoretically purified enzyme preparations was tested by use of the coupling technique (Burstone, 1958) against eight substrates, i.e., sodium alpha-naphthyl acid phosphate, naphthol As, naphthol AS-An, naphthol AS-E, naphthol AS-GR, napthol AS-Mx, naphthol AS-TR and naphthol AS-Bl. No substrate preference could be found. The enzyme system also reacted in the same way in every test when Mg^{++}, Mn^{++}, Zn^{++}, sodium fluoride and tartaric acid were added either individually or in combination. Demonstration of enzyme activity on all eight substrates indicates that this system is non-specific. A comparable acid phosphatase system, which also displayed non-specific activity, has been reported in the slime mold Dictyostelium discoideum (Gezelius, 1966). Therefore, on the basis of a pH optimum of 5, a general substrate specificity, and a lack of inhibition and activation by divalent cations, this enzyme system is classified as a phosphomonoesterase.

Inhibitors are the most characteristic modifying factors of phosphatases and sodium fluoride and tartaric acid are most commonly used to distinguish various phosphomonoesterases. These were used in concentrations of 10 mM with crude and with $(NH_4)_2SO_4$ and electrophoretically purified enzyme preparations. The inhibitors were added directly to the reaction mixture in one series of experiments, but in another series the enzyme preparations were incubated with the inhibitors prior to the addition of the reaction mixture. Sodium fluoride and tartaric acid caused complete inhibition in every experiment. The addition of the divalent cations Mg^{++}, Mn^{++} and Zn^{++} did not alter this inhibition. Therefore, on the basis of a pH optimum of 5, general substrate specificity and complete inhibition by sodium fluoride and tartaric acid this enzyme system is classified as a phosphomonoesterase II (E.C. 3.1.3.2).

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Minamori, S. and K. Ito, Hiroshima University, Japan. Mutagenic action of extrachromosomal element delta in D. melanogaster.

It was found that an extrachromosomal element denoted by delta in D. melanogaster may induce frequent lethal and semi-lethal mutations on the second chromosome. The average mutation rate induced on the chromosome carrying Dmb gene (allows the multiplication of delta) was about 9 percent when combining lethals and semi-lethals, and ignoring the clustering of lethals. While the rate in the chromosome without the Dmb gene was clearly not so high, even in the presence of delta.

Several instances of mutation cluster were observed. In four clusters, all lethals recovered from a single parent were allelic with each other. These findings lead to the conclusion that the mutation induced by delta may occur at the pre-meiotic cell stage, possibly in an early embryonal stage of the carrier.

A total of 113 lethal genes originating independently were found to locate at 27 different sites on the chromosome. The locations of these sites were determined and it was found that the distribution pattern of lethals along the chromosome was unique in contrast to the pattern reported by Ytterborn (1968) in the lethals induced by X-ray. The lethals were strongly concentrated at 0-10 (13.3%), 55-65 (30.0%) and 70-85 (50.4%) of the genetic map-unit, and none were located in the left part of centromere.

Reference: Ytterborn, K.H., 1968, Hereditas 59: 49-62.

Pachciarz, J.A.* and W.M. Luce. University of Illinois, Urbana, Illinois.

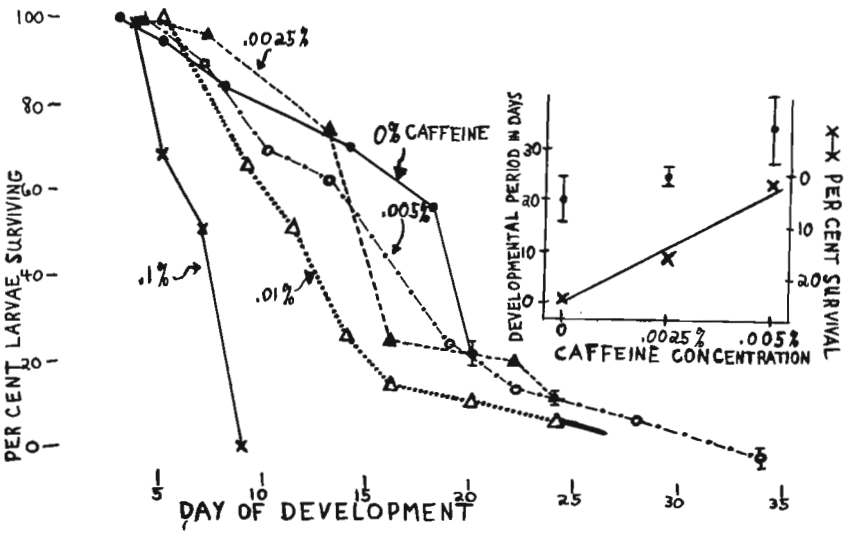
The effect of caffeine on axenically grown D. melanogaster.

During an investigation of caffeine mutagenesis in D. melanogaster, two effects of caffeine were noted: 1) a marked increase in the length of the developmental period; 2) a corresponding decrease in the percent of larvae surviving to adulthood.

Previous studies on caffeine mutagenesis (Andrews, 1959; Yanders and Seaton, 1962) have utilized standard media. This study used nucleic acid free, chemcially defined medium and the strain Livingston red, kindly provided by R. Rayle, which was axenically grown. The effect of several concentrations of caffeine in this medium was tested by the Muller-5 technique for sex-linked recessive lethals.

Livingston red eggs less than twenty-four hours old were collected, sterilized by a modification of Geer's method (Geer, 1963), and aseptically transferred to sterilized vials containing 5 ml of Geer's medium, omitting RNA and substituting sucrose for fructose. Each vial contained an average of four ± .5 larvae. Caffeine concentrations in the medium ranged from .0025% to .1%. Larvae were scored every three days for viability, and male adult survivors were mated to Muller-5 virgins. F_1 females were individually mated to test for sex-linked recessive lethals.

% Caffeine	% Larvae Reaching Adulthood	% Pupae From Larvae	No. of Broods	Length of Developmental Period	Matings LR♂ x M5♀	No. of chromosomes Tested	No. of lethals	No. Non-lethals	Mutation Rate
0	23	44	4	20d ± 4	10	551	3	390	.77%
.0025	14.4	18.4	2	24d ± 2	9	551	2	419	.48%
.005	2	5	3	34d ± 7	0	-	-	-	-
.01	-	-	3	-	1	18	0-	14	0%
.1	0	0	1	death at 7-9 d	0	-	-	-	-



Vials found to be contaminated are not included in the data. Periodic checks were made for the presence of aerobic bacteria and molds by plating on bacteriological media, but the presence of anaerobic bacteria, Mycoplasma, or viruses could not be excluded.

Forty-seven male flies were obtained and twenty successful matings were made, with 1120 chromosomes being tested. Results are summarized in the table.

Of the three lethal control mutations, two were cluster events with the flies phenotypically all Muller-5, one a block of two vials and the other a block of twenty-eight vials. The third control mutation and the two lethals with .0025% caffeine were single events. These lethal mutations were verified by F_2 crosses.

The graph shows the effect of caffeine concentration on the length of the developmental period and on the percent of larvae surviving to adulthood.

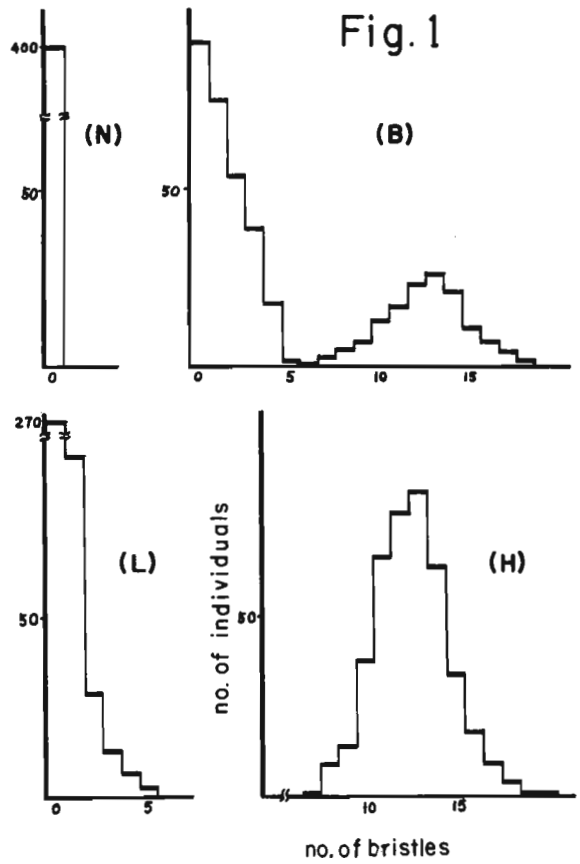
While no statistically valid conclusion can be drawn from the sex-linked recessive lethal mutation rates, caffeine is shown to prolong the developmental period with a corresponding decrease in survival of larvae, and this effect seems to be proportional within the limits of the caffeine concentrations used. That these low concentrations of caffeine demonstrate such an effect may be potentiated by the axenic nature of the experiment.

The assistance of W.R. Greenfield and E.B. Sheinin is gratefully acknowledged.

References: 1. Andrews, L.E. 1959, Am. Naturalist 93: 135. 2. Yanders, A.F. and Seaton, R.K. 1962, Am. Naturalist 96: 277. 3. Geer, B.W. 1963, J. Exp. Zool. 154: 353.

* Now in the Department of Microbiology, St. Louis University, St. Louis, Missouri.

Hihara, F. Tokyo Metropolitan University, Tokyo, Japan. On the first sternite bristles of D. busckii.



The first sternite bristle number of wild strains of D. busckii varies from zero to twenty. However, precise observation made it possible to divide the species into three groups concerning the bristle number. They are as follows: 1. N-type. All of the flies in a population have no bristles. 2. L-type. Flies having 0-1 bristle are most frequent, while those having 2-5 bristles are much less frequent in a population. 3. B-type. Bristle counts in a population consist of two modes, one of which is L-like, ranging from 0-5, the other of which is H-like, normal but rather flat, ranging from 6-20 (Fig. 1, N,L,B). H-type. Experimental population representing only high mean number of the bristles is isolated from the B-type wild strain (Fig. 1, H).

In natural populations, B-type seemed to be most popular. L-type seemed to be less frequent than the former. N-type was found in only one locality consisting of small population at Kiso Valley. No natural population has been known to come under H-type.

Relative frequency of the L-like and the H-like flies within a population designated B-type varies as geographical strain. But no population in which H-like flies exceeded L-like flies has been found in nature.

Wheeler (1960) has proposed that first sternite in the Drosophilidae has eventually been modified in shape during its evolutionary course. Further investigation on the present species may be available to confirm this proposition.

Enns, R.E. University of Oregon, Eugene, Oregon. Segregation in males with XY-X chromosomes with and without free Y's and the segregation distorter chromosome, SD-72.

Using a series of X·Y chromosomes in conjunction with SD-72 (recovery normally on the order of 100%) in males having an otherwise completely sensitive background to SD, it was shown that, while males with only X·Y chromosomes showed a marked decrease in SD recovery (from an expected k value of .99 to approximately .85), similar males containing a free Y in addition to the X·Y arrangement demonstrated k values on the order expected from SD-72 males (i.e., .99+).

When various Y's lacking fertility factors ("Y-testers", developed by Brosseau, 1960) in addition to a series of X duplications (for varying lengths of heterochromatin) and Y fragments ($Y^S \cdot Y^S$ and Y^L) were substituted for the free Y, all of the Y-testers with the exception of KL-2 gave high k values; high k values were also realized for the Y^L fragment but not for any of the X-duplications nor for the $Y^S \cdot Y^S$ fragment.

Chromosome or fragment	K value
KS-1	.99-1.00
KS-2	.97-1.00
KL-1	.98-.99
KL-2	.87-.91
KL-3	1.00
KL-3,4	.99-1.00
KL-4,5	1.00
$Y^S \cdot Y^S$	.81-1.00
Y^L	.97-.98
Dp(1;f) 112	.89-.95
Dp(1;f) 1488	.88-.95

It is interesting to note that the implied vital region of the extra Y, around KL-2, was found by Brosseau (1964) to suppress variegation in $X/B^{SV}Y$ males.

References: Brosseau, G.E., Jr., 1960 Genetics 45: 257-274. Brosseau, G.E., Jr., 1964. Genetics 50: 237.

Hunt, D.M., University College London, England. A comparison of the effect of acid amides supplemented to yeasted and sterile synthetic culture on the expression of the Bar phenotype.

It has been previously shown (Kaji, 1960) that the acid amides are effective in increasing the facet number of B eye. However, such experiments have not been carried out under completely defined conditions and it is possible that high concentrations of acid amides primarily alter the metabolism of the yeast populations and it is this change that in turn influences the expression of the B phenotype. Certainly, the expression of ey (Sang and Burnet, 1963) and ant (Gordon and Sang, 1941) are extremely sensitive to variations in culture conditions. To clarify this point, the effect of acetamide and lactamide supplemented to normal yeasted culture and to a sterile synthetic medium was examined. In the latter case, germ-free larvae were used to completely eliminate the microflora normally present in Drosophila cultures. Mean eye size is taken as a measure of gene expression. No significant changes in body size were recorded throughout this series of experiments.

In Fig. 1, the results of increasing concentrations of acetamide supplemented to yeasted culture and axenic synthetic culture are presented. In both cases, a marked increase in mean eye size is obtained, although equivalent concentrations are more effective in synthetic media than in yeasted culture. A concentration in excess of 0.5% proved toxic in synthetic culture. Increasing concentrations of lactamide also produce an increase in eye size (Fig. 2) in both types of culture media and, in this case, equivalent concentrations are equally effective. A concentration of lactamide in excess of 1.0% is toxic in synthetic culture.

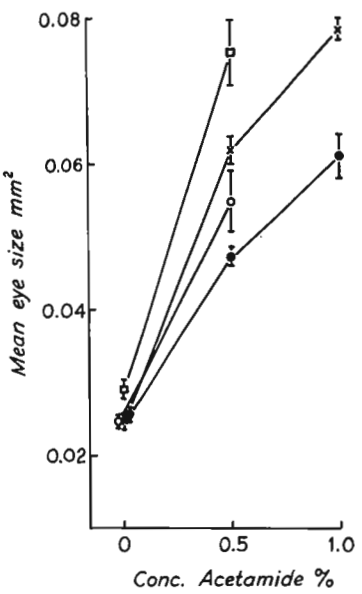


Fig. 1

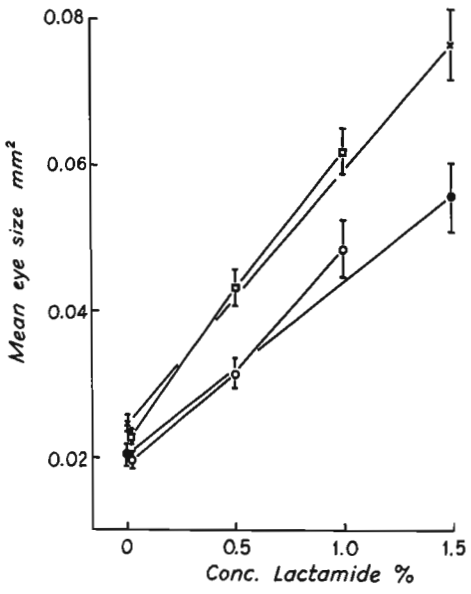


Fig. 2

Relationship between acetamide (Fig. 1) and lactamide (Fig. 2) concentration and mean eye size. x males, • females in live yeast medium, □ males, o females in sterile synthetic medium.

From these results, it is possible to conclude that high concentrations of acid amides directly affect the expression of the B phenotype, presumably by interacting with a biosynthetic process important for eye development. However, the presence of yeast populations in the supplemented cultures does considerably reduce the toxicity of these compounds.

References: Gordon, C. and Sang, J.H. 1941, Proc. Roy. Soc. B130: 151. Kaji, S. 1960, Mem. Konan Univ. 4: 1. Sang, J.H. and Burnet, B. 1963, Genetics 48: 1683.

Grossfield, J. Purdue University, Lafayette, Indiana. Crossing over between white alleles of D. auraria.

EMS treatment of D. auraria Type A (a member of the melanogaster species group) has produced two eye color mutants w and w^{saf} (see report on new mutants for description) which appear to be homologous with the white locus in D. melanogaster. The F_1 heterozygous female is phenotypically w^{saf} and progeny of this type of female and w males revealed 2 wild type males among 3802 F_2 individuals scored. The indicated recombination distance of .01 is tentative since no outside markers were available to verify the crossover origin of the wild-type males.

Burdette, W.J. and J.E. Carver. The University of Texas, M.D. Anderson Hospital and Tumor Institute, Houston, Texas. Tumors in Drosophila following treatment with oncogenic viruses.

The RNA Rous-sarcoma virus (Bryan high-titer strain: BH-RSV) was found to be associated with an increased incidence of melanotic tumors, mutations, and chromosomal aberrations in D. melanogaster (Burdette, W.J., 1969, Tumors, hormones, and viruses in Drosophila, Nat. Cancer Inst. Monogr. 31: 303-321; Burdette, W.J. and Yoon, J.S. 1967, Mutations, chromosomal aberrations, and tumors in insects treated with oncogenic virus, Science 155: 340-341). The results of these and similar studies in which the DNA virus: SV 40 (Simian virus: strain 40) and Rous associated virus (RAV-1) have been administered to two different melanogaster stocks ($sc^8 \cdot Y \cdot B^s/y^2$ w^i ct^6 f: "Multipurpose" and Oregon-R) in 1:1 and 1:50 dilutions are shown in the table below. Tumor frequencies significantly higher than controls were observed in all series except in the Oregon-R stock treated with SV 40 virus. Further, the tumorigenic effects of BH-RSV and SV 40 were found

Tumor incidence following treatment of pre-imaginal stages with oncogenic virus

Stock	Virus administered	Percent with tumors	Total number observed	P
MP	Control	2.3	2230	-
	BH-RSV, 1:1	5.2	852	<0.005
	SV-40, 1:1	8.5	1396	<0.005
	RAV-1, 1:1	4.2	1702	<0.005
	BH-RSV, 1:50	7.9	1846	<0.005
	SV-40, 1:50	7.8	742	<0.005
	RAV-1, 1:50	8.1	1530	<0.005
ORE-R	Control	0.3	1320	-
	BH-RSV, 1:1*	2.6	760	<0.005
	SV-40, 1:1*	0.9	1337	.05-.10
	BH-RSV, 1:50*	3.2	801	<0.005
	SV-40, 1:50*	1.0	314	.25-.5

*Yates' correction applied.

to be greater on the multipurpose than on the Oregon-R stock. The latter result suggests genetic differences in susceptibility to oncogenic viral agents among different strains of Drosophila. Comparison of 1:1 and 1:50 dilution treatments show higher frequencies of tumors at the 50-fold dilution for BH-RSV ($P<0.01$) and RAV-1 ($P<0.001$) in the M-P stock, and for BH-RSV in the Oregon-R stock ($P<0.001$). No significant difference between treatment concentrations was observed for SV 40 in either stock analyzed (MP: $P>0.5$) ORE-R: $P>0.8$, Yates' correction applied). Studies designed to elucidate the mechanisms of viral action in Drosophila are being continued.

Baimai, V. Mahidol University, Bangkok, Thailand. D. montium from Mt. Maquiling, Luzon, Philippines.

Karyotype variation in D. montium has recently been discussed (Baimai, 1969).

In February, 1969, an extensive sample of living Drosophilas was obtained from Mt. Maquiling, Luzon, Philippines (Mather 1970). A culture of D. montium was established from the collection which turned out to have a metaphase plate Type III similar to those from Tawau and Sandakan, Sabah. This strain proved to be cross-fertile with strains from Madang, (New Guinea) Kota Kinabalu (Sabah), and Tawau (Sabah).

Acknowledgement: This work was carried out as part of the Research Project "Evolution in the Genus Drosophila" directed by Dr. Wharton B. Mather, Head of the Genetics Laboratory, Zoology Department, University of Queensland.

References: Baimai, V. 1969. Karyotype variation in D. montium. DIS 44: 115. Mather, W.B. 1970. The Genus Drosophila at Mt. Maquiling, Luzon, Philippines. DIS 45: 111.

Hanks, G.D. Indiana University Northwest, Gary, Indiana. Frequency changes of marked Y chromosomes with RD background.

Marked Y chromosomes were tested in population cages (2Y's at a time with a frequency of 0.5 each). Y^{BS} increased to a frequency of 0.99 when placed in a cage with Y^{y+}. Y^{y+} increased to a frequency of 0.92 when allowed to compete with Y^{bw+}. Y^{BS} increased in frequency to 1.00 when allowed to compete with Y^{bw+}. The percentage of females is 59.3 for Y^{BS} male parents, 63.4 for Y^{y+} male parents, and 59.8 for Y^{bw+} male parents. Apparently the interaction of fitness in the diploids, meiotic drive of the Y chromosome, and selection due to the sex ratio determines which Y chromosome has the strongest competitive advantage. Probably fitness is most important in determining the frequencies of these particular Y chromosomes. The values on percentages of females given above were measured by mating each male singly to 5 females and counting progeny in 3 culture bottles. Several males of each type were tested. The relative abilities of the 3 Y chromosomes to gain in frequency are given in increasing order: $Y^{bw+} < Y^{y+} < Y^{BS}$.

Okada, T. Tokyo Metropolitan University, Tokyo, Japan. A numerical analysis of the drosophilid fauna centering around New Guinea.

In 1968-9, an opportunity was given to me through courtesy of Professor M. R. Wheeler of the University of Texas, Austin, Texas, to examine a large collection of New Guinean Drosophilidae at his Genetics Foundation. The examination resulted in finding more than two hundred species belonging to twenty-five genera, which highly surpassed the previous records, thirteen genera and about forty species. The identification of the species is still incomplete, and the faunal relationships at the genus level between New Guinea and the surrounding geographical areas, in which the drosophilid faunae have sufficiently been known and the endemic genera are relatively few, are analysed using numerical taxonomic methods, taking a geographical area as OTU and the presence and absence of a genus as states of a character, coded 1 and 0, respectively. The faunal comparison was based on several kinds of relatively simple similarity coefficients (S) and the clustering was made by WPGA and UPGA. The resulting phenograms were evaluated by means of the cophenetic correlation coefficients (r) between original and derived similarity matrices.

The highest cophenetic correlation coefficient, eventually the most reliable phenogram,

S	r,WPGA	r,UPGA	n_{jk} in numerator	Faunal inclusion in Europe-North American cluster
S_J*	0.91	0.89	-	-
S_{RR}	0.86	0.94	-	-
S_O	0.82	0.88	±	-
MCD	0.72	0.71	+	-
S_{SM}	0.66	0.66	+	Africa
S_S	0.85	0.85	-	Africa
S_{RT}	0.64	0.64	+	Africa, Japan

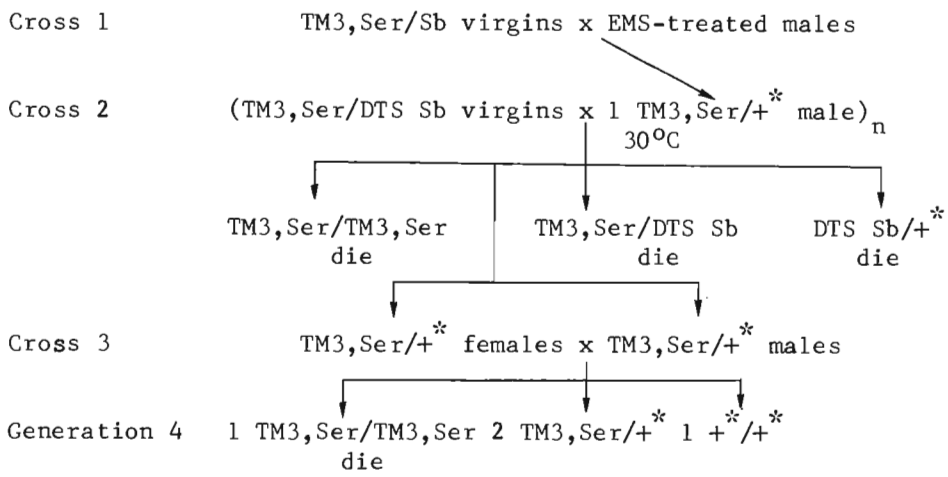
*S_J, Jaccard, 1908; S_{RR}, Russel and Rao, 1940; S_O, in the present study $= (2n_{JK} + n_{jk})/n$; MCD, Cain and Harrison, 1958; S_{SM}, Sokal and Michener, 1958; S_S, Simpson, 1943; S_{RT}, Rogers and Tanimoto, 1960.

was obtained in the case of S_{RR}, UPGA ($r = +0.94$), which showed that New Guinea is nearest to South Asia including Taiwan, with them Japan, Africa, and Micronesia being combined successively, and that Europe and North America make another cluster. In the cases of the cophenetic correlation coefficients higher than +0.80, the resulting phenograms are similar as in S_{RR}, UPGA, while in the cases lower than +0.70, Africa and also Japan are tended to be included in the Europe-North American cluster. The similarity coefficients such as including negative matches (n_{jk}) at least in numerator resulted in phenograms less reliable, showing the lower cophenetic correlation coefficients, and consequently, they should better be avoided in the faunal comparison.

Wright, T.R.F. University of Virginia, Charlottesville, Virginia. A short cut in making autosomes homozygous.

The following crossing scheme, if it works well, should obviate the necessity of collecting from n number of Cross 2 cultures the virgins usually required to set-up Cross 3 which produces individuals homozygous for an autosome in Generation 4. DTS = dominant temperature-sensitive lethal. Cross 2 must be set up at 29 or 30°C.

The dominant visible, recessive lethal mutation, Sb, in the DTS-carrying chromosome is not an absolute requirement, but is convenient for two reasons. If the DTS used is homozygous viable at permissive temperatures, Sb makes it possible to maintain a balanced stock of TM3, Ser/DTS Sb. Second, the absence of Sb in the progeny of Generation 4 indicates that Cross 2 behaved as expected without producing any surviving, fertile DTS Sb/+* or TM3,Ser/DTS Sb individuals. A similar series of crosses can be used for Chromosome 2 using the appropriate balancers and a good second chromosome DTS.



Since we have been blessed with an exogenous supply of third chromosome recessive lethals, we have used the above scheme only once in a very preliminary experiment. Males in Cross 1 were fed EMS according to the method of Lewis and Bacher, DIS 43: 193. For Cross 2 n was only equal to 100 and only 2 TM3,Ser/DTS Sb virgins were used in each vial at 30°C. Of these 26 didn't go. The parents were cleared from the remaining 74 cultures, and when the progeny hatched they were blindly shaken into new vials at room temperature to start Cross 3. These cultures did not go immediately (perhaps due to a temporary heat-induced male sterility), and it was 15 to 16 days at approximately 23°C before sufficient individuals of Generation 4 had hatched to check for lethals. Of the 74 Cross 3 cultures set up, eleven did not go. Of the 63 Cross 3 cultures that went, four produced some progeny in Generation 4 that carried Sb. The presence or absence of a lethal could still be determined in these Sb contaminated cultures, and therefore the overall yield of useful cultures was 63%.

The DTS used in the above experiment was DTS-I165 which along with a second chromosome DTS (which has not been used yet) was very kindly sent to us by David Suzuki.

Research supported by NSF Grant GB 7707.

Bennett, J. and M.A. Walke. Northern Illinois University, DeKalb, Illinois. Behavioral correlates of the w, w^+ gene substitution.

A pair of isogenic, inbred Oregon-R lines differing only at the white locus, were examined for behavioral differences. The lines represented 60 generations of sib-pair matings and 50 generations (25 cycles) of backcrossing with the w allele. 100 flies of each sex were used from each line (designated ORI for the w^+ line and ORIW for the w line). Observations were made in small polystyrene petri dishes under 10x and 20x stereoscopic magnification. Flies were several days old, but not selected for age. Observations were made of pairs of flies, male and female, for 10 minute periods. A behavioral sequence was only counted once in a period for each fly.

Continued at bottom of page 141

Vaidya, V.G., N.N. Godbole and R.M. Kothari
University of Poona, India. Analysis of the excretory products of some species of Drosophila.

An attempt is made to study the excretory products of D. melanogaster, D. ananassae and D. repleta. Cultures of these species were individually grown under identical conditions in sterilized containers on the standard agar-cornmeal medium. The excreta of adult flies were carefully collected from the walls of the containers. It was dissolved in ice-cold glass-distilled water separately for each species without resorting to acid-, alkali- or heat-treatment as these may cause certain chemical and degradative changes. The solutions were individually spotted by capillary on Whatman No. 1 qualitative papers, which were then run in glacial acetic acid:n-butanol:water:1:4:5 phase for 4 hours at 27 degrees centigrade by circular chromatographic method after taking the usual precautions (Long et al., 1961). The chromatograms were then dried in air. A set of chromatograms, four for each species, was developed to test amino acid contents of excreta by spraying with 0.5% ninhydrin in acetone and dried at 70 degrees centigrade for 2 minutes. A second identical set was developed for testing the carbohydrate contents of excreta by spraying with 0.5% aniline phthalate in acetone and dried similarly. A third identical set was viewed in dark under 'chromatolite' having emission range 230-290 mu for UV positive spots, if any.

Qualitative tests for uric acid (Brown's reaction), glyoxylic acid (Fearon's test), urea (Sumner's urease test), ammonia (Kroupas's paper test) and creatinine (Kolisch's test) were performed (Welcher, 1966).

All the species showed invariably the presence of uric acid band as judged by the Rf value (0.32) and by Brown's qualitative colour reaction (Brown, 1945). Characteristic absorption maxima at 292 mu also confirmed the presence of uric acid in the excreta of all the three species. Test for glyoxylic acid was positive while those for urea, ammonia and creatinine were negative.

D. ananassae shows an additional UV positive spot on the chromatogram, which from Rf value calculations (0.18) appears to correspond to either adenylic acid or uridylic acid. However, the presence of these components is not yet confirmed by other qualitative tests. Further studies are in progress.

References: Brown, H., 1945, The determination of uric acid in human blood. J. Biol. Chem. 158: 601-608. Long, C., King, E.J. and Sperry, W.M., 1961, Biochemist's Handbook, E. & F.N. Spon Ltd., London. Welcher, F., 1966, Chemical Solutions, D. Van Nostrand Co. Inc. New York.

Bennett, J. and M.A. Walke: Continued from page 140

Both lines showed a bimodal distribution of total activity on an arbitrary scale, but the distributions were radically different (χ^2 = 64, 8 d.f., $P \ll 0.0001$) between the lines. ORI had more individuals at the extremes of activity, ORIW had more with intermediate activities.

A leg rubbing operation where one middle leg was used in conjunction with the contralateral foreleg to rub the other foreleg, designated "three legged front", was observed. A "circling and backing" motion was also noted to have a different frequency in the two lines. "Wing combing" during the observation period also appeared to differ between the lines. The table shows the relationship:

Line	Expression	Wing combing	Circling & backing	Three legged front
ORI	+	151	1	111
	-	49	199	89
ORIW	+	131	12	83
	-	69	188	117
	χ^2	4.81	8.02	7.84
	P	0.03	0.0045	0.005

Of 13 behavioral patterns observed 3 appear to show differences that we may attribute to the substitution of w for w^+ in the homozygous Oregon-R background. In addition a general activity difference is apparent. The association of 4 of 14 measures with the single gene difference can be taken as an indication that such studies are likely to be worth continuing effort.

Metcalfe, J.A. University of York, Heslington, England. A dumpy lethal affecting larval moulting in D. melanogaster.

The change from one larval instar to the next is far from fully understood. The transition is under the control of the moulting hormone (ecdysone) and involves not only the actual physical process of moulting (ecdysis) but also the duplication of chitinous structures which precedes it.[1,2] That both these processes require ecdysone for at least their initiation, has been demonstrated by Hanser[3] in Ephestia.

Both the events - duplication and ecdysis - are affected in "Lethal Stuck". These larvae homozygous for one of the dumpy alleles, $dp^{lm\ 4}$, fail to complete these processes. Death usually occurs at the first/second larval boundary, and, rarely at the second/third larval boundary. The majority of lethals attempt to free themselves from their old chitin coat, but the coat itself always remains intact. The suspensoria of the mouthparts always remain within the body although the jaws are almost always thrown off. Many larvae withdraw from their posterior spiracles which remain attached to the tracheal trunks (figure 1); and, some withdraw from the old chitin coat incompletely, for, certain regions of the body wall, usually the anterior and/or the ventro-posterior tips, still remain attached to it. These regions of the body become stretched as the larvae contract while attempting to free themselves (figure 1).

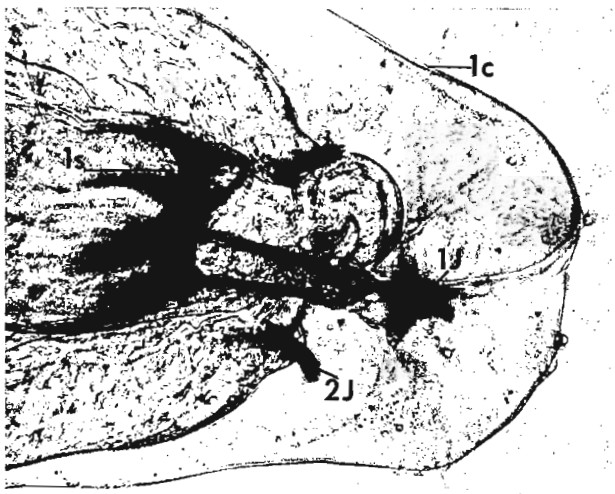


Figure 1. "Lethal Stuck" withdrawn from posterior spiracles (p) and still attached to the ventroposterior part of body wall (b).

Figure 2. Anterior region of a "Lethal Stuck" with incompletely duplicated mouthparts, i.e. second instar jaws only (2J); withdrawn from first instar coat (1c); thrown off first instar jaws (1J); first instar suspensoria still within the body (1s).

The duplication of chitinous structures viz., mouthpart apparatus, tracheae, posterior spiracles and chitin coat may also be incomplete. The lethals show a wide variation in the amount of duplication and differentiation of these structures. It is interesting that the individuals attempt to moult even in the absence of duplicated chitinous structures (figure 2). But even where the structures are fully duplicated, ecdysis is not brought to completion.

The "Lethal Stuck" shows that these two processes - duplication and ecdysis - can be separated because they can vary independently of each other. Such lethals may prove to be useful in providing information about the mode of action of hormones involved in moulting, the target organs of the hormones and their response.

The expression of other dumpy lethals is to be reported elsewhere[5]

References: 1. Bodenstein, D. 1944, The induction of larval moults in D., Biol. Bull., Wood's Hole 86: 113-124. 2. Novák, V.J.A. 1966, "Insect Hormones". Methuen & Co. Ltd. 3. Hanser, G. 1957, Wirkung eines Metamorphose - Hormons bei Ephestia kühniella. Zool. Anz., 20: 209-215. 4. Lindsley, D.L. and Grell, E.H. 1967, Genetic Variations of D. melanogaster. Carnegie Inst. Publ. 627. 5. Metcalfe, J.A. (in press) Development and complementation patterns of lethal alleles at the dumpy locus of D. melanogaster.

Kaneshiro, K.[1] and M.R. Wheeler. University of Texas, Austin, Texas. Preliminary report on the species of the ananassae subgroup.

Using stocks at the University of Texas laboratory, comparisons of male genitalia suggest that this subgroup of the melanogaster species group may consist of 11 species, divisible into the ananassae complex (5 spp) and the bipectinata complex (6 spp). Table 1 shows our tentative separation of the available stocks, some morphological traits, and the origins of the strains used. Figure 1 shows the mataphase chromosome complements.

Table 1. Tentative arrangement of the ananassae subgroup species.

	metaphase type	front basitarsus	second tarsomere	male abdomen	origin of stocks used
sp. 1 (ananassae)	I	4-5 rows of small combs	3 rows of small combs	pale*	Marshall Is., Tonga, Samoa, Niue I., Hawaii
sp. 2	I	2 rows of small combs	2 rows of small combs	dark	Philippine Is.
sp. 3	II	as in sp. 2	as in sp. 2	pale	Palau (Caroline Is.)
sp. 4	I	4-5 rows of small combs	3-4 rows of small combs	dark**	Philippine Is.
sp. 5	I	as in sp. 4	as in sp. 4	bit darker	Fiji Is.
sp. 6 (bipectinata)	III	2 sets of strong combs	1-2 strong apical spines	pale	India, Nepal, Pakistan, Taiwan, Philippines, Fiji, Cambodia, Samoa, New Guinea***
sp. 7	III	as in sp. 6	as in sp. 6	dark	Cambodia, Philippines
sp. 8	I	2 rows of small combs	1 set of small combs	pale	Australia, New Guinea
sp. 9	IV	as in sp. 8	as in sp. 8	dark	Malaysia, Borneo
sp. 10	I	1-2 rows of small combs	1-2 rows of small combs	pale	Cambodia, Philippines
sp. 11 (? - malerkotliana)	III	as in sp. 10	as in sp. 10	dark	Malaysia

*darker in Samoa-Fiji area. **thorax also dark. ***type culture of D. szentivanii.

TYPE I TYPE II TYPE III TYPE IV

Fig. 1. Metaphase types seen in ananassae subgroup.

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Kurokawa, H. Tokyo Metropolitan University, Japan. Experiment on sexual isolation between two different strains of D. asahinai.

Two wild strains collected at distantly separated localities, Komi in Okinawa and Yunsui in Formosa were examined for sexual isolation. To test for mating preference, the usual male multiple choice technique was employed at 25°C±. The result is summarized in the table. Incipient sexual isolation is one-sidedly demonstrated. The cross using Komi male showed high K-value (Levene 1949) with 0.313, which was significantly deviated from random mating. Accordingly, the χ^2 value was also large. On the other hand, the cross using Yunsui male showed very small K-value with -0.047.

Cross	Homo(%)	Hetero(%)	$K_{1,2}$ & $K_{2,1}$	K_1 & K_2	χ^2	p
Komi,Yunsui♀ x Komi♂	60.0	38.0	0.313	0.133	10.62	0.01
Komi,Yunsui♀ x Yunsui♂	50.0	53.1	-0.047		0.18	0.7 ~ 0.5

Lifschytz, E.* and R. Falk. Hebrew University, Jerusalem, Israel. Fine structure analysis of the chromosome. Recombination values in the ma-1 region.

Lethals covered by Y mal^+ have been mapped by complementation to a sequence of 34 complementation units. Some recombinational data in this region of the euchromatic-heterochromatic junction have recently been obtained in crosses of the type y v f 1^L+/+ + + + 1^R x M5/Y where the only viable males are recombinants.

The region investigated, which spans units 2-34 in our recent map (Mutation Research 8, 1969), encompasses 2.3 recombination units.

The gene order obtained in this recombination analysis is consistent with that obtained by the complementation tests.

It should be emphasized that we use only complete lethals, and only those giving no indication of any crossover inhibition. Additive recombination values were obtained in the various crosses.

Assuming the region to be saturated with lethals, the recombination value per cistron for the different regions of the map can be calculated. Some data presented in the table show that the region can be divided into two subregions.

	Units	Total Recombination	Recombination per Cistron
Subregion I	2-17	1.5%	~0.1%
Subregion II	17-34	0.78%	~0.045%

Although the data do not exclude some variation in the amount of recombination in different cistrons of each of the two subregions, it seems clear that in Subregion I the average level of recombination is twice as high as that in Subregion II.

Parental Constitution	Complementation Units	No. Females Counted	% Recombinants	Recombinants/ Cistron
1. P235/Q463	2-34	26,813	2.310	0.69
2. P235/1^{A7}	2-17	7,144	1.530	0.10
3. E54/Q463	17-34	12,075	0.778	0.045
4. E81/E54	6-17	9,600	9.937	0.085
5. P235/Q256	2-7	7,646	0.444	0.085
6. W3/Q256	6-7	18,795	0.117	0.11
7. E81/Q256	6-7	19,490	0.123	0.12
8. W3/R9-28	6-7	18,632	0.107	0.10
9. E54/Q2	17-23	13,144	0.076	0.015
10. 3^{DES}/Q463	28(30)-34	16,355	0.025	0.012-0.005

It is worth indicating that a hot spot for X-ray induced breaks (presumably an intercalary heterochromatic region) is located just to the right of unit 17.

Data characterizing the two subregions in different pairing conditions as well as other features of recombination at this region will be presented elsewhere.

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Sakaguchi, B. Kyushu University, Fukuoka, Japan. Effects of chloramphenicol and actinomycin-D on SR spirochetes in Drosophila.

In order to examine whether SR spirochetes in SR flies are artificially eliminated by antibiotics, chloramphenicol and actinomycin-D were injected into female flies of a SR line of D. melanogaster, Oregon strain, with nebulosa SR spirochetes. Concentrations of chloramphenicol and actinomycin-D were 1400 μg and 400 μg per milliliter respectively. The injected volume was 0.5 microliter per fly. The injected SR flies were kept for 27, 50 and 190 hours in 25°C and their hemolymphs were sucked into a micropipette, then they were injected into each 15 normal female flies of Oregon inbred line. Sex ratios of progenies from the injected flies were examined. These results are summarized in the Fig. 1 and 2.

It has been demonstrated from dilution experiments of the SR spirochetes by Sakaguchi and Poulson (1961) that the SR flies of a certain species of Drosophila have a large number of the SR spirochete in their hemolymph and the time of appearance of SR condition in the progenies from the flies injected with the spirochete was dependent upon the number of the micro-organisms. It can be said from the facts that sensitivities of the SR spirochetes to chloramphenicol and actinomycin-D will be seen by the length of time in appearance of SR condition in the progenies from normal female flies injected with the SR hemolymphs treated by those antibiotics.

When hemolymphs from SR females of 27 and 50 hours after injection of chloramphenicol were injected into normal females, SR condition which produces one hundred percent females in the progeny, appeared from the first to the successive broods (Fig. 1). In the case of hemolymphs from SR females of 190 hours injected into normal females, SR condition appeared at the 15th day brood (Fig. 1). The concentration of chloramphenicol was very high and the injected SR females never produced their progenies. However, effect of the concentration of the antibiotics on the SR spirochetes was rather weak.

When hemolymphs from SR females of 27 and 50 hours after injection of actinomycin-D were injected into normal females, SR condition appeared at the 21st and the 24th day brood (Fig. 2). With the concentration used in this experiment of actinomycin-D, they never produced their progeny, but the SR spirochetes were not completely eliminated by the antibiotics.

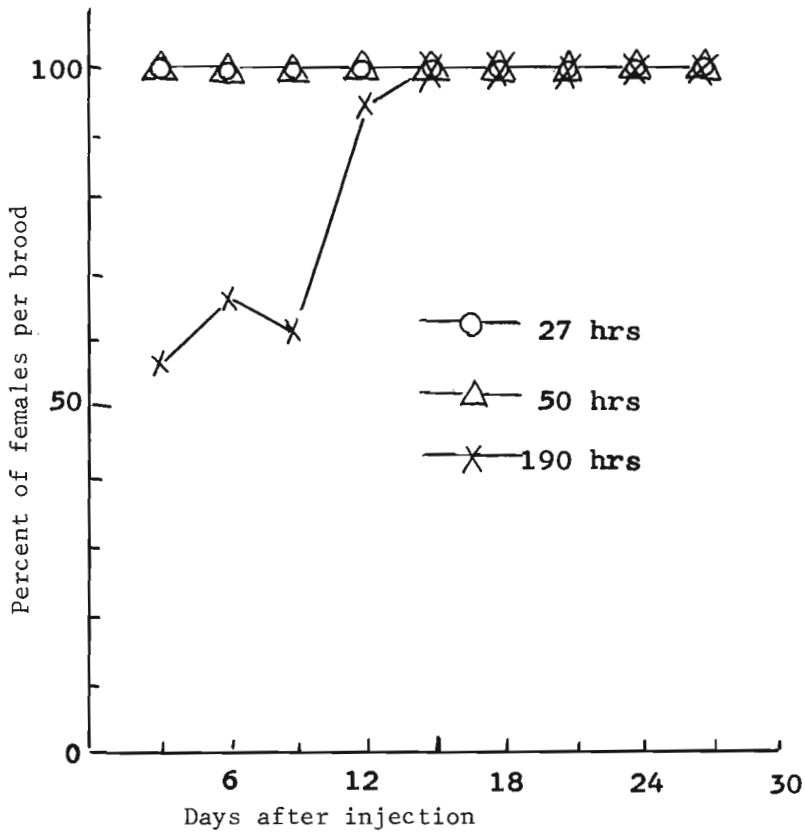


Fig. 1. Effect of chloramphenicol on SR spirochetes.

Solution of chloramphenicol was injected into SR females flies and hemolymphs of the injected flies were sucked out at the time of indication in the figure. The hemolymphs were then injected into normal females of Oregon strain and were examined for female percent per brood.

These results show that the effect of actinomycin-D which inhibits DNA-dependent RNA synthesis on inactivation of the SR spirochete is more predominate than chloramphenicol which inhibits protein synthesis.

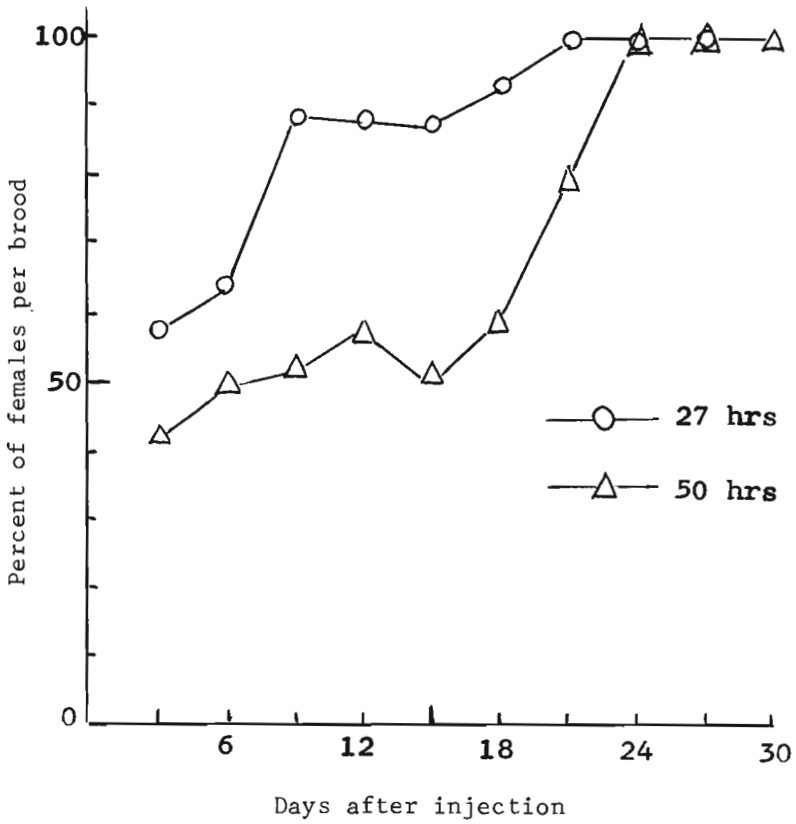


Fig. 2. Effect of actinomycin-D on SR spirochete.

Procedures of this experiment were the same as in Fig. 1.

To make clear the properties of multiplication of the SR spirochete, a more detailed examination of this sort is now underway. (Support by PHS Grant GM10238 of USA and a Grant 36001 from the Ministry of Education of Japan.)

Yoon, J.S. and W.C. Kim. Yonsei University College of Medicine, Seoul, Korea. Genetic effects of a synthetic ovarian steroid in D. melanogaster.

Effects of a synthetic ovarian steroid on genetic materials were studied in Drosophila treated with Lyndiol 2.5 (Lynostrenol 2.5 mg and Mestranol 0.075 mg/tablet). Germ cells of males ($sc^8.y.B^S/y^2\ w^i\ ct^6\ f^1$) reared on the medium containing 0.5 ml of Lyndiol (50% in Drosophila Ringer's) solution through imaginal stages were tested for genetic damage. When males treated were crossed individually to multipurpose virgin (y sc^{S1} In49 sc^8;dp bw;st p^p),

Table 1. Mutations and chromosomal abnormalities in D. melanogaster treated with Lyndiol 2.5.

	Treated		Control	
Aberrations	Total No. Studied	% With Aberration	Total No. Studied	% With Aberration
Loss of Y	8,829	0.14	6,605	0.08
Nondisjunction	18,453	0.37	12,323	0.14
Visible mutations	18,453	0.07	12,323	0.00
Lethal mutations	1,628	0.49	1,389	0.07
Translocations	1,558	0.00	1,215	0.00

increased nondisjunctions, losses of the Y chromosome, and other visible mutations were found. The rate of sex-linked recessive lethal mutation was 0.5% (8 out of 1,628 chromosomes tested) in the group treated, and no translocation was found (Table 1). The data suggest that the hormone may act as a mutagen in Drosophila.

Meyer, Helen U. University of Wisconsin, Madison, Wisconsin. An iso-allele of the dumpy lethal.

Two similar cases of an unconventional mutation were found, located on second chromosomes of D. melanogaster from nature (Madison, Wisconsin, 1966); they were probably of common origin. These chromosomes from nature ($+_n$) were lethal in combination with $dp^{lv\ I}$ Cy, Ins CyO pr cn^2 sp ($dp^{lv\ I}$ = dumpy-Thoraxate of Ives). This suggested that we were dealing with an allele of some lethal present in this Curly chromosome, most likely with a lethal allele in the dumpy region. But contrary to expectation it was then found that homozygotes $+_n/+_n$ were viable and of wild type appearance. A homozygous stock could be established which, however, showed higher egg mortality than a typical wild type stock.

The tests with $dp^{lv\ I}$ were repeated with the same result, and crosses made to other mutant alleles of the dumpy region. The following results were obtained:

Lethal combinations:		Viable combinations and wild type heterozygotes	
$+_n/dp^{lv\ I}$	(dumpy-Thoraxate)	$+_n/dp^{lv}$	(dumpy-thoraxate)
$+_n/dp^{olv}$	(dumpy-Truncate)	$+_n/dp^{cm2}$	(dumpy-comma)
$+_n/dp^{lM}$	(dumpy-lethal)	$+_n/dp^{ov}$	(dumpy)

Non-lethality (complementation) with thoraxate was unexpected. Localization of the factor responsible for lethality in combination with dp^{lM} was carried out by crossing females $+_n/S$ Sp Bl L^{rm} bw^D to males dp^{lM}/S^2 Cy, Ins(CyL+R)... Classification of the non-curly offspring for the dominant markers S, Sp Bl, L^{rm}, bw^D placed the factor between S and Sp and indeed showed that it was located in the dumpy region.

This mutation can be interpreted as an iso-allele at the dumpy-lethal sublocus, or at one of them, if there should be more than one such sublocus. It must be considered a hypomorph, since it produces some, but less than the normal amount of a gene-initiated product necessary for survival. Two doses of it, as in homozygotes, are sufficient; one dose is not sufficient in combination with other (amorphic) dumpy-lethals. An exception is the case of dp^{lv}, which likewise must be a hypomorph and apparently is a less drastic mutation than $dp^{lv\ I}$.

This dumpy-lethal isoallele might be useful in attempts to discriminate between the potentialities of various dumpy-lethal mutations, in a similar way as dp^{cm2} is useful. It also might be a tool in some biochemical investigations of that region. On the basis of this tentative interpretation the symbol dp^{lMi} (dumpy-lethal iso-allele) is suggested for this mutation.

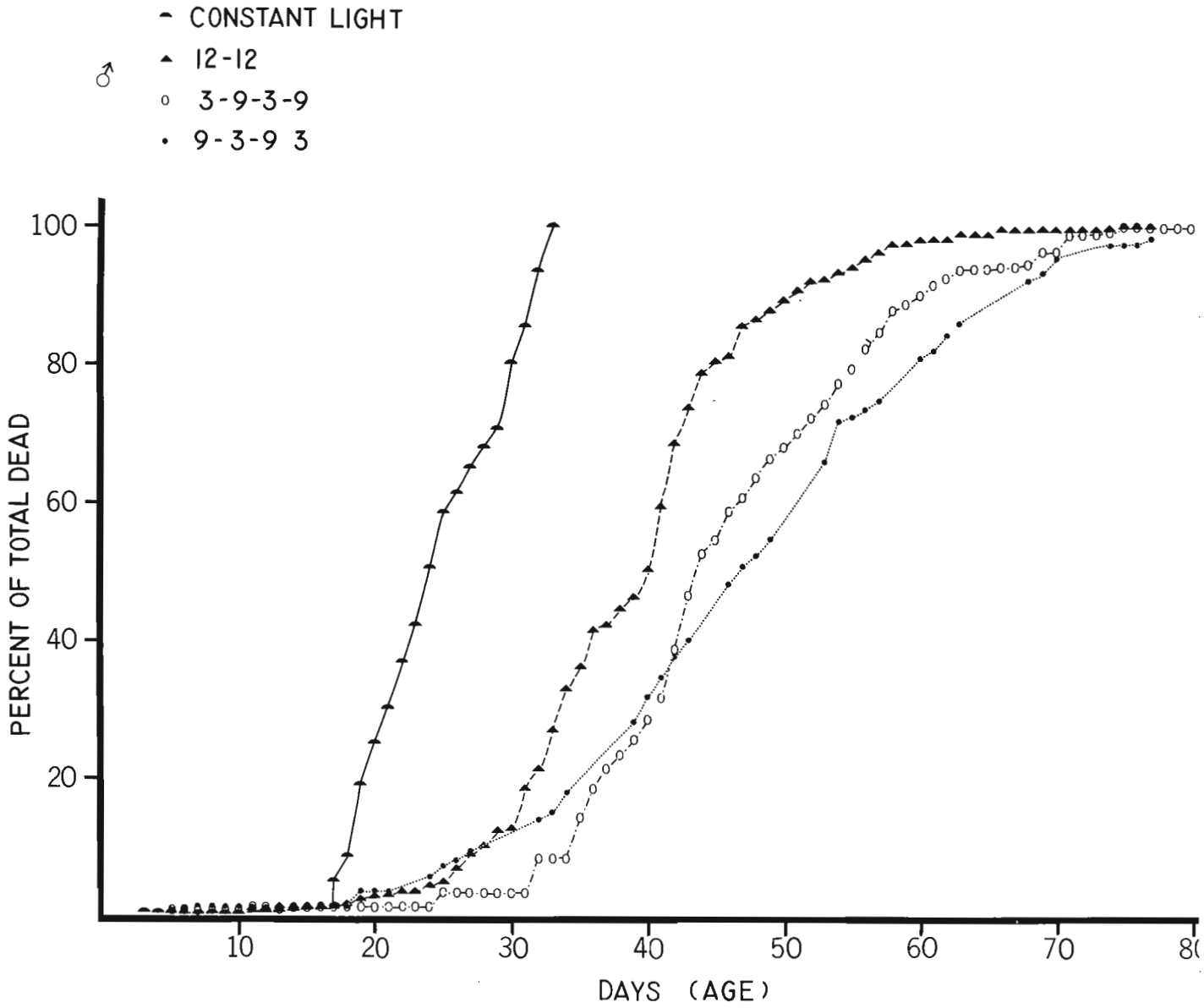
Williamson, J.H. University of California Riverside, California. Simultaneous recovery of two detachment-X chromosomes from an irradiated female.

The model of directed disjunction predicts that subsequent to an induced interchange in immature oocytes the affected centromeres will segregate during anaphase I (Parker, 1969). Consistent with this prediction is the observation that induced detachments of a compound X chromosome are recovered singly. An exception to this rule was recently recovered from a C(1)RM, y v bb/0;$y^+ \cdot spa^{pol}$/ci ey^R female treated with 2000 r of X rays, mated to y w^a $Y^L \cdot Y^S$/Y;spa^{pol} males and brooded daily. The exceptional female was v and at first assumed to be triplo-4 or (more likely) to carry a recombinant $y^+ \cdot$ci ey^R fourth chromosome. All exceptional progeny were being tested to determine their chromosomal complements and this female was found to carry two detachment-X chromosomes. One was y v bb$\cdot y^+$, the other was y v bb$\cdot$ci ey^R. In addition she carried a paternal fourth marked with spa^{pol} and a Y chromosome. The recovery of these two detachments required at least three induced breaks and cyclical interchange. At anaphase I the centromeres from the compound-X and the y^+-marked fourth segregated from the other fourth centromere. At anaphase II non-randomness would prefer the detachment capped with ci ey^R but not the captured detachment marked with y^+. However, both were incorporated into the oocyte, with no free maternal fourth chromosome. This exception, along with those described in DIS 43: 178, adequately demonstrate that multiple break rearrangements can be recovered and recognized only if one thoroughly analyzes all exceptional progeny.

Reference: Parker, D.R., 1969, Mutation Res. 7: 393-407.

Erk, F.C. and H.V. Samis, Jr. Masonic Medical Research Laboratory, Utica, New York. Light regimens and longevity.

Preliminary experiments designed to test the effects of various environmental stresses on duration of life in D. melanogaster strongly suggest that the total amount and sequencing of light have an important bearing on longevity. In these experiments, mass bred Oregon-R flies were collected as pupae, and upon hatching, 20 flies of one sex were placed without etherization into each of 10 vials containing standard medium. Each set of replicates (N=200) was placed in a particular 24-hour light regimen: (1) constant light; (2) 12 hours light, 12 hours dark; (3) 3 hours light, 9 hours dark, 3 hou light, 9 hours dark; and (4) 9 hours light, 3 hours dark, 9 hours light, 3 hours dark. Daylight fluorescent lights were used; temperature was maintained at 25°C, and hunidity was about 46%. Survivors were counted daily in most instances. The mortality curves for males are shown in Figure 1; data for females are comparable.



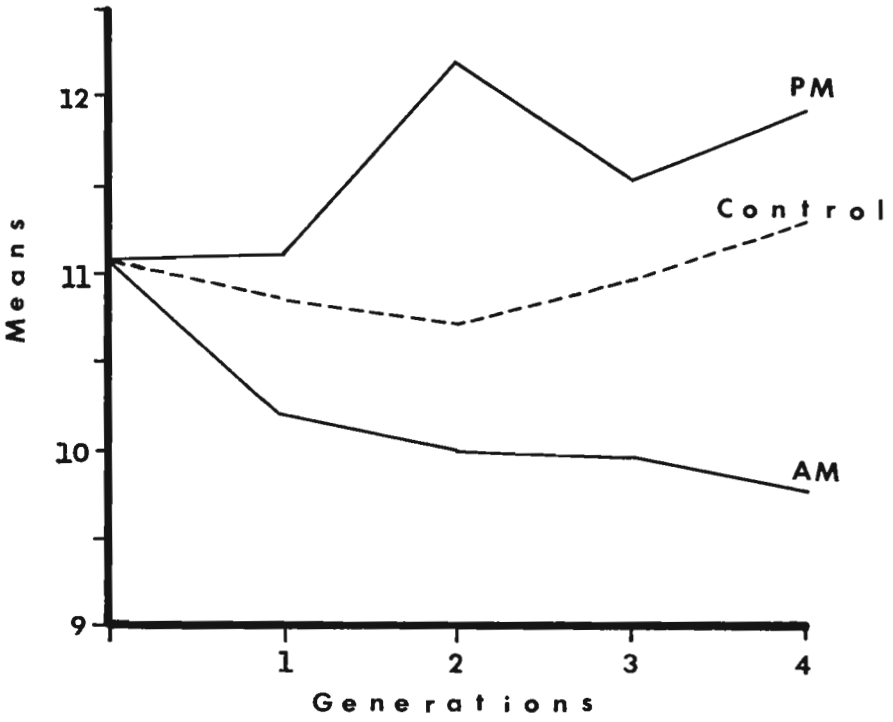
In constant light, all flies are dead within about a month. When dark periods are interposed, the shapes of the mortality curves are quite different, and some flies live for 75-80 days. Flies are longest lived when two subcycles per day are imposed, and it appears that under these conditions a total of 6 hours or 18 hours of light per 24-hour period gives similar results. It would be desirable to have data for longevity in constant darkness, but these experiments involve certain technical difficulties, as yet unsolved.

Grant, B.S. and W.L. Harrison. College of William and Mary, Williamsburg, Virginia.

Selection on the eclosion rhythm of D. melanogaster.

In preliminary directional selection experiments performed as the first phase of a disruptive selection program, we have confirmed the results of Pittendrigh (P.N.A.S. 58: 1762) demonstrating that the eclosion profile of Drosophila can be altered via selective breeding.

Our base population was derived from a four-way gene pool cross of the inbred lines Swedish-B, Oregon-R, Samarkand and Canton-S. Within the first generation, small but distinct differences between the eclosion profiles were obvious for the populations selected for early morning and late afternoon emergence from the puparium. The mean eclosion times of the selected lines continued to diverge from each other and the control (unselected) population without overlap (see figure). The response to selection, thus far, has been asymmetric. Both selected populations continued to peak at "dawn" (12:12 LD cycle) but the "PM" population showed an additional peak at "dusk" which appeared to increase each generation with the concomitant diminution of the morning peak. The "AM" population profile appeared quite similar to the unimodal control except for an exaggerated increase in the peak of emergence at "dawn."



Over four generations of selection, the average realized heritabilities for AM and PM are 0.24 and 0.10 respectively. These estimates are somewhat atypical. Since the data are cyclical, the extremes in eclosion time, either very early (just after midnight) or very late (just before midnight) would differ greatly in score on a linear time-of-day scale of one through 24 hours; however, in terms of a diurnal rhythm of eclosion, rather than simply developmental rate, such individuals differ only slightly. In order to avoid the ambiguities of possible overlapping of daily distributions, the tails of the distributions were truncated arbitrarily for a four hour block of time between the two hours immediately preceding and succeeding midnight. Actually, because so very few flies emerge during this interval, the effect of truncation on mean estimates is negligible. (Supported by NSF-GU-3111-M.)

Miller, D.D. University of Nebraska, Lincoln, Nebraska. Evidence of "eastern" D. athabasca XL inversion associations in the XL patterns of other D. affinis subgroup species.

As reported by Miller and Voelker (1969), the salivary gland chromosome patterns of the long arm of the X of "western" and "eastern" D. athabasca appear to be differentiated by a minimum of five inversions: MI, MII, MVI, MVII, and MVIII. Recently XL patterns have been studied in five related species: D. affinis, algonquin, azteca, narragansett, and tolteca. Although it is not yet possible to interpret the XL sequences of these other species in terms of all the material of the athabasca XL strand, one can nevertheless recognize some pattern associations attributable to certain of the inversions (MI, MVII, and MVIII) distinguishing the sequences of "eastern" athabasca from numerical Sequence I of "western" athabasca. These are either actual stretches of pattern like those of "eastern" athabasca inversion break point regions or, at least, cases involving discontinuities coinciding with the "eastern" athabasca inversion break points and

hence interpretable as possibly related to the athabasca Sequence I by way of these inversions. The following table presents these findings. The athabasca inversions are identified by their symbols and by the associations at their break points (section numbers from

D. athabasca inversions		D. affinis Subgroup Species				
		affinis	algonquin	azteca	narragansett	tolteca
M I	3'15	+?	present	present	+?	present
	4'16	+?	+?	+?	?	+?
M VII	27d'34	+?	present	+?	+?	+?
	27p'35	present	present	present	present	present
M VIII	27d'30	+?	-	present	present	?
	34'29	+?	-	present	+?	present

the Sequence I XL map of Miller and Voelker '69). Cases in which the indicated inversion break point region association was found are designated by "present", those in which the same inversion break may have occurred as an intermediate step by a "+?", those in which the inversion association was definitely absent by a "-", and cases in which no decision could be reached by a "?". These findings provide additional evidence of an intermediate phylogenetic position of "eastern" athabasca between "western" athabasca and other D. affinis subgroup species (though not necessarily in a linear phylogeny). Such a position of "eastern" athabasca was also implied by patterns of the C Chromosome (Miller and Sanger, 1968).

1) Miller, D.D. and Sanger, W.A. 1968. Journal of Heredity 59: 322-327. 2) Miller, D.D. and Voelker, R.A. 1969. Journal of Heredity 60 (in press at the time of this report).

Baldwin, D.G. University of Arizona, Tucson, Arizona. The frequency of inversion sequences in D. pseudoobscura in southern Arizona.

D. pseudoobscura females were collected during the months of October through January through two winters (1968-69 and 1969-70) from four locations in southern Arizona. Sycamore Canyon and the Patagonia Dam Road in the Patagonia Mountains are at 4500 ft. in oak woodland. Madera Canyon Road, at the base of the Santa Rita Mountains, is at 3500 ft. in desert scrub. Soldiers Trail, at the base of the Santa Catalina Mountains, is in desert scrub at 2900 ft. The collections were taken from the Patagonia Mountain sites in 1968-69 only and from Soldiers Trail in 1969-70 only, but collections were made during both winters at Madera Canyon Road. The traps consisted of large cans baited with fermenting bananas.

The gene arrangements of both homologues of the third and X-chromosomes were scored for one female larva from each wild female collected. The total number of chromosomes examined

Locality	n	AR	ST	CH	PP	SR
Sycamore Canyon	8	87.5	0	12.5	0	0
Patagonia Dam Rd.	40	77.5	20	2.5	0	10
Madera Canyon Rd.	102	67.6	25.5	5.9	1.0	9.8
Soldiers Trail	28	64.3	28.6	7.1	0	17.8

(n) was 178. The frequency of the sex-ratio (SR) sequence of the X-chromosome is significantly greater at Soldiers Trail than at the other sites. No larvae were found to be homozygous for sex-ratio. Only one female produced unisexual offspring, indicating that only one of the females collected had been inseminated by a male with the sex-ratio inversion.

The decrease in frequency of Arrowhead (AR) and the increase in frequency of Standard (ST) from Sycamore Canyon to Soldiers Trail probably reflects the decrease in elevation (Patton and Heed, DIS 40: 69). The other third chromosome inversion types found in the study were Pikes Peak (PP) and Chiricahua (CH).

Burdette, W. J. and J. E. Carver. The University of Texas, Houston, Texas. Frequency of tumors in several laboratory stocks of D. melanogaster.

The characteristic frequency with which melanotic tumors occur spontaneously in several different strains of Drosophila is listed below for the years 1951 and 1968. Comparison of these frequencies reveals that, although the observed percentage of tumors in some of the stocks has decreased over the intervening period of 17 years, the frequency of the others has remained relatively constant or has increased. Nutritional conditions, the method of maintenance, and temperature have been kept reasonably constant over the period between observations. A wide spectrum of tumor penetrance among these stocks remains.

Stock	Characteristic tumor location*	1951 with tumors	1951 total observed	1951 percent tumors	1968 with tumors	1968 total observed	1968 percent tumors
tu^{36a} st sr e^{s} ro ca	ab	182	3394	5.4	48	600	8.0
f^{257-19}/In(1)AM	ab	415	2449	17.0	49	700	7.0
tu^{wps}	h	1423	8077	17.6	0	550	0.0
w^{bf} f^{257-5}	ab	715	2827	25.3	196	670	29.2
tu^{50d}	ab	1901	7144	26.6	62	480	12.9
tu^{bw}	ab	2434	8614	28.3	100	100	100.0
tu^{h}	h	6616	12236	54.1	128	350	36.6
vg mt^{A} bw	ab	5944	10069	59.0	637	740	86.1
y B^{263-43}	ab	2274	3120	72.9	47	580	8.1
tu^{g}	ab	9113	11967	76.2	306	600	51.0
tu vg bw	ab	10540	10555	99.7	315	350	90.0

* Tumor location: ab = abdomen; h = head.
Ref: 1951. Burdette, Walter J., DIS 25: 101-102.

Surridge, J. F. University of Nebraska, Lincoln, Nebraska. Some effects of amphetamine salt feeding upon D. melanogaster.

Eggs were collected from D. melanogaster of the Canton-S strain. They were reared in 25 x 95mm shell vials packed half full with "Cellucotton" (Kimberly-Clark) absorbent wadding impregnated with 10ml of yeast suspension. Amphetamine sulfate and methamphetamine hydrochloride were added to autoclaved yeast suspension (14gr of dry yeast/100ml H_2O) at 1.0gr/100ml and 1.5gr/100ml dosages. Eggs were reared in yeast suspension as a control.

Males hatching from control and amphetamine treated eggs were mated with Muller-5 virgins to test for the frequency of recessive lethality. The tests were run in three series. F_1 pair matings were scored for fertility and their offspring for evidence of recessive lethality. The results are summarized in the following tables.

Table 1. Percentage of successful cultures in F_1 pair matings.

	I. TOTAL	I. % SUCCESS	II. TOTAL	II. % SUCCESS	III. TOTAL	III. % SUCCESS
Control	113	89.38%	219	81.25%	73	90.42%
Am. sulf. 1.0	337	79.83%	-	-	-	-
Am. sulf. 1.5	-	-	189	72.59%	117	82.05%
Meth. HCl 1.0	-	-	123	86.18%	-	-
Meth. HCl 1.5	-	-	24	79.13%	165	90.30%

Table 2. Frequencies of recessive lethality in X chromosomes.

	CHROMOSOMES TESTED	LETHALS	PERCENTAGE
Control	426	2	0.47
Am. sulf. 1.0	268	0	-
Am. sulf. 1.5	229	3	1.31
Meth. HCl 1.0	105	0	-
Meth. HCl 1.5	168	0	-

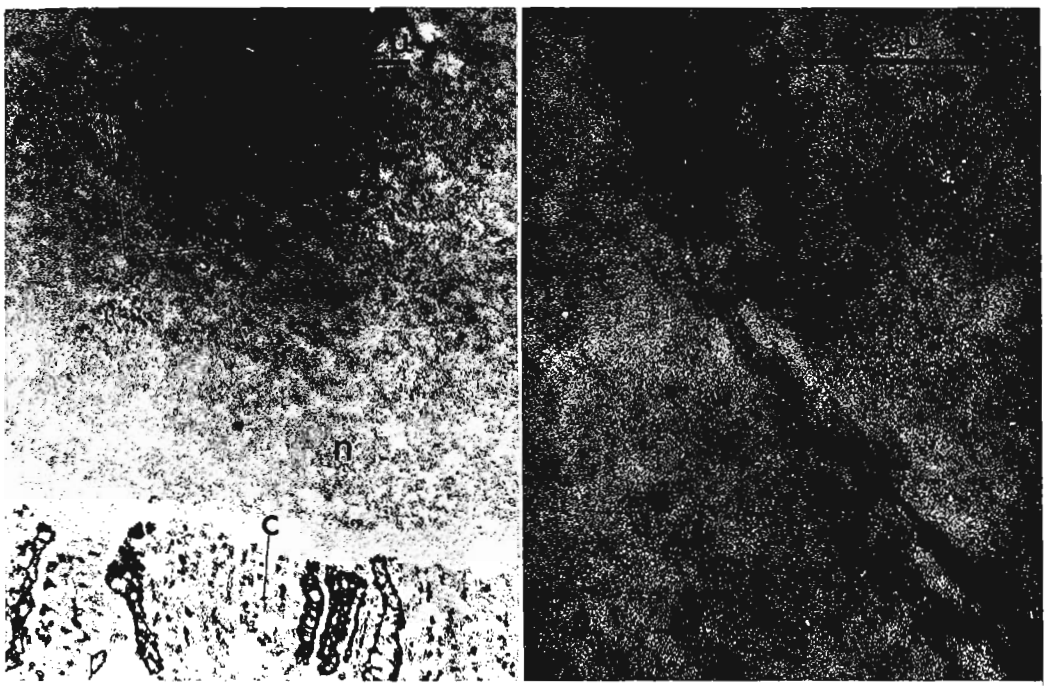
Continued on bottom of page 152

Ellison, J.R. and N.A. Granholm,
University of Oregon, Eugene.

Multi-stranded nucleolar DNA in polytene salivary gland cells of Samoaia leonensis Wheeler (Drosophilidae).

The Feulgen positive bodies in the nucleoli of salivary gland cells from late third instar larvae were first described by Nash and Plaut (1965). Barr and Plaut (1966) showed that these bodies vary greatly in morphology among the various species of Drosophila. In S. leonensis these bodies take the form of strands of varying degrees of development and appear in both sexes. In extreme instances periodic banding can be seen at the light microscope level which is reminiscent of salivary chromosome banding. The salivary glands were prepared as described elsewhere (Ellison, D.I.S. 45). The electron micrographs showed that the strands were multiple in nature. Some banding could be seen. In general the strands resembled very severely stretched polytene salivary chromosomes. The strands did not appear to be connected to the chromosomes.

Barr, H.H. and Plaut, W., 1966, J. Cell Biol., 31, C17. Nash, D. and Plaut, W., 1965, J. Cell Biol., 27, 682.



Electron micrographs of S. leonensis female nucleolar DNA.

s. Nucleolar chromatin strand
c. Polytene chromosome
n. Edge of the nucleolus
b. Periodic banding

Surridge, J.F.; continued from page 151

Amphetamine sulfate treatment at 1.0 and 1.5gr/100ml apparently causes a reduction in the percentage of successful F_1 crosses of heterozygous Bar females and "Basc" males. Methamphetamine hydrochloride does not seem to alter the success of F_1 pair matings significantly. There appears to be an elevation of the frequency of recessive lethality in 1.5 amphetamine sulfate treated flies. Further investigation is necessary to substantiate this elevation. Injection experiments are planned for subsequent experimentation.

Angus, D.S. University of Queensland, Brisbane, Australia. The relationship of two sibling species within the quadrilineata species group of Drosophila.

In 1964 during a cytological analysis of D. tetrachaeta two flies (a male and a female) from Brown River, near Port Moresby were detected that, although morphologically identical with D. tetrachaeta, were very different as regards inversions present. In 1966 eight flies (three females and five males) were examined from Cairns which were cytologically similar to and would freely cross with the Brown River flies. Cultures established from Brown River and Cairns would not hybridise with D. tetrachaeta from Brown River beyond F_1 pupae. On this evidence a sibling species to D. tetrachaeta viz. D. pseudotetrachaeta was described (Angus 1967).

It is the purpose of this paper to describe as far as possible the specific inversions of D. pseudotetrachaeta and to record the degree of sexual isolation from D. tetrachaeta.

Sexual isolation tests between the two species were carried out by confining 10 sexually mature flies of one sex with 10 flies of the opposite sex and strain and examining the female tract for sperm after 10 days. Giant chromosome preparations were made by the acetic-lactic-orcein method (Strickberger 1962).

The very high sexual isolation between the two species is apparent from the table. Salivary chromosomes from hybrid larvae always show very poor pairing (Figure 1). However, five simple and one complex inversions have been detected in relation to the standard strain of D. tetrachaeta (Figure 2). The limits of the inversions in relation to the D. tetrachaeta map (Angus 1968) are IIA 9.0-11.3, IIIA 3.6-6.3, IIIB 11.0-chromocentre, IVA 3.5-4.9, IVB 14.6-chromocentre, VA 11.0-21.6. This last inversion is complex.

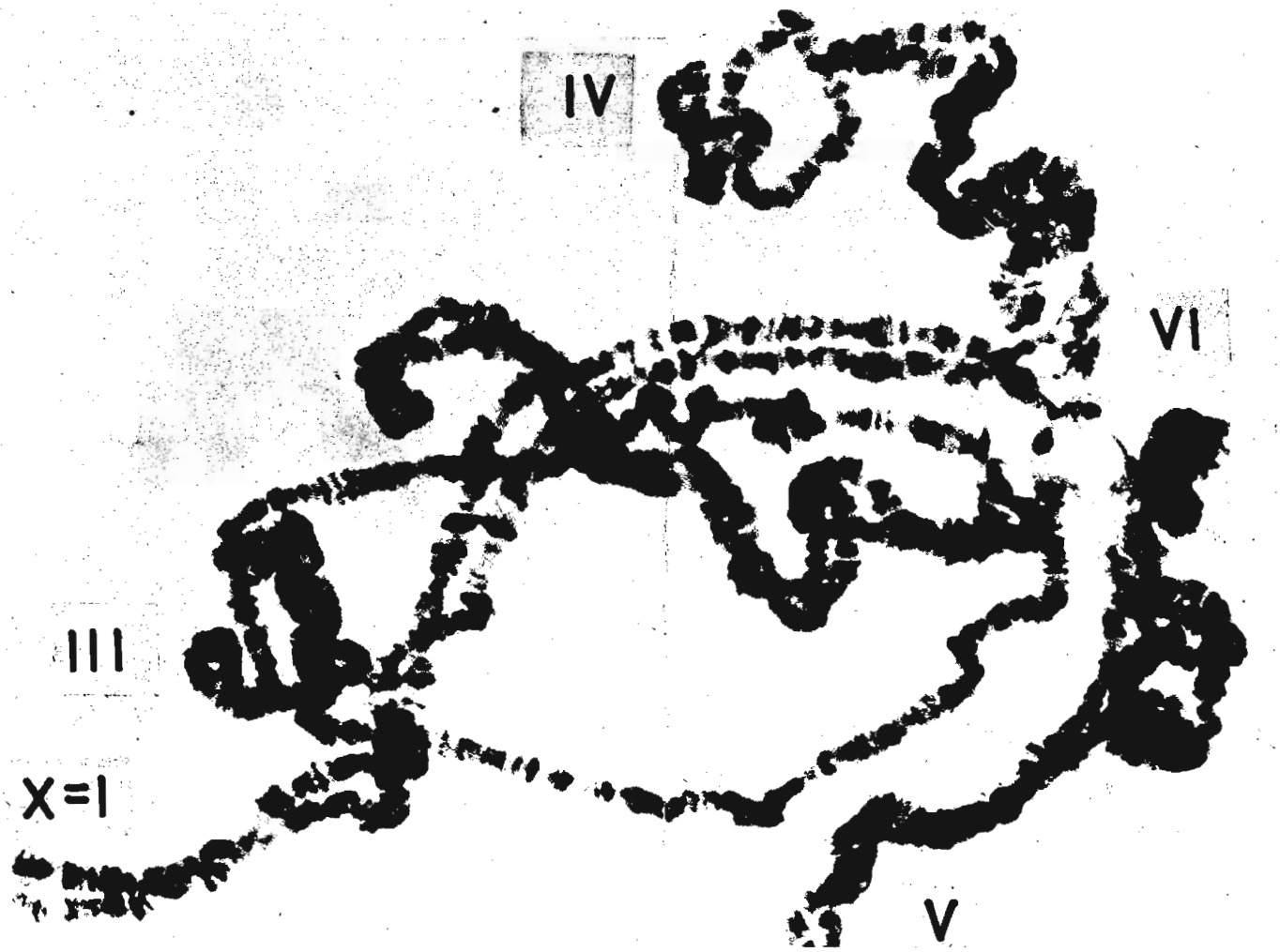


Figure 1

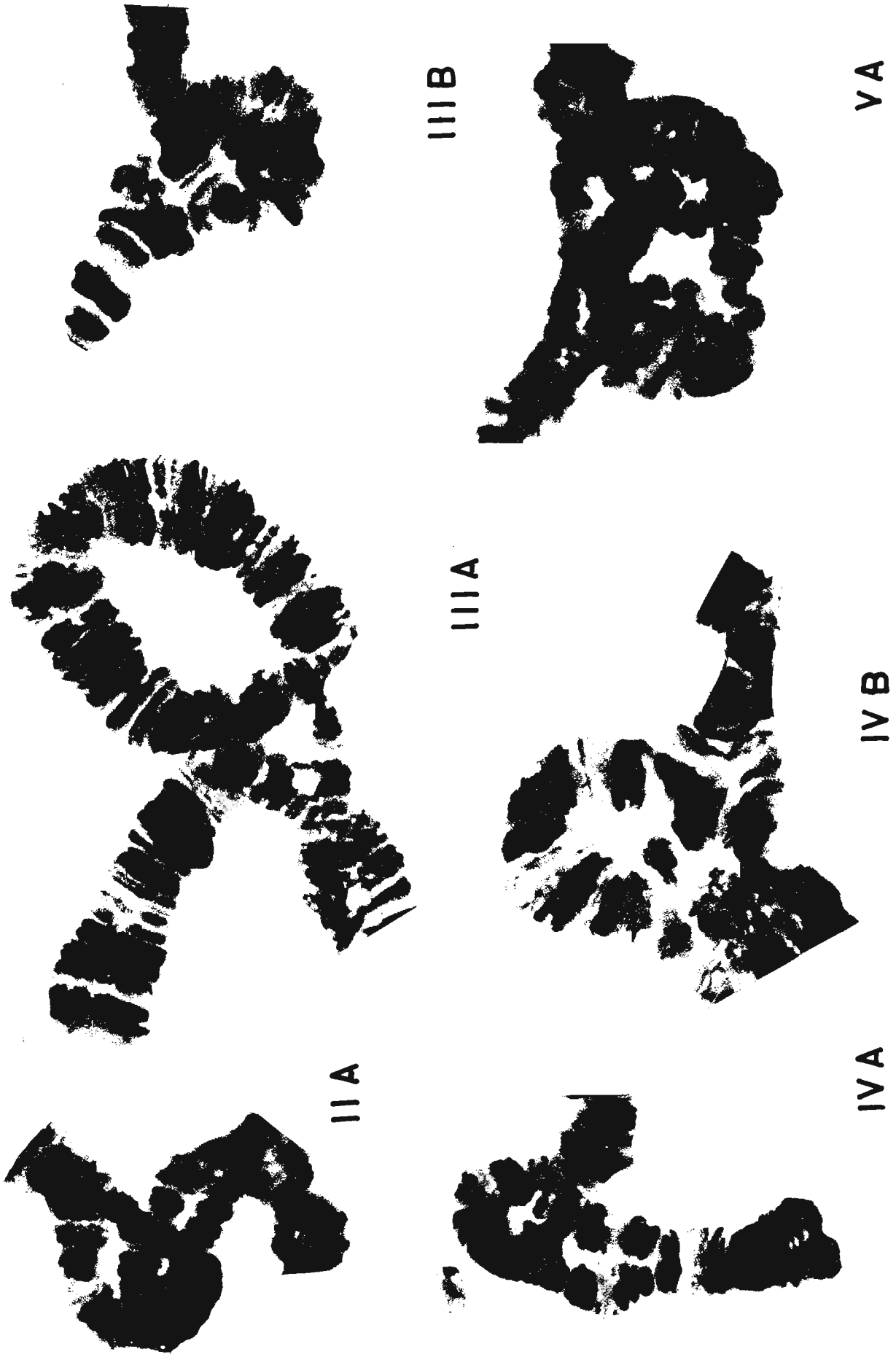


Figure 2

In the Australian region the situation found in the quadrilineata species group where these two cytologically differentiated sibling species have been detected contrasts with the situation in D. rubida where four geographical races have arisen by various isolating mechanisms and are characterised by different inversion patterns (Mather 1963, 1964, 1968 a and b).

SEXUAL ISOLATION TABLE

Females	Males	Females Tested	Number Insem.	% Insem.	Comment
D. pseudo.(Cairns)	D. tet.(Brown R.)	101	0	0	
D. tet.(Brown R.)	D. pseudo.(Cairns)	92	2	2	F_1 larvae
D. pseudo.(Brown R.)	D. tet.(Brown R.)	76	7	9	F_1 larvae
D. tet.(Brown R.)	D. pseudo.(Brown R.)	77	2	3	

Acknowledgement: This work was carried out under the supervision of Dr. Wharton B. Mather, Head of the Genetics Laboratory and arises out of a thesis for the Degree of Doctor of Philosophy in the University of Queensland.

References: Angus, D. 1967. Additions to the D. fauna of New Guinea. Pap. Dep. Zool. Univ. Qd, 3: 31-42. Angus, D. 1968. Chromosomal polymorphism in D. tetrachaeta. J. Hered. 59: 289-296. Mather, W.B. 1963. The races of D. rubida. Proc. XI Int. Congr. Genet., The Hague, 1: 161-162. Mather, W.B. 1964. Speciation in D. rubida. Evol. 18: 10-11. Mather, W.B. 1968(a). A third race of D. rubida. Pap. Dep. Zool. Univ. Qd, 3: 75-77. Mather, W.B. 1968(b). Evolution in D. rubida. Proc. XIIth Int. Congr. Genet. Tokyo. 1: 332. Strickberger, M.W. 1962. Experiments in Genetics with D. New York: John Wiley and Sons.

Faltus, F. and H. Oberlander. Brandeis University, Waltham, Massachusetts. Ecdysone induced differentiation of pulsating regions in genital imaginal disks after culture in vivo. (1)

Although the genital disks of D. melanogaster have been cultured for years in the abdomens of adult flies without differentiating, Nöthiger and Oberlander (2) have found that male genital disks from mature larvae regularly form pulsating regions after being cultured in young flies for two weeks. They showed that injected ring glands increased the percentage of disks which pulsate, and suggested that ecdysone was responsible. Since the ring gland is a composite gland it was necessary to test the effect of ecdysone directly.

The wild stock "sevelen" of D. melanogaster was used in these experiments as both donor and host. The animals were reared on standard food (maize, sugar, agar and yeast) at 25°C. Larval donors were used 117-120 hours after egg laying, and adult hosts were used one day after emergence.

Whole male genital disks were injected into adult flies and examined after two weeks. In one experiment one half of the adult hosts were injected with 6×10^{-4} ug of ecdysone (3) dissolved in 10% alcohol, while the controls were injected with an equal volume (0.003 ul) of 10% alcohol. Of 38 surviving experimental hosts 55% contained pulsating disks, while only 35% of 43 surviving controls did so. The difference between these two groups was significant within 90% confidence limits according to the binomial probability model.

A second experiment in which the experimented hosts received 6×10^{-4} ug ecdysone on days one and five resulted in the following: 88.5% of 26 surviving experimented hosts contained pulsating disks, but only 37.5% of 24 control hosts did so. This was significant within 99% confidence limits.

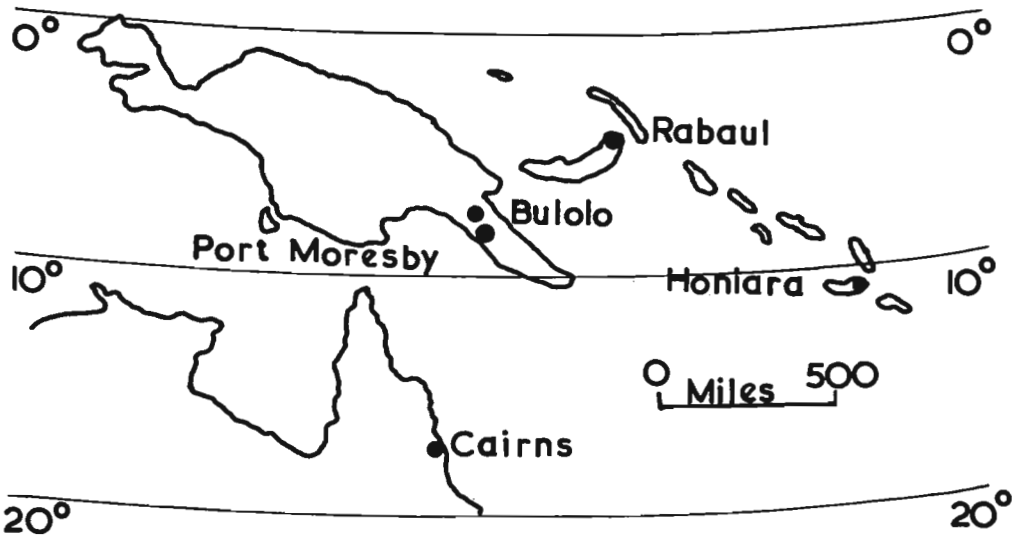
Presumably a single dose was less effective because of hormone inactivation. However, even the double dose of ecdysone was sufficiently low to support the conclusion that pulsating regions in cultured male genital disks differentiate in response to the action of residual ecdysone in the adult host. It is thus unnecessary to consider an ecdysone independent mechanism of differentiation to explain the origin of the pulsating regions.

(1) Supported by grant No. GB-7992 from the National Science Foundation. (2) Nöthiger, R. and Oberlander, H., 1967, J. Exp. Zool. 164: 61-68. (3) The ecdysone used in these experiments was generously provided by Dr. John Siddall (Zoecon Corporation).

Mather, W.B. University of Queensland, Brisbane, Australia. A fourth race of D. rubida.

It has previously been shown (Mather, 1964 and 1968) that the immigrans group species, D. rubida from Northern Queensland and Papua-New Guinea can be divided into three races on the basis of both chromosome inversion difference and sexual isolation. This paper is a report on a fourth race from Honiara, Solomon Islands and its relationship with races A, B and C.

As well as the stock from Honiara the same stocks from Rabaul, Bulolo and Cairns that were used in the 1964 and 1968 study have been employed (Map).



The methods used for sexual isolation tests (no choice) are given in Mather (1964). All flies used were aged for from 10 to 15 days. The cytological technique used follows Strickberger (1962) and the methods used in inversion analysis are given in Mather (1961).

In all crosses it was found that only very few offspring were produced. On the other hand except in the case of Honiara males x Bulolo females there is very little sexual isolation (Table). Thus Honiara flies are reproductively isolated from other strains by mechanisms other than sexual isolation. It will be noted that an F_1 was produced in all crosses except Honiara females x Bulolo males. In the cases where an F_1 was produced "gene flow" past the F_1 could be established by back-crossing to the original parents or proceeding to the F_2.

Sexual Isolation Tests - Honiara

	Females Tested	Number Insem.	% Insem.	F_1	F_2	R_2
Hon. ♂ x Rab. ♀	96	96	100	+	-	+
Hon. ♀ x Rab. ♂	94	93	99	+	-	+
Hon. ♂ x Bul. ♀	94	32	34	+	+	
Hon. ♀ x Bul. ♂	73	69	95	-		
Hon. ♂ x Cai. ♀	93	93	100	+	-	+
Hon. ♀ x Cai. ♂	89	89	100	+	-	+

Because of the heavy reproductive isolation between Honiara and Cairns the inversion picture could not be obtained by mating Honiara flies to the standard Cairns strain. However, ninety pair matings of Honiara flies showed no heterozygous inversions. Further a single larva from a female Honiara fly x male Cairns fly showed the IIRE and IILA inversions only, thus establishing that Honiara flies are homozygous for IIRE and IILA.

Whereas races A, B and C of D. rubida are separated by strong sexual isolation (Mather 1964, 1968), Honiara flies appear to be separated from other races by reproductive isolating mechanisms other than sexual isolation. Cytologically the unique feature of the Honiara strain is being homozygous for IILA. Homozygosity of IIRE also occurs in Race B from Rabaul

and Race B is heterozygous for IIID.

Thus strong non-sexual reproductive isolation between a strain of D. rubida from Honiara, Solomon Islands, and the three established races of D. rubida together with a unique inversion pattern justifies the designation of a fourth race of D. rubida.

Literature Cited: Mather, W.B. 1961. Chromosomal polymorphism in D. rubida Mather. Genetics Princeton 46: 799-810. Mather, W.B. 1964. Speciation in D. rubida. Evolution Lancaster Pa. 18: 10-11. Mather, W.B. 1968. A third race of D. rubida. Pap. Dep. Zool. Univ. Qd. 3: 75-77. Strickberger, M.W. 1962. Experiments in Genetics with Drosophila. Wiley, London.

Lifschytz, E.* and Falk, R. Hebrew University, Jerusalem, Israel. Some further studies of reversion at the K-pn locus.

A.1. An attempt was made to obtain a dose curve for induced reversions of K-pn (RK's) using X-ray in mature sperm. Preliminary results are given in Table I. Details of the experimental procedures are given in Lifschytz and Falk, Genetics, 1969. The number of females/culture indicates larval density. Each female represents ca. 200 tested zygotes or 400 hatched larvae. At the bottom of the table the averaged result of E.M.S. treatment is given.

Table I

Dose	Female Culture	Replicates	Total Females	No. Revertants	Revertants/Recovered Females
500	4	2	1,020	3	1/340
1,000	4.1	3	924	5	1/185
2,000	3.7	4	1,647	13	1/196
3,000	2.0	2	852	15	1/57
4,000	1.7	3	488	10	1/49
Control					1/3000
			E.M.S.		
0.2%	1.54	2	593	15	1/40

2. The conclusions one can draw are:

a. The induction of RK's mutant (recessive lethals, presumably small deficiencies) follows one hit kinetics.

b. The efficiency of E.M.S. in inducing RK mutation, as compared to the efficiency of X-ray, is 20%. This conclusion is based on the fact that with the same E.M.S. treatment and with the same flies, 48% recessive lethals are induced on the X-chromosome. By extrapolation from the known dose-effect relations for X-ray induced sex-linked-recessive lethals, it is possible to estimate that a dose of X-rays that would produce 48% lethals would produce one RK mutant per 10 females. Moreover, this is an underestimate since with 48% lethals at least one-third of the chromosome carries two lethals.

Assuming that RK's are deficiencies, and X-ray induced lethals are mostly deficiencies, one can hopefully use this system for estimation of the point mutation/deficiencies ratio following different mutagenic treatments.

B. Apart from being all recessive lethals and allelic to each other, about 30 RK mutants both from X-ray and E.M.S. were tested for 2;3 translocation or gross inversions. Surprisingly enough none of them was associated with a translocation or an inversion. The implication of this finding will be discussed elsewhere.

In agreement with previous findings, 15 pairwise combinations of different RK's (hence recessive lethals) that were tested for complementation of the K-pn effect turned out to be noncomplementing.

This has been done using free duplication (Falk and Shamai) for the K-pn gene, thus enabling us to test whether the genotype

$$pn/Y;\frac{RK^1}{RK^2},\ Dp(3;f)ca^+bv^+K\text{-}pn^+$$

is lethal. Up to now none of the $RK^1/RK^2/K\text{-}pn^+$ combinations regain the K-pn/K-pn$^+$ interaction with pn.

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Ytterborn, K.H. University of Stockholm, Sweden. Homozygous lethal effects of II-III translocations in D. melanogaster.

One to two day old wild type males from an inbred stock (KÄS-I) were X-rayed with three different doses. They were immediately after mated for 8 hours in order to determine the frequency of translocations induced in mature spermatozoa. The test stock was cinnabar;brown;ebony ($cn;bw;e^{11}$) and translocations including the Y and/or II and/or III chromosomes were scored in F_2. The results of the translocation tests are shown in Table 1.

Table 1. Translocations induced in spermatozoa by three different doses of X-rays.

Dose in r	Tests	Translocations between II & III		Translocations between Y & II &/or III	
		Number	Percent	Number	Percent
500	813	10	1.2	2	.2
2000	538	35	6.5	21	3.9
3500	390	58	14.9	28	7.2

The data agree with other studies on irradiation induced translocations in that the rate of translocations increases approximately by the 1.5 power of the increase in dose.

Most of the II-III translocations obtained were tested for their homozygous effect on the viability. Males heterozygous for the translocation and the chromosomes containing the recessive markers were mated to females from the stock In(2L + 2R)Cy, Cy/T(2;3)ap^{Xa}/TM2, Ubx^{130} e^{s}. In the progeny males and females heterozygous for the translocation to be tested and the Cy and TM2 chromosomes were collected and mated to each other. If the progeny of such a mating consisted of at least 25 animals phenotypically Cy Ubx and no others, the translocation was regarded as homozygous lethal. The results of the lethal tests of the translocations are shown in Table 2.

Table 2. The occurrence of homozygous lethal II-III translocations among translocations induced by the different doses.

Series	Tested translocations	Homozygous lethal translocations	
		Number	Percent
500r	9	2	22
2000r	35	23	66
3500r	54	46	85

The frequency of translocations associated with recessive lethal factors increases with increase in dose. In Table 3 are shown the results of pairwise two-tailed statistical comparisons between the different series. Though one of the comparisons is not statistically significant, the result may be interpreted to mean that recessive lethal effects in the chromosomes constituting the translocations is influenced by the dose.

Table 3. Comparisons of the frequencies of homozygous lethal II-III translocations in the three series.

Compared series	Statistical test	Probability
500r - 2000r	Exact method	P=0.05
500r - 3500r	Exact method	P<0.001
2000r - 3500r	χ^2_c	0.10>P>0.05

The present results are at variance with the information usually given in this connection, according to which most II-III translocations are homozygous lethal. This effect has been referred to the appearance of recessive lethals or deletions at the break points of the translocation. However, at irradiation there will be induced recessive lethals, point mutations or deletions, in the second and the third chromosomes independently of the breaks giving rise to the translocations. Very roughly the frequency of these independently induced lethals in spermatozoa should be approximately one percent per 100 r X rays in the two chromosomes together. Therefore, it is concluded from the present results that only some of the induced II-III translocations are

homozygous lethal because of lethals or deletions at the break points, the rest being homozygous lethal because of mutations appearing independently of the rearrangements.

Hanks*, G.D. and D.J. Remondini**, Indiana University Northwest, Gary, Indiana*, and Gonzaga University, Spokane, Washington**.

General inverse relationship between percentage of females and progeny number.

Males were taken from natural populations (provided by Bruce Wallace except YO_1) and crossed to RD females. Progeny males were then backcrossed for a total of 7 to 9 crosses to RD females. Males were then tested extensively by mating to 5 al, ru females. This gives a test of the population Y in virtually a complete RD background. The summarized data are given in Table 1 and Figure 1. It seems clear that with a single exception (one run of BV8) the greater the mean percentage of females, the lower the mean progeny number. This is consistent with the proposed mechanism in RD -- the loss of spermatids receiving a broken Y chromosome during meiosis (Erickson, 1965; Hanks, 1964). The single exception ($p<.05$ for progeny number) means that the high percentage of females and the lowered progeny number are not necessarily associated. Maybe the RD mechanism in spermatogenesis is the same in the exceptional line but some component surrounding fertilization or sperm storage is different or else the meiotic mechanism is different but still giving a high percentage of females. It should be interesting to find out. There is of course a slight possibility that during backcrossing to RD females that an RD Y chromosome was substituted for the natural population Y chromosome. Even if this did occur in one or two cases it would not change the general result nor explain the one exception. It is noteworthy that a number of population Y chromosomes do have significant RD activity.

Table 1. Pooled results of testing single males (10 to 17) extensively by mating to five al, ru females. The mean percentage of females given is unweighted.

Stock source	Sterile cultures	Total progeny	$\bar{X}$ ♀%	$\bar{X}$ No. of progeny per ♂
Barcelona, Spain (BS_8)	3	5,477	54.5	548
Blacksberg, Virginia (BV_8)	2	5,789	64.2	482
	1	8,796	66.2	800
Capetown, Africa (CA_7)	0	8,776	59.0	585
Controls	1	8,153	66.6	480
	0	6,373	67.4	425
	1	5,049	68.5	459
Quiryath, Israel (QI_{11})	2	9,830	53.3	819
Riverside, California (RC_9)	2	7,051	59.8	588
	0	5,812	64.0	484
Santiago, Chile (SC_{11})	1	6,265	63.0	448
Syosset, New York (SY_6)	0	6,519	65.7	543
Yoncalla, Oregon (YO_1)	1	7,128	63.0	548

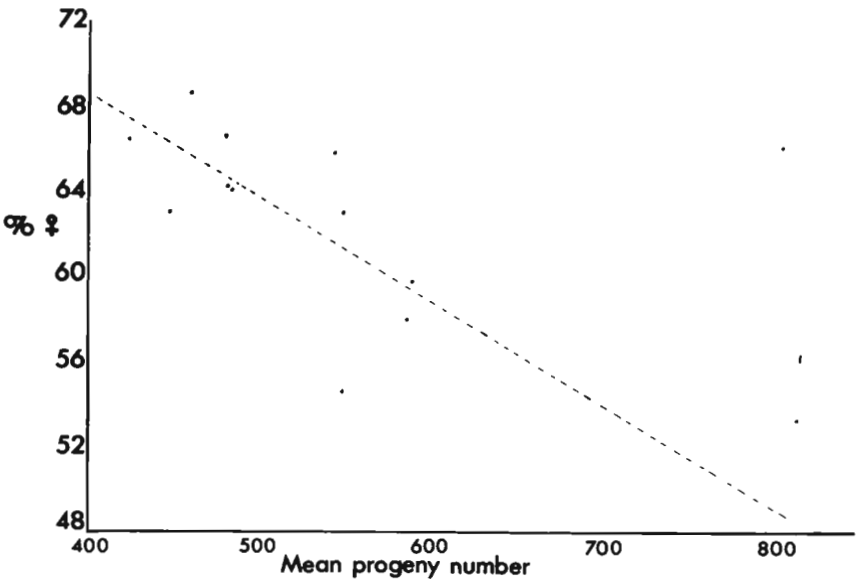

Fig. 1. Data from Table 1 is plotted in graph form. It shows generally an inverse relationship between percentage of females and mean progeny number.

Hall, J.C. University of Washington, Seattle, Washington. Non-independence of primary non-disjunction for the sex and fourth chromosomes in D. melanogaster.

Rates of primary non-disjunction in males and females are virtually always reported for the sex chromosomes only. However, if the flies being tested have marked 4th chromosomes - and if the flies to which they are crossed carry marked attached-4th chromosomes - then it is possible to measure simultaneously rates of primary non-disjunction for the sex chromosomes and the 4th chromosomes. This has been done recently in several different experiments, in some of which there is heterozygosity for one or more inversions in the females or males being tested. In the majority of cases, and for all experiments summed, it is observed (Table 1, Table 2) that the frequency of double exceptions is much greater than what one would

Table 1. Primary non-disjunction in females; marked females with and without heterozygosity for inversions were crossed to $\overline{XY}$, vfB/0; RM(4), ci ey^R/0 males.

Inversions present	Single exceptions		Double exceptions		Total	Source
	X	4	obs.	exp.		
1) none	6	5	2	.002	14,099	Sandler et al (1968)
2) none	4	3	0	.001	13,419	" " "
3) none	14	27	2	.006	6,316	Davis, B.K.
4) none	3	14	0	.004	11,904	" "
5) none	1	2	1	.0002	8,462	Hall, J.C.
6) d1-49	26	16	11	.025	16,496	" "
7) SM1	14	10	1	.009	15,262	Sandler et al (1968)
8) SM1	5	8	2	.009	4,377	" " "
9) SM1	5	20	5	.013	7,751	Hall, J.C.
10) TM2	9	12	1	.007	16,020	Sandler,et al (1968)
11) TM2	4	14	0	.010	5,724	" " "
12) SM1; TM2	1	8	0	.001	16,017	" " "
13) SM1; TM2	3	12	0	.007	5,470	" " "
Totals	95	151	25	.101	141,317	

Table 2. Primary non-disjunction in males; marked males with and without heterozygosity for inversions were crossed to y pn; RM(4), ci ey^R/0 females.

Inversions present	Single exceptions		Double exceptions		Total	Source
	sex	4	obs.	exp.		
1) none	26	7	2	.017	10,600	Sandler et al (1968)
2) none	9	5	1	.007	6,312	Davis, B.K.
3) none	9	10	0	.017	5,267	"
4) SM1	50	7	0	.021	16,345	Sandler et al (1968)
5) TM2	19	8	1	.011	13,671	" " "
6) SM1; TM2	25	11	2	.017	15,992	" " "
Totals	138	48	6	.097	68,187	

expect if the sex chromosomes and the 4's were non-disjoining independently (i.e. the product of the two frequencies of single exceptions). The excess of double exceptions seen here does not result from non-homologous pairing, since it is observed in males and because, for the females, there is among the double exceptions no preponderance of the nullo-X, double-4 or double-X, nullo-4 classes. The high coincidence could result from a situation in which the sex and 4th chromosomes are in fact non-disjoining independently, but only in a small fraction of meiotic cells in which meiosis goes awry such that, for example, the sex chromosomes and the 4's move to the poles at random at anaphase I. It should also be noted that there is a rather high degree of variability among experiments.

Reference: Sandler, L. et al., Genetics, 60: 525-558.

U, R. Duke University School of Medicine, Durham, North Carolina.

Miracil-D: Inhibitor of Ribonucleic acid synthesis and chromosome loss in Drosophila male germ cells.

The effect of Miracil-D (1-diethylaminoethylamino-4-methyl-10-thia-xanthenone), a profound inhibitor of RNA synthesis(1,2), on the production of chromosome loss due to breakage has been investigated. The structural features of this chemical compound are similar (dialkylaminoalkylamino side chain attached to heterocyclic ring system) to those of acridine and actinomycin D which are known to interact with DNA(3,4). The evaluation of genetic damage in this investigation was by the XO method. D. melanogaster males carrying a ring-shaped X-chromosome ($X^{c2}y$ B/Y sc^8 y^+), 4 to 6 hours old, were collected and food withheld for 18 hours. These males were then given Miracil-D (1 mg/ml of regular Drosophila food) for 24 hours prior to mating with 3 day old y w f virgin females. The data on table 1 shows the effect of this chemical treatment. The brood 1 represents those males mated to y w f virgin females for 48 hours continuously. Broods 2 through 7

Table 1. Effect of Miracil-D by feeding and the spontaneous rate of chromosome loss. XO males and mosaics.

Brood		No. of Gametes tested	No. of XO males & mosaics	Percent of XO males & mosaics	Chi-square	Probability
1	Miracil-D	2329	39	1.68		
	Control	2300	26	1.13		
2	Miracil-D	2311	29	1.26		
	Control	2140	30	1.40		
3	Miracil-D	2091	25	1.20		
	Control	2489	27	1.09		
4	Miracil-D	1859	23	1.78		
	Control	2189	21	0.96		
5	Miracil-D	1305	15	1.15	$\chi^2_c = 5.083$	< 0.03
	Control	1193	6	0.50		
6	Miracil-D	1315	23	1.75	$\chi^2 = 2.397$	
	Control	1781	14	0.79		
7	Miracil-D	1480	28	1.89	$\chi^2 = 5.941$	< 0.002
	Control	1498	23	1.54		
Total	Miracil-D	12690	192	1.51	$\chi^2 = 9.588$	< 0.002
	Control	13590	147	1.08		
Total 4 - 7 broods only	Miracil-D	5959	99	1.66	$\chi^2 = 12.107$	< 0.0005
	Control	6661	64	0.96		

Chemical concentration: 1 mg/ml of regular Drosophila food.
Control : regular Drosophila food.

represent consecutive 24 hours re-matings to y w f virgin females. The overall total of these broods (1 through 7) shows about 40 percent increase of XO males and mosaic individuals (due to chromosome breakage and subsequent loss) compared to those in the control group. The data reveals a significant difference between treated and control group (Chi-square of 9.588 with a probability of less than 0.002). In order to calculate those cells affected most prominently, the data on broods 5, 6 and 7 were added. A statistical analysis by 2 x 2 contingency table showed a Chi-square of 12.102 with probability being less than 0.0005. For males

mated daily (or every other day), the first appearance of induced crossing-over which can occur prior to meiosis is in the 7-9 day broods (5). Therefore, the broods 5, 6 and 7 in these experimental series represent those cells affected during the early spermatid and meiotic stages.

The relation of concentration of this chemical in food to incidence of chromosome breaks, in the most sensitive stages, is shown in figure 1. There were three control groups. Control

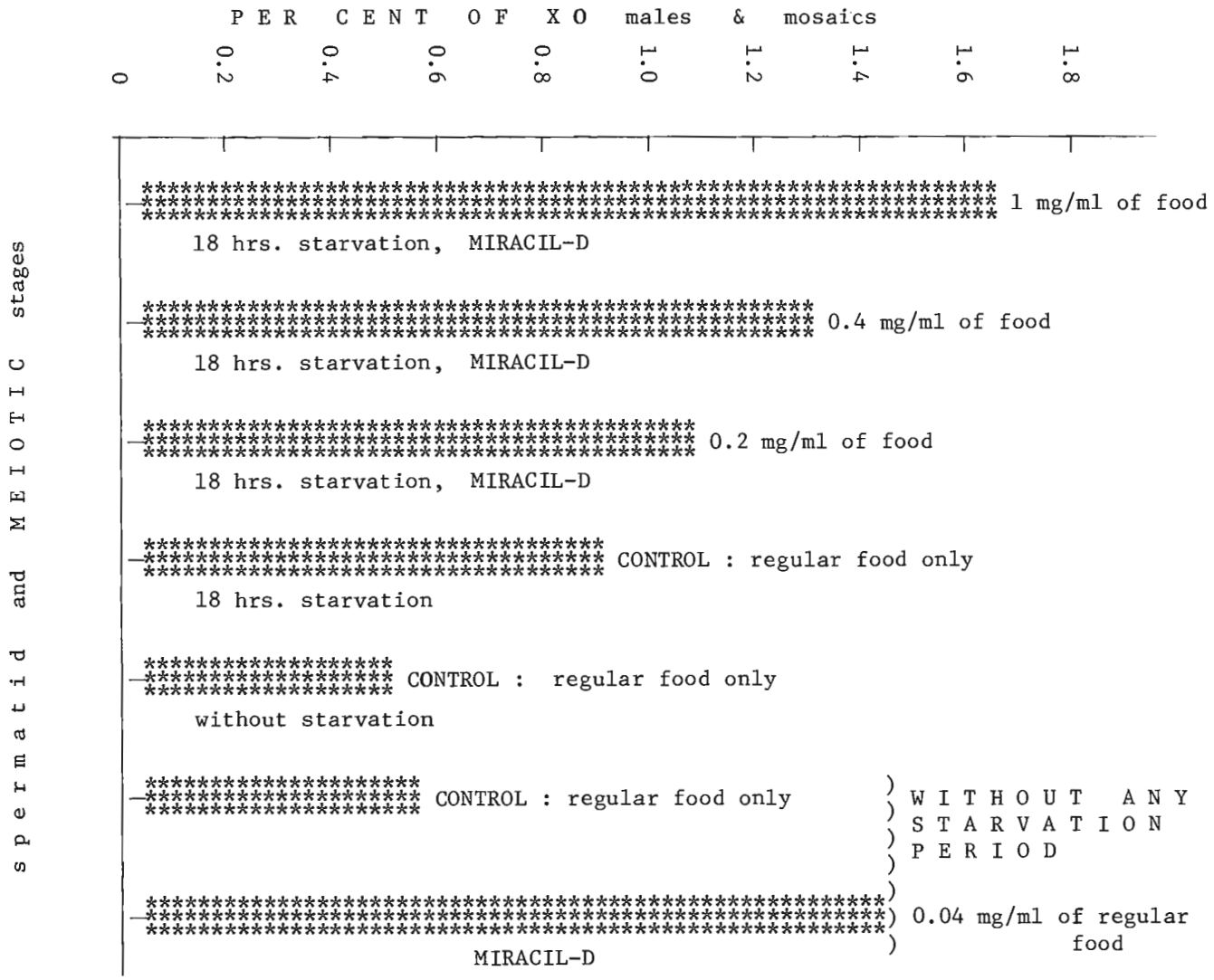

Figure 1. A summary of the various concentration of Miracil-D treatment and the frequency of chromosome loss.

group two had only regular food diet, while the third control group had an 18 hour starvation period prior to returning to the regular food. As seen in figure 1, starvation alone gave some increase in chromosome breaks. Surprisingly, feeding a concentration of 0.04 mg/ml of regular Drosophila food for 24 hours without any starvation period revealed more chromosome breaks than doses five and ten times greater. This may be explained by death of XO males and mosaic individuals from drug toxicity plus starvation in the group receiving higher doses.

These results are similar to those obtained from X-irradiation and from specific inhibitors of DNA synthesis such as mitomycin C(6,7). However, DNA-mediated RNA inhibitor, actinomycin D reduced the frequency of sex-linked recessive lethal mutations in Drosophila(8).

References: 1) Weinstein, B., et al. 1965, Mol. Pharmacol 1: 297. 2) U, R. (unpublished) work with mammalian cells in vitro. 3) Lerman, L.S. 1964, J. Cell. Comp. Physiol. 64:

suppl. 1: 1-18. 4) Reich, E. 1964, Science 143: 684-689. 5) Mittler, S., et al. 1966, Science 152: 1087-1088. 6) Shiba, S., et al. 1959, Nature 183: 1056-1057. 7) Mukherjee, R. 1965, Genetics 51: 947-951. 8) Burdette, W.J. 1961, Science 133: 40.

This work was supported by Research Grant No. 363-0428 from Duke Endowment Fund, and experiments were conducted at Radiation Therapy Research Unit, Director, Prof. J.C. Evans, U.S. Veterans Administration Hospital, Durham, N.C. 27706.

Gvozdev, V.A., V.J. Birstein and L.Z. Faizullin. Kurchatov Institute of Atomic Energy, Moscow, U.S.S.R. Gene dependent regulation of 6-phosphogluconate dehydrogenase activity of D. melanogaster.

The structural locus Pgd for the 6-phosphogluconate dehydrogenase (PGD) of D. melanogaster has been located on the X-chromosome at 0.64 between the broad (0.6) and prune (0.8).

The variation of Pgd dose from 1 to 2 results in the proportional increase of PGD activity showing the absence of the feed-back regulation. The increase of Pgd dose using $w^{+}Y$ and $Dp(1;3)w^{vco}$ duplications (thrice as much for males and twice as much for females) resulted in 2-3- or 1.5-2.0-fold increase of PGD specific activity in males and females respectively. The PGD activity of normal males and females is twice as much as that of the $Df(1)w^{vco}/+$ and Df(1)Pgd-pn/+ females with a single dose of Pgd.

The quantitative determination of PGD activity in the flies with different doses of Pgd^{A} and Pgd^{A}/Pgd^{B} heterozygotes of either sex show that the gene activity of both alleles in males was twice as much as that of females.

PGD activity in females hyperploid for the distal pieces of X-chromosome (1-3C, 1-9A and 1-9B) including Pgd locus increases for 1.4-1.5 times as compared to that of normal females. Introduction of the 16A1-20 fragment has no effect on PGD activity while 9B-20 and 9E-13C reduces it to 80% level. These results are in accord with Muller's views on the presence of X-linked dosage compensators with negative action.

Chen, P.S. and R. Bühler. Zoologisches Institut der Universität, Zürich, Switzerland. Further studies of the paragonial substance in D. melanogaster.

In our previous study (Chen and Diem. J. Insect Physiol., 7: 289-298, 1961) we located a peptide in the accessory glands (paragonia) of Drosophila male adults. Judging from its mobility on paper chromatogram and amino acid composition it corresponds obviously to the sex peptide found by Fox (Science 129: 1489-1490, 1959). Transplantation of male genital discs into female larvae demonstrated that the synthesis of this peptide is autonomous. This has been confirmed by the recent study of Smith and Bischoff (D.I.S. 44: 122) using the mutant "doublesex". The work done by Garcia-Bellido (Z. Naturf. 19b: 491-495, 1964) showed that grafting of the glands or injection of the paragonial fluid into virgin females resulted in a distinct increase in oviposition. The same results have been reported by Leahy and Lowe (Life Sciences 6: 151-156, 1967). In an attempt to answer the question if the paragonial substance or sex peptide is really the active principle for stimulating egg deposition, methanol extracts were prepared from a large number of male adults and analysed by ion-exchange chromatography. We found that on the amino acid analyzer this peptide was eluted as an acidic component in the region between phosphoserine and glycerophosphoethanolamine. This has been confirmed by fractionation of extracts from a total of 1070 pairs of accessory glands dissected out individually from 8-day-old adult males. On the analyzer the sex peptide appeared as the only prominent peak in the same position revealed by using extracts from whole flies. Injection of the peptide isolated from the column and desalted by high voltage elecrophoresis into virgin females resulted in a two- to threefold increase of oviposition. Our hitherto observation suggested that a single injection is sufficient for the whole adult life. The biosynthesis and turnover of the sex peptide are now under investigation.

Bakula, M. Saint Louis University, Missouri. Beta-galactosidase activity in axenic and nonaxenic adults of D. melanogaster.

Beta-galactosidase activity of adult flies was measured by a method modified from Sellinger et al (1) using 5mM o-Nitrophenyl-β-D-galactoside (Sigma) as the substrate. In preliminary experiments (Figure I) the optimal buffer and pH were determined. Citrate-phosphate buffer (ionic strength 0.05) at pH 5.6 was chosen for all subsequent experiments on the basis of these results. An adult homogenate prepared as follows was used as the enzyme source. A number of flies sufficient to give a final concentration of 5 flies/0.5ml (minimum number necessary for a detectable reaction) were hand homogenized in cold 0.25M sucrose with added Triton X-100 (0.01%)(Rohm and Haas). Since beta-galactosidase is typically a lysosomal

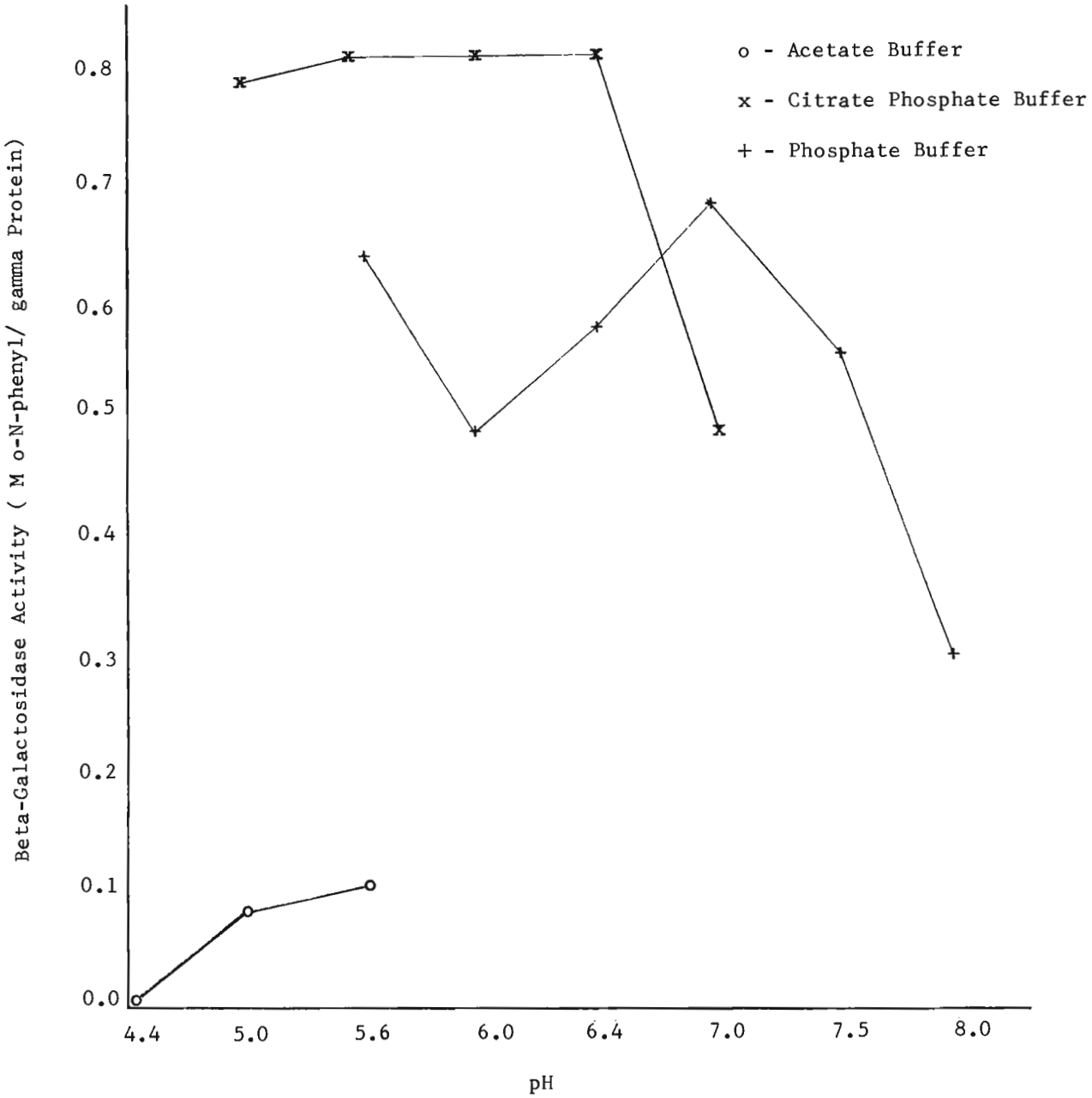

Figure I. Beta-galactosidase activity per gamma protein in the pH range 4.4-8.0. Three buffers (ionic strength 0.05) were employed as indicated. All reactions were carried out at 25°C for 2 hours.

enzyme in other animals the Triton X-100 was used to rupture the lysosomal membranes. The homogenate was centrifuged at high speed in a clinical centrifuge, the precipitate rehomogenized and centrifuged as before. After the final centrifugation the supernatants were combined, their volumes adjusted, and 0.5ml aliquots were pipetted into tubes containing 1.25ml buffer and 0.5ml substrate. The reaction was allowed to proceed for 2 hours at 25°C, and was stopped by plunging the tubes into an ice bath. The amount of o-nitrophenol liberated by the enzyme was measured colorimetrically at 420mu immediately after adding 0.5ml 1M NaOH to each tube. Protein determinations were performed according to the method of Lowry (2). The results of the assay were expressed as uM o-nitrophenol per gamma protein.

The enzyme determinations were run on non-axenic live yeast fed adults (P_1) and on 2 successive axenic generations of adults (P_2 and P_3) raised on sterile medium containing 0.5% Brewer's yeast, 1.5% agar and either 0.8% sucrose or 0.8% lactose. All tests were made on flies 2 to 5 days of age. In Table I the beta-galactosidase activities of the axenic lactose

Table I. Beta-galactosidase Activity of Non-Axenic and Axenic D. melanogaster Adults.

	Non-Axenic		Axenic				
			Lactose Fed		Sucrose Fed		
Generation	No. of Tests	Mean uM o-N-phenyl/ gamma protein	No. of Tests	Mean uM o-N-phenyl/ gamma protein	No. of Tests	Mean uM o-N-phenyl/ gamma protein	t
P_1	3	0.336					
P_2			4	0.14	4	0.12	o.490
P_3			5	0.73	5	0.47	4.19*

* Significant at 5% level

and sucrose raised are compared to each other. These results are not compared to the P_1 generation since it is probable that bacteris may be biasing the result by contributing to the total enzyme activity.

Beta-galactosidase levels were highest in the P_3 adults after an initial, though non-significant decrease. It appears that the lactose fed flies have the greatest enzyme activity.

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(This investigation was supported by PHS Training Grant No. GM00989 while the author was a postdoctoral trainee in the Department of Zoology, University of Michigan.)

Schalet, A., G. Lefevre and K. Singer. University of Connecticut, Storrs, Connecticut; San Fernando Valley State College, Northridge, California. Preliminary cytogenetic observations on the proximal euchromatic region of the X chromosome of D. melanogaster.

We have undertaken a cytogenetic investigation of deficiencies located in the proximal region of the X chromosome covered by the $y^+Y\ mal^+$ chromosome (Schalet and Finnerty, DIS 43: 128). Salivary analysis based upon at least 12 deficiencies of independent origin permits the following preliminary observations.

1) Cytological extent of the proximal X covered by $y^+Y\ mal^+$: From a left breakpoint in 18F through sections 19 and 20.

2) Location of visible loci: ot, 19A3-6; sw and mel, 19B3-19C2; mal, 19C4-19D3; lf, 19E5-6; unc, 19F1-2; su(f), to the right of 20A2 (probably to the right of 20A). The "mal" locus of Lifschytz and Falk, (see note of Schalet and Finnerty in this issue), defined by the overlapping deletions A118/Q539, 19E7 or immediately next to it.

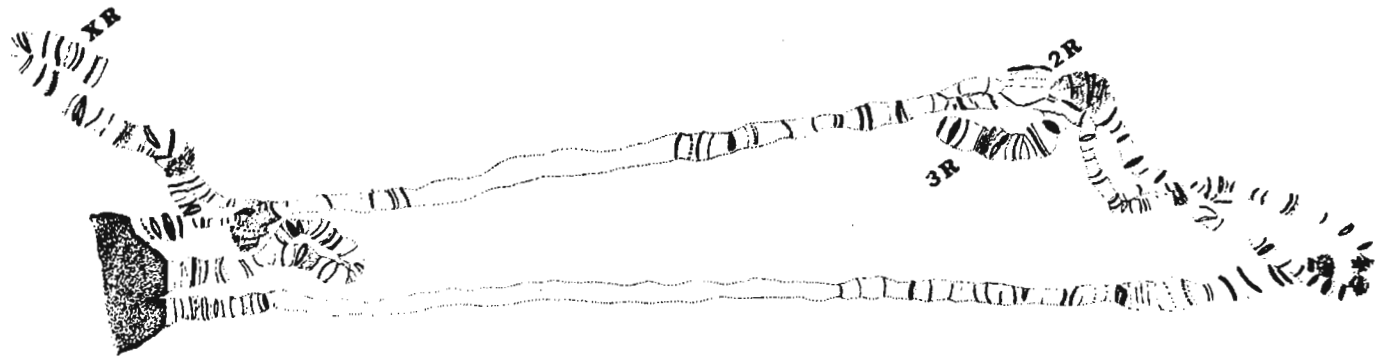
3) Lethal loci in section 20: Lethal A7 has been localized to 20A1-2. Complementation tests have demonstrated at least 7 lethal loci between lethal A7 and su(f). Consequently, these 7 lethals and su(f) are located within bands generally considered to be truly chromocentral. Since su(f) is to the left of the proximal breakpoint of the sc^4 inversion, these results are in conflict with Cooper's assignment of 19F for that breakpoint.

Sajjan, S. Nirmala and N.B. Krishnamurthy. University of Mysore, Manasagangotri, Mysore, India. Report on two new translocations in a natural population of D. ananassae from Hiriyur (Mysore State, India).

Translocations are of very rare occurrence in natural populations of Drosophila, although certain special kinds of translocations, called centric fusions, have played an important role in the phylogeny of a large number of species. Patterson, Stone, Bedichek & Suche (1934) have pointed out that many mutual translocations lead to inviability or infertility in homozygous condition due to some kind of position effect in Drosophila. Further, they have stated that the impaired fertility of heterozygotes confers strong negative selection pressure on the survival of these translocations.

There are a total of five translocations reported in the literature in D. ananassae (Kikkawa 1937, 1938, Kaufmann 1936b, 1937; Dobzhansky & Dreyfus 1943; Freire-Maia 1961; Ray-Chaudhuri & Jha 1965; Futch 1966). Of these, one (Kikkawa 1937, 1938 & Kaufmann 1936b, 1937) was shown to be karyotypically fixed, involving the translocation of basal region of the X-chromosome to chromosome number four. The other four translocations (probably floating types) have been reported between 2L and 3L in Brazilian population by Dobzhansky and Dreyfus (1943), between 3R and 2R from Uberlandia, Minas Gerais population of Brazil by Freire-Maia (1961), between 3L and 4 in Mughalsarai population of North India by Ray-Chaudhuri and Jha (1965), and between XL and 2R in one larva of Niue Island population by Futch (1966).

The two new translocations reported in this article were observed in a single larva obtained from a naturally inseminated female collected from Hiriyur of Mysore State, India. Both of these translocations are reciprocal heterozygous translocations, one involving the basal portion of the right arm of the second chromosome and the right arm of the X-chromosome and the other is between the terminal portion of the right arm of the second chromosome and the right arm of the third chromosome. This is a unique case where one arm of the second chromosome has participated in the formation of translocation heterozygote with two arms of two different chromosomes, namely XR and 3R (Fig. 1). Both of these translocations are dif-



ferent from those described earlier. At this stage, it is risky to draw any conclusion because of the rarity of these new translocations. However, the presence of these suggests that this cosmopolitan species is experimenting on its own Karyotype by instituting novel translocations. It may be that these are floating ones; none the less, these two translocations are reported for the first time in this species.

Acknowledgements: We are highly grateful to Dr. M.R. Rajasekarasetty, Prof. and Head of the Department of Zoology, University of Mysore, Manasagangotri, Mysore, for his constant help and encouragement. This work is supported by the Department of Atomic Energy, Government of India.

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Oshima, C. National Institute of Genetics, Misima, Japan. Frequencies of intra- and interpopulational allelisms of lethals in the several natural populations and the relationship between the dispersal of flies and the frequency of allelism of lethals in a natural population.

In late October of 1967, a particular collection of D. melanogaster was carried out simultaneously at four sites in Katsunuma and one site in Kofu, both were located in the central part of Japan. The distances between four sites, A, B, C and D, were relatively short, from 430 to 950 meters, and the distance between any of them and site E in Kofu was about 14 kilometers. The allelism test was performed by intercrossing 112 Cy/lethal balanced strains, whose lethal chromosomes were extracted from the above 5 natural populations. From the results of a total of 6,216 crosses, the frequencies of intra- and interpopulational allelisms were obtained as shown in Table 1.

Table 1. Frequencies of intrapopulational and interpopulational allelisms of the lethal chromosomes.

Locality	Population	No. of lethal chromosomes	Frequency of intrapopulational allelism	Population	Distance between populations	Frequency of interpopulational allelism
	A	34	5.35%	A-B	430m	5.34%
	B	27	3.70	B-C	450	4.40
Katsu-	C	16	2.50	C-D	450	5.00
numa	D	25	13.33	B-D	630	6.81
				A-C	880	5.70
				A-D	950	8.82
		Mean	6.22%		632m	6.01%
	E	10	6.67	E-A	14,000m	6.47
Kofu				E-B	14,000	7.04
				E-C	14,000	3.75
				E-D	14,000	9.60
Total		112		Mean	14,000	6.72%

Overall frequency of allelism = 6.44%

The overall frequency of allelism (6.44%) was the highest among those obtained for the past five years, and the similar frequencies found in the intra- and interpopulational allelisms may be attributed to high frequent and widespread lethals. Most of them appeared to have been lethals persisting for a long time in the natural populations. Either such lethals were transplanted from one population to another by recurrent migration or have already persisted independently, in those populations examined, though both possibilities are not necessarily mutually exclusive.

At the beginning of October 1968, Drosophila were collected in Katsunuma by 10 traps containing a mixture of banana and yeast. These traps were put almost linearly at intervals of 30 meters from the first one which was set up in a large natural population of D. melanogaster. All flies in these traps were caught by a net four times for the 24 hours, but only a certain number of flies were sampled from the first trap. Flies totaled 2,699 and the number of D. melanogaster was 2,039 (75.5%). The dispersal of males appeared to be greater than females because the mean sex-ratio (2.16) of flies collected from the second to the tenth trap was greater than the sex-ratio of flies collected in the large population (1.08). The dispersal of the natural populations seemed to decrease linearly with the square root of distance.

Among 1,330 males, 611 were mated with virgin Cy/Pm females and viabilities of homozygotes for each of 571 second chromosome were estimated in the F_3 generation. 87 chromosomes (15.2%) carried a lethal gene and 4 chromosomes among them were identified with at least two different lethal genes. Among a total of 91 lethal genes observed as above, 58 (63%) lethals were high frequent ones and classified into 14 allelic groups; the other 33 (37%) lethals were single ones. In the half diallel cross between these lethal strains, 143 crosses among 3,741 crosses were allelic, with a frequency of 3.82 per cent. The frequencies of allelism between lethal chromosomes isolated from flies collected from the same and different traps were obtained as represented in Table 2.

Table 2. The number of flies of D. melanogaster collected from the traps and the number of lethal second chromosomes isolated and the crosses in the allelism test grouped according to the distance between the traps from which the tested lethals were obtained.

Trap	D. melanogaster ♀	D. melanogaster ♂	No. of second chromosomes isolated	No. of lethal chromosomes	Distance (meters)	No. of crosses	No. of allelic crosses	Frequency of allelism	Mean (%)
1	230	249	88	5	0	647	29	4.48	4.63
2	139	342	103	20	30	1,173	56	4.77	
3	100	245	101	16	60	936	33	3.52	3.33
4	118	237	95	21	90	572	18	3.14	
5	42	91	73	13	120	293	4	1.37	1.64
6	17	33	44	9	150	105	2	1.90	
7	8	17							
8	26	42	37	1					
9	29	72	30	2					
10	0	2	0	-					
Total	709	1,330	571					Overall frequency of allelism = 3.82%	

The relationship between the dispersal of flies and the frequency of allelism of lethals in a natural population was found to be similar as B. Wallace reported (1966, Amer. Nat. 100: 565-578) in a tropical population of the same species.

Gupta, J.P. and S.P. Ray-Chaudhuri.
Banaras Hindu University, Varanasi, India.

Drosophilidae of Chakia forest, Varanasi, India.

Collections were made during the period July 1965 to March 1966 at Chandraprabha, Chakia forest, which is situated about forty-five miles southeast of Varanasi, Uttar Pradesh. A total of 2043 specimens were collected comprising seventeen species. Among them, Cacoxenus punctatus, Leucophenga albicinota, Leucophenga guttiventris, D. seguyi and D. trisetosa are newly recorded from India, whereas D. Chandraprabhiana, D. silvalineata, D. paratriangulata and D. latifshahi, all belonging to the subgenus Scaptodrosophila, are new species.

Species	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	March	Total
Cacoxenus punctatus	-	10	13	5	7	9	-	-	-	44
Leucophenga albicinota	-	-	-	1	-	2	2	9	-	14
L. guttiventris	-	-	-	2	1	-	-	-	-	3
D. seguyi	27	49	132	278	91	55	63	39	3	737
D. raychaudhurii	8	14	12	9	-	-	-	-	-	43
D. takahashii	-	-	-	-	-	3	9	7	-	19
D. kikkawai	7	4	2	7	-	-	-	-	-	20
D. bipectinata	-	-	15	20	-	-	-	-	-	35
D. malerkotliana	78	245	94	304	35	-	-	-	-	756
D. melanogaster	-	-	-	-	8	9	4	3	4	28
D. chandraprabhiana sp. nov.	8	6	8	-	-	-	-	19	75	116
D. silvalineata sp. nov.	-	-	2	-	-	-	-	2	124	128
D. paratriangulata sp. nov.	9	12	1	-	-	-	-	-	-	22
D. latifshahi. sp. nov.	-	4	1	13	-	-	-	-	-	18
D. trisotosa	-	-	-	-	1	-	-	-	-	1
D. nasuta	2	5	1	48	-	-	-	-	-	56
D. busckii	-	-	-	-	-	-	-	3	-	3
Total	139	349	281	687	143	78	78	82	206	2043

Collection records of Drosophila species collected from a forest, 45 miles S-E of Varanasi, India, July 1965 - March 1966.

Van Delden, W. Genetics Institute, University of Groningen, Haren, The Netherlands. Selection for competitive ability.

An experiment was started to study the genetic effects of combining two Drosophila species in a competitive situation. The strains used were a wild type laboratory strain of D. melanogaster and a vermillion D. simulans strain. Flies of both strains were combined for a number of generations. Each generation was started with a fixed number of parents, which were removed seven days after introduction of the simulans flies. The generation interval was 21 days during the first 60 generations, 14 days during the next generations. The experiment was done in bottles at 25°C. At the present time the lines are kept for more than 80 generations. Four selection lines were initiated: a control D. melanogaster line (A-line) continued each generation with 5 pairs of flies per bottle; a control D. simulans line (D-line) with 20 pairs of parents; a competition line (C-line) with 5 pairs of melanogaster and 20 pairs of simulans; and a competition line (B-line) with 5 pairs of melanogaster and 20 pairs of simulans, the simulans flies in this line however were derived each generation from the D-line. The melanogaster flies were added to the bottles 72 hours after introduction of the simulans flies.

To compare the competitive performance of the competition and control lines, melanogaster flies from the A-, B- and C-lines were combined with simulans flies from C- as well as D- lines in the same way as for the maintenance of the regular lines. Such tests performed during the first 10 generations did not show any differences in performance between A-, B- and C-lines. Tests done after generation 65, however, showed considerable differences between lines.

Table 1 gives the results of a test done in generation 77; recorded are the mean numbers of melanogaster flies emerging within 15 days after introduction of the melanogaster parents (each entry is based on 9 bottles). Both B- and C-lines produced more melanogaster than the A-line when combined with simulans from the C- as well as from the D-line. All melanogaster lines show a higher productivity with C-simulans than with D-simulans. The mean number of C-simulans flies (242.3) in competition lines is also higher than the number of D-simulans flies (200.6). In pure cultures the mean number of C-simulans flies is 284.9, compared to 261.3 for the D-simulans flies.

Table 1. Mean number of D. melanogaster flies emerging from combined and pure cultures.

	sim. C-line	sim. D-line	no sim.
mel. A-line	66.9	44.8	286.3
mel. B-line	131.0	82.6	381.1
mel. C-line	106.0	50.3	220.2

These data suggest selection for increased competitive ability in the B- and C-melanogaster lines. The phenomenon of the higher total productivity in the C-simulans competition lines is still under study.

Genetic changes in the melanogaster lines B and C are also suggested by the results of the heritability tests for sternopleural chaetae number in the melanogaster lines done in generations 40 and 50. Table 2 shows an increase in heritability and additive genetic variance for B- and C-lines compared to the base population and the A-line.

Table 2. Heritabilities and additive genetic variances for sternopleural chaetae number.

	generation 0		generation 40		generation 50	
	h^2	V_G	h^2	V_G	h^2	V_G
Base population	0.33	0.81	-	-	0.29	0.77
A-line	-	-	0.32	0.63	0.25	0.52
B-line	-	-	0.50	1.25	0.42	1.62
C-line	-	-	0.43	1.75	0.52	2.34

Further research is done now on the differences in developmental time between the lines (melanogaster from B- and C-lines showing a faster development than the control line).

Part of this work was supported by a grant from the Netherlands Organization for the Advancement of Pure Research (Z.W.O.) and was done at the University of Chicago.

Crossley, S. Monash University, Clayton, Victoria, Australia. Mating reactions of certain mutants.

Rendel (1951, Evolution) and Jacobs (1961, Ecology) reported that ebony mutants of D. melanogaster mated better in the dark than in the light. Attempts to confirm this result have been unsuccessful. Laboratory stocks of ebony, vestigial and Oregon-R were compared in homozygous mass matings in the light and dark as in Rendel's experiment. Ebony and Oregon-R mated equally well in both light conditions. Vestigial mated less successfully than either winged form in both light and darkness. In the tables below + = number of females inseminated and - = number of females which did not mate during the 2 hour test period.

	Light		Dark				
	+ ♀♀	-	+ ♀♀	-	χ^2	d.f.	p.
++ x ++	112	31	108	31	0.02	1	>.8
e x e	291	105	281	107	0.11	1	>.7
vg x vg	23	67	16	78	2.4	1	>.1

This differs from Rendel's results for he found that ebony mated better in the dark than in the light and wild-type and vestigial mated better in the light than in the dark.

In a second experiment mating times were obtained by viewing single pair homoganic matings every three minutes from introduction. Ebony and vestigial outcrossed stocks were used. Dark tests were examined in red light. Mating occurred in 3" x 1" vials and onset of courtships (lag) and copulation times were noted. There were no differences in lag times all pairs in the dark and light beginning to court within a few minutes of introduction.

Copulation times were significantly delayed by darkness in vestigial but not in ebony matings. The table compares the numbers mating within 15 minutes from introduction ($p<.01$*).

	Light		Dark			
	+ ♀♀	-	+ ♀♀	-	d.f.	χ^2
e x e	27	10	19	13	1	0.88
vg x vg	29	13	9	20	1	8.50*

(Average copulation times did not give a good measure because of a few long courtships in all conditions.)

The reason why darkness delayed vestigial matings but not ebony was investigated by analysing the courtship of these mutants. Male and female behaviour was recorded on alternate beats of a metronome set at 80. The wild-type which had been used for outcrossing served as a basis for comparison. The behaviour of ebony males did not differ in the light and dark in contrast to the behavious of wild-type and vestigial males which differed in the light and dark. Ebony males lost courted females frequently in the light and in the dark. Breaks in courtships followed, which delayed copulation times. Frequent breaks in courtships are typical of ebony courtships in the light and in the dark.

Vestigial and wild-type males are persistent courters in the light and seldom break off courtship. In the dark however they behave like ebony, losing their females and breaking off courtships. It was concluded that ebony behaves similarly in the dark and the light because it does not use visual stimuli during courtship. This finding supports Hotta and Benzer's conclusion that ebony has defective vision (1969, Nature). Other ways in which ebony courtships differ from the wild-type, such as decreased amounts of vibration and licking could not be related to defective vision. Vestigial males differed from the wild-type in lacking the vibration component and in decreased licking. Neither mutant male mated as quickly as wild-type males with wild-type females because mutant male courtship was less stimulating. No significant differences were found between the behaviour of mutant and wild-type females.

Several observations suggest that ebony is not completely blind. Studies in progress of activity in the dark and light may throw more light on the earlier findings of Rendel and Jacobs.

Hihara, Y.K. Tokyo Metropolitan University, Tokyo, Japan. Temperature sensitivity of the suppressor of SD action in D. melanogaster.

Two lines of recombinant SD males with and without suppressor (Kataoka, Japan. J. Genet. 42: 327-337, 1967) were treated at low temperature (17°C, for 2 days, except for Exp. 1, treated for 1-3 days), at various stages of development. Treated males, within 24 hrs after imagination, were crossed to cn bw females for 3 days.

In males without suppressor, the greatest effect was seen in stages 4 to 5 (primary spermatocytes in the testes of the 3rd instar larvae or young pupae), showing remarkable

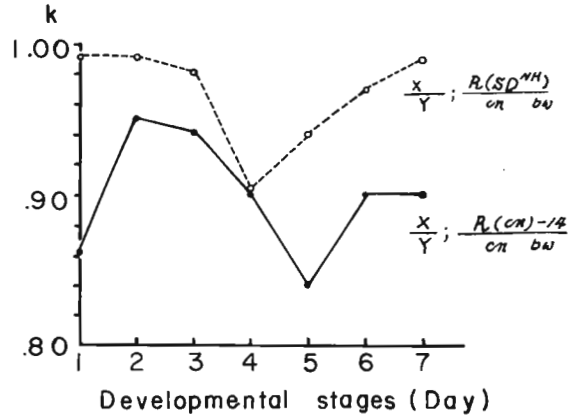


Figure 1

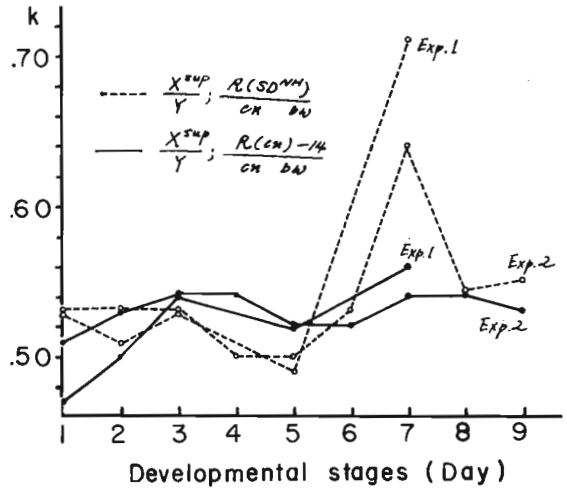


Figure 2

reduction of k value (the proportion of SD bearing sperm)(Fig. 1). This result seemed to be consistent with that of Mange (Genetics 58: 399-413, 1968).

In contrast to this, the greatest effect in suppressor bearing males was seen at stage 7 (early spermatids in pupae testes), showing remarkable rise of k value in SD^{NH} line (Fig. 2). But, cn-14 lines seemed to be not as sensitive as SD^{NH} line.

The results suggest that the active stages are different between SD and suppressor genes.

Basden, E.B. Institute of Animal Genetics, Edinburgh, EH9 3JN.
A systematic catalogue of world Drosophilidae.

There are attendant difficulties with the preparation of a catalogue of Drosophilidae. Apart from the vast volume of publications containing descriptions of new species from 1758 (Linnaeus) until today, the choice of content is not easy.

A check-list of names is the bare mimimum, but is this enough? The forward looking systematist and the backward looking science historian may require rather more. The assaying of a taxon will engage the former; the development of that assaying will intrigue the latter. The 21st Century worker may use methods for the separation of species not used today,

Since taxa are separated according to their differences and grouped according to their similarities, would it not be useful to give references (at least the main ones) to where not only quantitative but also qualitative comparisons are made between species? Besides the usual morphological descriptions (including those of chromosomes), details of geographical distribution would be required by some workers; or breeding biology; or behaviour; or reactions to vapours or dusts. Biochemical differences will become increasingly used.

Normally the morpho-species is investigated for its reactions; or its composition; or its intimate relationships with other morpho-species. These investigations may themselves discover, and decide, a species and its relationships.

Therefore the most useful systematic catalogue would include references to the above particulars. But is it too much to expect?

Lamborot, M. and S. Koref-Santibañez. Universidad de Chile, Santiago, Chile. Temperature and sexual isolation between D. gaucha and D. pavani.

A study of mating activity of the sibling species D. pavani and D. gaucha (DIS 1966 42: 106; Biológica 1967 61: 3-6) revealed that the optimum temperature for sexual activity was lower (18°C) in the Chilean than in the Brazilian species (25°C). Interspecific crosses showed a differential receptivity of the females. As the isolation indices could not be obtained, a new set of experiments was set up, using the "male choice method" at nine different temperatures: 6°C, 8°C, 12°C, 16°C, 20°C, 24°C, 28°C, 32°C and 34°C). For each temperature the activity of approximately 100 males was studied. 5 males were placed for 6 hours with 5 females of their own and 5 of the sibling species in 5 x 20 cm. vials. The ventral receptacle and the spermathecae of the females were examined for the presence of sperm.

Tables 1 and 2 summarize the results obtained, together with the isolation coefficients (K) (Malogolowkin-Cohen et al. Evolution 19: 95-103). While D. pavani males show little isolation throughout the temperature range, having even greater preferences for the foreign female, D. gaucha males reveal marked preferences for their own females, specially notorious at extreme temperatures; at the temperature at which the activity of D. gaucha was optimal, isolation was lowest. The results point to the conclusion that female receptivity seems to be more responsible for sexual isolation than the activity of the male.

Table 1. ♂ pavani

Temp. °C	Homog. %	Heterog. %	K
6	32.0	16.0	o.43±0.19
8	48.9	34.8	0.22±0.11
12	42.3	39.6	0.05±0.08
16	71.2	72.5	0.01±0.07
20	77.3	79.2	0.01±0.07
24	72.5	83.2	0.16±0.08
28	59.6	61.5	0.02±0.09
32	23.6	34.6	0.22±0.04
34	6.0	24.0	0.63±0.01

Table 2. ♂ gaucha

Temp. °C	Homog. %	Heterog. %	K
6	24.0	17.0	0.16±0.22
8	48.9	17.2	0.51±0.10
12	51.2	21.2	0.49±0.07
16	66.2	22.8	0.61±0.06
20	75.7	27.9	0.59±0.06
24	89.1	72.2	0.27±0.08
28	79.4	40.4	0.45±0.08
32	71.8	20.0	0.69±0.07
34	40.4	4.7	0.85±0.14

Homogamic, heterogamic preferences and isolation index (K) of D. gaucha and D. pavani males at different temperatures

Kuroda, Y. National Institute of Genetics, Misima, Japan. Effects of X-irradiation on the differentiation of eye-antennal discs of D. melanogaster in organ culture.

Eye-antennal discs were irradiated with 0 R, 500 R, 1,000 R, 1,500 R and 2,000 R of X-rays (180 kV, 25 mA, 1.0 mm Al filter, distance 40 cm, dose rate 300 R/min) immediately after their preparation in hanging drop cultures from mature third-instar larvae of the Oregon-R strain of D. melanogaster. After irradiation the discs were cultured in a chemically defined medium containing 10^{-4} mg/ml rubrosterone and examined for the effects of X-rays on the differentiation of ommatidia. When eye-antennal discs were irradiated with 500 R or 1,000 R no marked inhibition was observed in the differentiation of ommatidia after 24 hours of cultivation. The organization of ommatidium-forming cells into cell clusters was observed in the eye disc portion as seen in eye-antennal discs in non-irradiated control cultures. With 1,500 R the differentiation of ommatidia was partially inhibited 24 hours after explantation. 2,000 R inhibited almost completely the differentiation of ommatidia when examined after 24 hours of cultivation.

When eye-antennal discs were irradiated first with a dose of 1,000 R immediately after explantation, then they were exposed to a second dose of 1,000 R at 2 or 4 hours after explantation, the effects of X-ray were found to be different depending on the extent of the intervals between the first and second doses. With the second dose given at 2 hours after explantation the differentiation of ommatidia was partially inhibited after 24 hours of cultivation; whereas with the second dose given at 4 hours after explantation no inhibitory effect of X-ray was observed on the differentiation of ommatidia.

These results suggest two possibilities; the presence of repair 4 hours after the first irradiation, and alternatively, the differential sensitivity of the eye-antennal discs at different stages of cultivation. To examine these alternatives, eye-antennal discs were irradiated with single dose of 2,000 R 2 hours or 4 hours after explantation. When eye-antennal discs were irradiated with 2,000 R 2 hours after explantation, the differentiation of ommatidia was partially inhibited. However 2,000 R of X-ray had no inhibitory effect on the differentiation of ommatidia when given 4 hours after explantation. These results suggest that the organization of ommatidium-forming cells was inhibited by 2,000 R of X-ray when eye-antennal discs were irradiated at 0-2 hours after explantation. After 4 hours of cultivation eye-antennal discs showed no pronounced changes in morphology but they had a lesser sensitivity to X-ray and resulted in the full organization of ommatidium-forming cells following 2,000 R of X-irradiation.

Kuroda, Y. National Institute of Genetics, Misima, Japan. Effects of BUdR, actinomycin D and puromycin on the differentiation of eye-antennal discs of D. melanogaster in organ culture.

Eye-antennal discs dissected from mature third-instar larvae of the Oregon-R strain of D. melanogaster were cultured in chemically defined medium as described in the previous paper (1). In the medium supplemented with 10^{-4} mg/ml rubrosterone (an ecdysone analogue isolated from plants) a pronounced differentiation of ommatidia was observed in 92% of eye-antennal discs after 24 hours of cultivation at 28°C (2). When 10^{-5} M BUdR (5-bromodeoxyuridine, Sigma Chem. Co., crystaline) was added to the medium containing 10^{-4} mg/ml rubrosterone, eye-antennal discs showed differentiation of ommatidia similar to that in control cultures without BUdR. Similarly, the addition of 1 μg/ml actinomycin D (Daiichi Pure Chem. Co., Ltd.) to the medium containing 10^{-4} mg/ml rubrosterone also had no effect on the hormone-induced differentiation of ommatidia. The presence of 10 μg/ml puromycin (Nutritional Biochem Corp.) also did not inhibit the hormone-induced differentiation of ommatidia. These results are summarized in Table 1.

Table 1. Effects of BUdR, actinomycin D and puromycin on the differentiation of ommatidia in eye-antennal discs cultured in chemically defined medium containing 10^{-4} mg/ml rubrosterone

	No. of explants tested	No. of explants in which ommatidia differentiated	Percent of differentiation
Control	12	11	92
BUdR (10^{-5} M)	7	6	86
Actinomycin D (1 μg/ml)	14	11	79
Puromycin (10 μg/ml)	10	7	70

This suggests that the organization of ommatidium-forming cells in eye-antennal discs in organ culture promoted by an ecdysone analogue was not inhibited by inhibitors of RNA and protein synthesis and that the process of the formation of ommatidial cell clusters may be conducted by pre-existent macromolecules which were activated into their functioning by ecdysone analogue.

1. Kuroda, Y. and Tamura, S. 1956, Med. J. Osaka Univ., 7: 137. 2. Kuroda, Y., 1969, Japan. J. Genetics, 44, Suppl. 1: 42.

Zuill, E.E. Oxford University, England.
A measure of behavioural heterosis in D.m.

The occurrence of deleterious, recessive alleles in natural populations has, for a long time, been subject to much conjecture and investigation. Single gene heterosis has been suggested as a possible mechanism for the maintenance of these alleles in natural populations. The evidence is far from conclusive, for there are many unknown interactions caused by epistasis, pleiotrophy and convergence of gene effects. The hypothesis under investigation is that the heterozygote has a behavioural advantage over the equivalent homozygote. I postulate that this advantage is caused by the more stimulative components of the male's courtship pattern which enables him to mate more quickly and with more females hence passing on his complement of alleles to more individuals than a male lacking this behavioural advantage.

Heterozygous males were obtained from the F1 generation cross of KAD 5 males and ebony females. 35 males of this cross were tested singly with virgin 48-hour old females in 2cm. perspex chambers. Male courtship was recorded using the Bastock metronome technique. It has been shown that male wing vibration is one of the components that stimulates the female to accept the male. The percentage of wing vibration is significantly higher in the heterozygote pattern than in the homozygote pattern, (Student's 't', $P<0.1$). This 'heterotic' behaviour may have been caused by heterozygosis at many loci and may, therefore, merely be an expression of outcrossing. A programme of sib-mating between heterozygote male and ebony female has been established to try and reduce the heterozygosity of the background gene pool. Two things may occur; first, the increased wing vibration percentage may be retained in the heterozygous males, or second, the influence of the deleterious ebony allele may become apparent. Inbreeding depression may also reduce the wing vibration score. The behaviour of the female in courtship observations is also very important and this will be investigated.

MATERIALS REQUESTED OR AVAILABLE

Quarter pint milk bottles may be obtained from Monroe Machinery and Supply Company, 1421 S.E. Gideon, Portland, Oregon 97242, attention Mr. Ernie White. The supply is limited, therefore it is advisable to place orders at once.

J.E. Purkyně, Dept. of Genetics, Faculty of Science, Brno, Czechoslovakia would like a test stocks Gl/Ubx^{130}, Ubx^{130}/ Sb and M-5; Cy/Pm; Sb/Ubx.

E. Ortiz, Instituto de Genética y Antropología, Madrid, Spain, would appreciate receiving any strains of D. kuntzei, D. limbata, D. phalerata, D. transversa and D. andalusiaca (= forcipata).

V.G. Vaidya, Department of Zoology, University of Poona, Poona, India, would appreciate receiving reprints of old and new publications on Drosophila for the library of the newly started Drosophila laboratory.

C.C. Dapples, Department of Biology, Rocky Mountain College, Billings, Montana 59102, would greatly appreciate receiving reprints on current work on Drosophila to supplement the library of this department.

Vials (34 X 98 mm) - Cardinal Products has approximately 60 gross on hand. For further information write to Dr. Thomas Amore, Cardinal Products, P.O. Box 1611, Durham, North Carolina.

Bakula, Marion. Saint Louis University, Missouri. Food preference testing in D. melanogaster adults.

A simple, economical apparatus has been designed to test the preferences of adult Drosophila when presented with two different nutrient sources (Figure I). Etherized flies are placed in the center chamber (A) and allowed to move freely for 24 hours. At the end of 24 hours the test tubes (C) containing the foods being tested are removed and the flies in each tube counted. 24 hours has been found to be the minimum time necessary for approximately 100 flies to distribute themselves nonrandomly between an agar-Brewer's yeast medium containing either 0.8% sucrose or 0.8% lactose. The two sugars were selected in order to test the reliability of the apparatus since the threshold concentration of sucrose is low while that of lactose is relatively high in Phormia (1) and Calliphora (2). In addition, although Drosophila larvae develop equally well on 0.8% sucrose or lactose (3), survival of adult flies is good on sucrose and poor on lactose (4). It was therefore expected that Drosophila adults would prefer sucrose both on the basis of threshold levels in other Dipterans and on the apparently poor nutritional utilization of lactose. (Preference of Pseudosarcophaga larvae for nutritionally optimal diets has recently been demonstrated by House (5).)

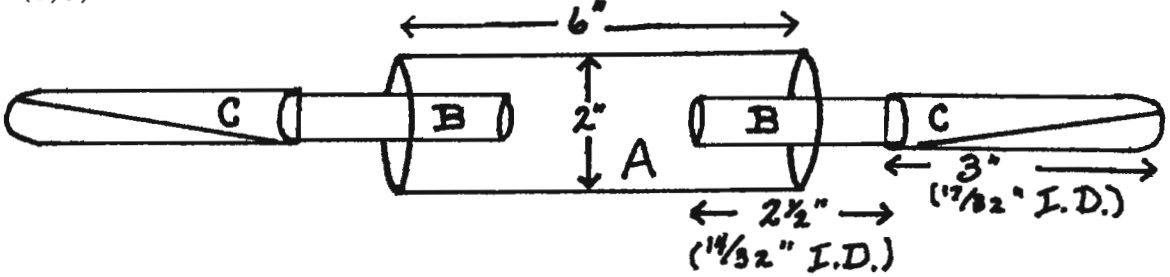


Figure I. Taste testing apparatus. Etherized flies are placed in the center chamber (A), and after awakening are free to move through the connecting tubes (B) to the food tubes (C). Sterile nonabsorbent cotton holds the connecting tubes in place while permitting ventilation of the apparatus. The food tubes slide over the connecting tubes and may be easily removed for fly counting, and may be changed without interrupting the experiment.

The movements of the flies within the testing chamber was tested by removing the food tubes (C) at intervals, counting the number of flies in each tube and marking the wings of flies on lactose medium with black ink. The results over a 24 hr. period are shown in Table I. The results would suggest that nearly all of the flies move back and forth before making a final choice, often remaining stationary in the center chamber, and it is only towards the end of the test period that a significant difference in distribution occurs. In one experiment the flies were kept in the apparatus for 48 hours without significant change in distribution from 24 hours. The flies were not starved before the experiments and it is probable that hunger prompts the final selection of an optimal carbohydrate. Results obtained using less than 75 flies are unsatisfactory, no significant distribution being obtained in about 50% of the experiments. When more than 125 flies are employed the food tubes become too crowded, and many flies are pushed into the food and immobilized, the final distribution however, being equivalent to that obtained with 75-100 flies.

Table I. Random movement of Drosophila melanogaster adults.

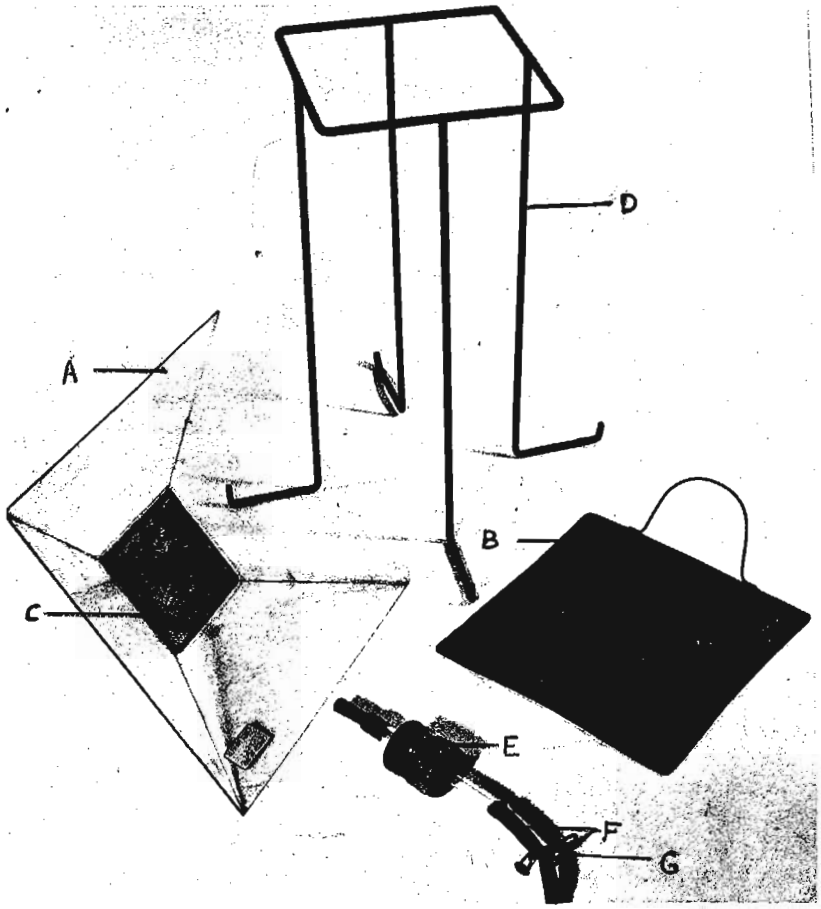
No. of hours after start of test (0 hrs.)	No. of flies on Lactose		No. of flies on Sucrose	
	Total No.	No. previously on Lactose	Total No.	No. previously on Lactose
1	5	-	3	-
2	17	2	6	0
4	14	3	4	0
6	35	9	8	0
18	18	4	12	0
24*	29	16	66	38

* chi^2 calculated on an expected 1:1 distribution assuming no food preferences = 13.5 ($P<0.5\%$)

References. (1) Evans, D.R. 1961. Science 133: 327-328. (2) Minnich, D.E. 1929. Z. vergl. Physiol. 11: 1-55. (3) Sang, J.H. 1956. J. Exp. Biol. 33: 45-72. (4) Hasset, C.C. 1948. Biol. Bull. 95: 114-123. (5) House, H.L. 1967. Canad. Entomol. 99: 1130-1321.
(This investigation was supported by PHS Training Grant No. GM00989 while the author was a postdoctoral trainee in the Department of Zoology, University of Michigan.)

Rey, B.M. and W.F. Kirschbaum. Atomic Energy Commission, Buenos Aires, Argentina. A simplified "ovitron".

Since it was not possible for us to obtain an "ovitron"of the type described by Yoon and Fox (Nature, 206(4987): 910-913, 1965), we designed a simpler, less expensive model which could be made in our shop. It consists of a large square shaped lucite funnel, held in a metal frame and provided with the appropriate screens and egg-collecting apparatus.



Although this apparatus is not as convenient to use as the Yoon and Fox model, it has given good results. In figure 1, A is a square lucite funnel, 29 X 29 cm., B is a movable bronze screen, and C is a fixed bronze screen. D is a metal support for the funnel. E is a glass recipient whose removable base holds a fine cloth filter which collects the eggs. Rubber tubes (E) connect the parts and the system is closed or opened by a Mohr clamp (G).

Cuperus, P., J.A. Beardmore and W. van Delden. Central Electronics Service and Genetics Institute, University of Groningen, The Netherlands. An improved circuit diagram for an electronic fly-counter.

An improved circuit diagram for the fly-counter described in DIS 44: 134 is available on request from the senior author.

Ellison, J.R. University of Oregon, Eugene. An improved method for electron microscope preparation of salivary gland polytene chromosomes.

The following technique allows both high resolution phase contrast light microscopy and electron microscopy to be performed on the same cell. The salivary gland is dissected out in saline and placed in a drop of a mixture of 1 part acetic acid, 2 parts lactic acid, and 2 parts water on a silicone coated slide and allowed to stand for 3 minutes. The gland is covered with a silicone coated cover slip and squashed. The slide may be surveyed at this time using phase contrast microscopy. The slide is then frozen in liquid nitrogen and the cover slip is removed. Immediately the slide is immersed in 95% ethanol (5 min.). The slide is then placed in a mixture of equal parts of absolute ethanol and acetone (5 min.), followed by 100% acetone (5 min.). The slide is then stained with acetone saturated with uranyl acetate (5-10 min.). The slide is rinsed in 100% acetone (1 min.) and put into a 20% epon/acetone mixture (5 min.). Epon is then applied over the squash with a 10 ml. disposable syringe. Broken pieces of slides are used as spacers and another slide is placed on top of the epon (fig. 1). The epon is allowed to polymerize for 48 hours at 60°C. The slide is then placed on a hot plate at about 200°C. with the slide

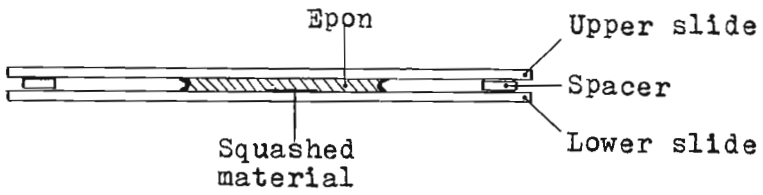


Fig. 1

on which the squash was made against the hot plate. Immediately the slides are pried apart by inserting a small screwdriver between the slides and gently twisting. The epon will separate from the lower slide taking the squash material with it. The cells are now on the surface of the plastic and can be observed with phase contrast optics. Desired cells can be marked with either a diamond stylus or ink objective marker. If oil immersion microscopy is desired, glycerine may be placed on the epon and a cover slip applied. Immersion oil can then be used on top of the cover slip. The glycerine washes off and does not interfere with subsequent sectioning. The selected cells are then cut out and glued (with epoxy glue) on to 1 cm. segments of 8mm plastic rods, and sectioned.

R. Nöthiger. Zoological Institute, University of Zürich, Switzerland. Sucrose density separation - a method for collecting large numbers of Drosophila larvae.

Materials required: a separatory funnel (1000 ml, with a valve opening of 4-6 mm) fixed to a ringstand, a long glass rod, a brush, a piece of fine meshed nylon cloth, solution of ca. 20% sucrose in water.

Pour sucrose solution into populated food container, stir with brush and bring larvae "into solution". Pour the suspension of larvae, corn meal, and perhaps dead carcasses and empty pupal cases into separatory funnel. Add slowly a little water, stir with the glass rod until corn meal sinks to the bottom and the rest floats. Bigger pieces of corn meal are crushed with the glass rod. Now open valve and release cornmeal fraction. Repeat this washing procedure, if necessary. Then add water: larvae will sink to the bottom, carcasses and empty pupal cases will float. Now release larvae onto nylon mesh, wash if desired, and collect.

This method is especially useful for collecting young larvae. Development after this treatment is normal.

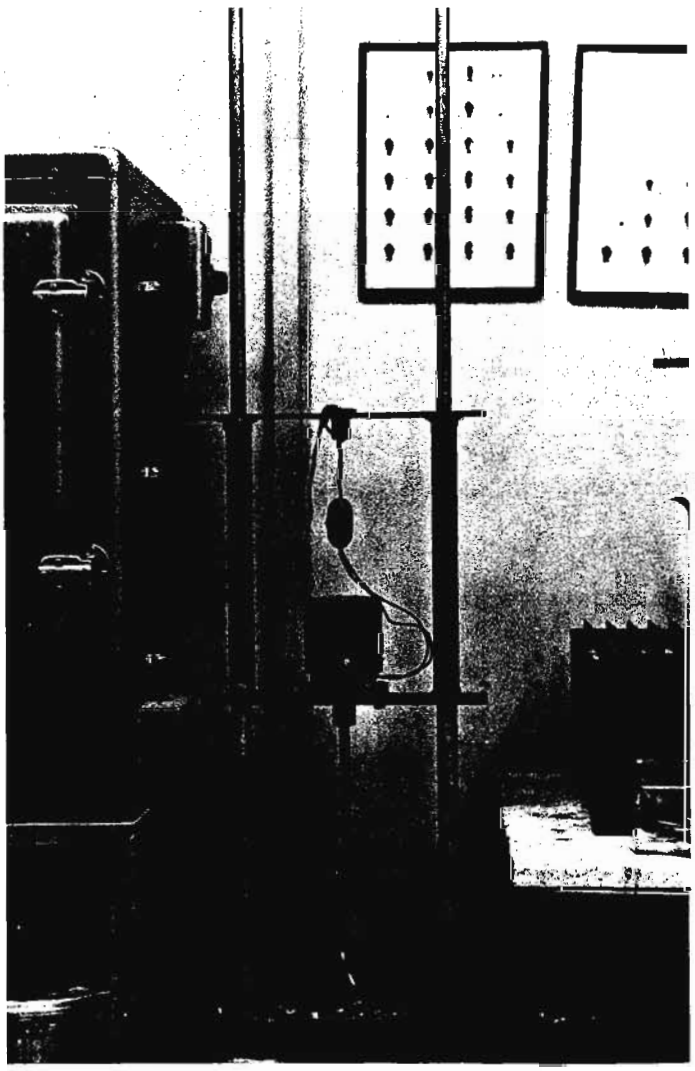
The essentials of this method have been brought to my attention by Drs. F. Ratty and R. Rinehart of San Diego State College, California.

Félix, R. Programa de Genética y Radiobiologia. Comisión Nacional de Energía Nuclear. México City, México.

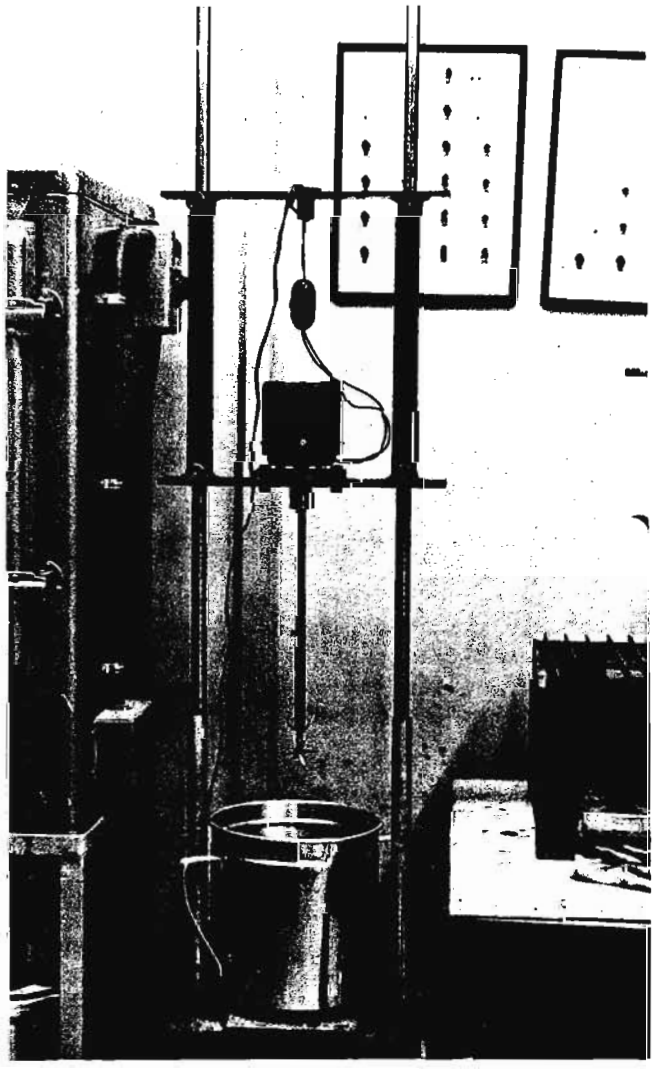
High speed stirrer and mixer for Drosophila food medium.

A compact, quiet-running unit for mixing the ingredients of Drosophila food medium has been used in this laboratory since five years ago, proving to be very useful. The outstanding points in this stirrer are: (a) a sturdy motor (1/20 HP, 1.5 amp., 115 volts, 60 Cy. 1590 r.p.m. and 110 w., Payton Model 5K001. Dayton Electric Mfg., Co., Chicago 48, Ill.); (b) the stirring rod ½ inch in diameter and 12 inches long is welded to the motor shaft, and goes through a brass axle box (1 1/8 inch in diameter) fixed on a rectangular support; (c) a four-blade and three two-blade cutting propellers (2 inches in diameter) of stainless steel (replacement parts of waring blenders) are screwed to the distal end of the stirring rod in two units separated by one inch along the axis. When mixing is going on there is a distance of ½ inch between the lowest propellor and the bottom of the pot containing the food medium. Aluminum pots from 2.5 to 6 litres may be used for the boiling and mixing of the food medium. Centrifugal effect is minimized, no vortex is created, liquid level remains essentially constant, allowing use of nearly filled containers without risk of slosh over.

There are two vertical steel support rods, 3/4 inch in diameter and 48 inches long each, separated by 10 inches and screwed on a rectangular support. The sliding frame with two horizontal plates welded to vertical tubes with four brass thumb screws and four brass axle boxes permits the adjustment for height as required in order to move the pot before and after stirring. Two aluminum tubes, 15 inches long, stop the sliding frame to its correct position before the motor is started.



View of the stirrer when not working



Adjustment for height to move the pot

Sega, G.A. and W.R. Lee. Department of Zoology, Louisiana State University, Baton Rouge. A vacuum injection technique for obtaining uniform dosages in D. melanogaster.

A new method to administer quantitatively chemical mutagens to D. melanogaster by vacuum injection has been developed at our laboratory. Previous micro-injection techniques have been criticized by Carlson and Oster (Genetics 47:561) because individual flies vary as to the amount of injected material that is retained. Preliminary tests in our lab in which 0.2µl. of ^{14}C-ethyl methanesulfonate (EMS) was injected per fly confirmed this variability. In several experiments we obtained a coefficient of variation (C.V.) of the retained radionuclide that averaged near 60%. (The coefficient of variation is the percentage ratio of the sample standard deviation to the sample mean: $s/\bar{x} \cdot 100\%$)

The feeding method of E.B. Lewis and F. Bacher (DIS 43:193) was also used and we obtained in this case a C.V. of 16%. However, a considerable amount of mutagen was necessary to carry out a feeding experiment. A method of treatment was therefore devised that gives a comparable C.V. while using only 10µl. of mutagenic solution.

In our vacuum injection method, a C.V. of 10% was obtained using ^{14}C-EMS. The procedure was to place 10 nonetherized adult males in a 25 ml. serum vial and then lower the absolute pressure to between 40 and 50 mm of Hg. Freshly etherized flies were killed by the vacuum treatment. Increasing the number of flies per vial above 10 gave a lower received dose per fly. Ten µl. of water containing ^{14}C-EMS was then taken up into a syringe needle attached to a 1 ml. syringe with the syringe plunger withdrawn to take in 0.1 ml. of air. The syringe needle containing the 10µl. was then inserted through the rubber stopper of the serum vial, and atmospheric pressure forced the mutagen into the vial as an aerosol. Absolute pressure rose only slightly. After flies were left in the vials from one to two hours the vacuum was broken by inserting a large hypodermic needle through the rubber stopper. It was thought that the sudden increase in air pressure caused the mutagen that had diffused into the trachea to be forced into the tissues of the fly, although this point is still open to question.

Increasing the concentration of ^{14}C-EMS by a factor of 10 resulted in a ten-fold increase in the amount of retained radionuclide. The incorporation of the radionuclide was found to increase with time in the vacuum; however, leaving the flies in the vials for longer than two hours caused death in some cases. Genetic tests have shown this method to be effective in producing mutations. A test of 323 chromosomes from males treated with 10µl. of 0.12 M non-labeled EMS for one hour gave 42% sex-linked recessive lethals in the F_2.

Hunt, Virginia. Developmental Biology Center, Case Western Reserve University, Cleveland, Ohio. A qualitatively minimal amino acid diet for D. melanogaster.

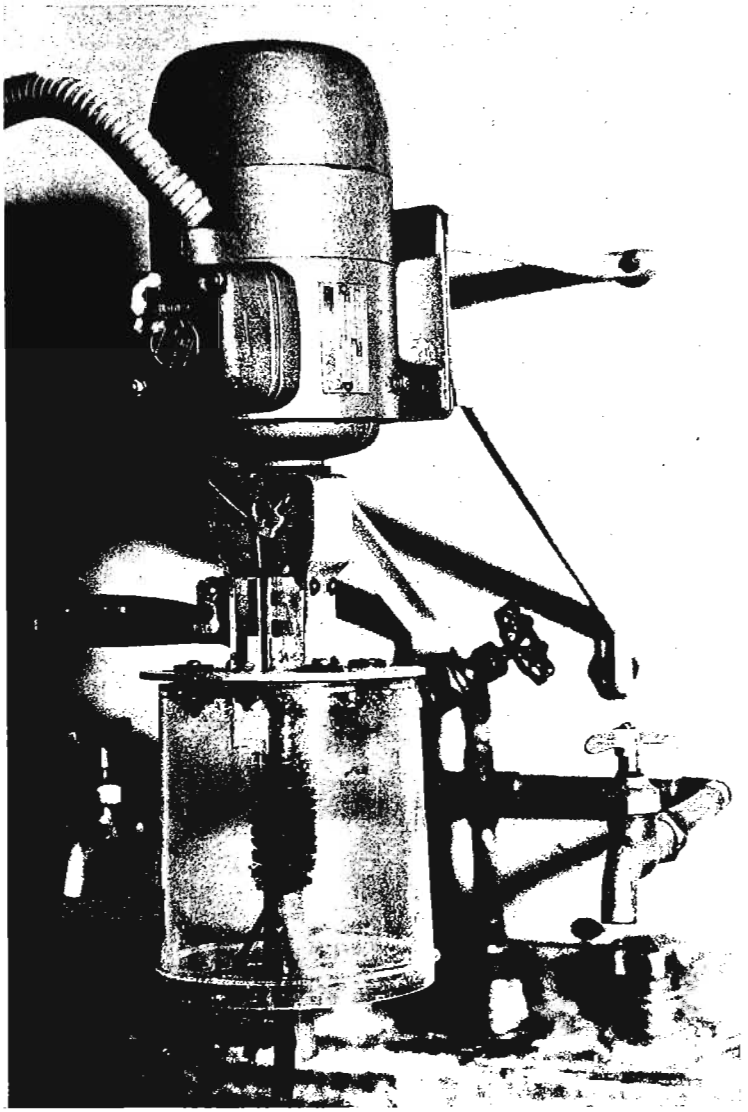
Using a modification of Geer's amino acid diet (DIS 40:96) as a basic test medium, we have developed a medium which is completely defined and quantitatively minimal. Three amino acids can be omitted from Geer's original formulation and RNA replaced by inosine and uridine, without adversely affecting either development time or survival. The omission of either glycine or tyrosine causes no significant change in development time or survival, while the omission of cystine causes a significant decrease in development time and a significant increase in survival

	mg.		mg.		mg.		mg.
L-Arginine·HCl	80	L-Threonine	200	Thiamine	0.20	$MgSO_4 \cdot 7H_2O$	24.60
L-Glutamic acid	840	L-Tryptophan	50	Nicotinic acid	1.20	$NaHCO_3$	100.00
L-Histidine·HCl	100	L-Valine	280	Riboflavin	1.00	KH_2PO_4	71.90
L-Isoleucine	300	Agar ("Bacto")	1500	Ca pantothenate	1.60	K_2HPO_4	373.60
L-Leucine	200	Sucrose	1000	Pyridoxin	0.25	Water to 100 ml.	
L-Lysine.HCl	190	Inosine	80	Biotin	0.03		
L-Methionine	80	Uridine	70	Folic acid	1.00		
L-Phenylalanine	130	Cholesterol	30	Choline chloride	8.00		

Glutamic acid was found to be non-essential for survival but essential for normal development time. The minimal medium follows: ($FeSO_4$, $CaCl_2$, $MnSO_4.H_2O$ were omitted as they were found to be unnecessary by Sang (J. Exptl. Biol. 33: 45-72).

Félix, R., and V.M. Salceda. Programa de Genética y Radiobiologia. Comision Nacional de Energía Nuclear. México City. México. A motorized, waterflown device to wash rapidly large numbers of culture bottles and vials.

Washing of culture containers consumes considerable time in Drosophila laboratories. For this reason automatic washing devices are desirable and economic where large numbers of culture containers, such as bottles and vials are regularly used. The motor driven apparatus assembled at our laboratory has proved to be very helpful for speeding the washing of half-pint bottles and vials. The apparatus consists of a rotating brush with water outlets which perform the washing and rinsing of the material in a very short time. In order to illustrate the framing of the apparatus a photograph, as well as a figure, are annexed. The assembled device is employed during several hours a day thoroughly washing hundreds of bottles and vials per day, a task that otherwise would be impossible to accomplish by only one person.



View of the apparatus
with the shielding of acrylic plastic

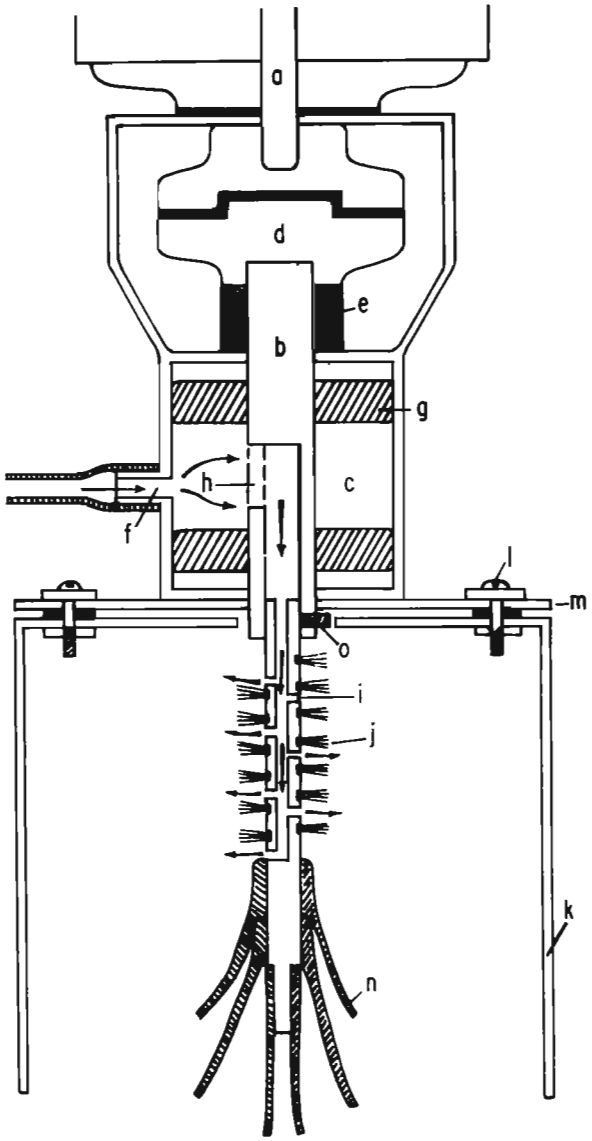


Diagram of
the waterflow apparatus

The whole assembly is driven by an a.c. motor (1/10 HP, 110-220V, 0.09 KW, and with 1,340 r.p.m. Mez Mohelnke, Checoeslovakia). The rotation of the motor shaft (a) is transmitted to the hollow shaft (b), in the water chamber (c) by means of a rubber-steel couple (d) in order to soften the rotation movement. A brass axle-box (e) below the couple supports the water chamber. The water flows through the water inlet (f) into the water chamber which is sealed above and below with hermetical pieces of rubber (g), avoiding leaks of the inflowing water. Several holes (h) are drilled in the proximal portion of the hollow shaft. The water goes through the hollow shaft and flows out at the water outlets (i) drilled among the turns of the steel brush (j) fixed to the spiral furrow on the surface of the washing shaft by means of a copper wire.

In order to shield the operator from splashing, an acrylic plastic container available commercially for refrigeration storage (k) is fixed by screws (l) to a circular aluminum plate (m) in the base of the apparatus. The washing shaft goes through a circular hole made in the bottom of the plastic container. The steel brush is limited to the upper part of the washing shaft, in order to remove the wastes and pupas accumulated in the neck of the bottle. An additional cleaning additment is adapted to the distal end of the shaft, inserting pieces of latex or rubber tubing cut to make stripes (n) as shown in the photograph. The centrifugal force given by the high speed of the rotating shaft lifts up the bottle cleaning it thoroughly.

An interchangeable washing shaft for vials is adapted by means of a screw (o). It has cut tubing with free ends of proper length. The water flows out from the distal end of the hollow shaft, thus helping to eject the medium and wastes out of the vial.

The whole assembly is mounted on the wall, above a washstand; the collected water with the ejected medium is disposed directly through the cesspool. The temperature of the water is regulated by mixing with a metallic T-shape tubing connector the hot and cold water flowing from the faucets of the washstand. Rubber gloves may be used to protect the operator's hands, however, the few defective vials which may break during the washing operation offer no danger due to the softness of the rotating latex stripes and to the protection offered by the shielding acrylic plastic container.

Wattiaux, J.M. Medical School, Facultés Universitaires N.D. de la Paix, Namur, Belgium. Squash preparation of nurse cells for Feulgen photometry and autoradiography.

Since they are highly polyploid and involved in vitellogenesis, nurse cells prove a very interesting material for histophotometry and autoradiography work. The ovaries are labelled either by injection or by incubation. Schneider medium or even buffer I devised by Ristow and Arends (1968) turn out to be quite satisfactory. Buffer I is made from tris buffer (0.01M pH 7.0), 3 mM $MgCl_2$ and 0.22 M sucrose. We used Schneider medium only for incubation. Afterwards, the ovaries are fixed for 20 to 30 mins. in formalin (4%, pH 7 in M/15 phosphate buffer) and rinsed overnight in hypertonic sucrose (refrigerator). They are rinsed in buffer I and incubated in a solution of 0.1% of pronase (in buffer I) during 15 mins. at 37°. Mature eggs are then easily removed with dissecting needles and the ovaries are squashed very gently on albumized slides - spreading should be checked with a binocular. After cooling in dry ice (10 to 15 mins.), coverslips are quickly removed and the slides dried in warm air. They are ready for the regular autoradiography processing: removal of unincorporated labeled precursor by immersion in a cold solution, stripping or coating, exposure and development.

For grain counting nuclei have to be examined in phase contrast and mapped to allow further investigation.

For histophotometry the gelatin of the autoradiography preparations has to be removed. This will be best performed during the first stage of the Feulgen processing, by 5 N HCl treatment at 25° during 60 mins. Silver grains are then either removed or completely dispersed and do not interfere at all with the cytophotometric measurement. The slides are rinsed immediately in ice cold SO_2 and dipped during 60 mins. in Schiff reagent at pH 2.6 (room temperature). They are afterwards rinsed again in SO_2 saturated water (2 x 30 mins.) dehydrated in ascending alcohols and mounted in Harleco resin. The preparations are then ready for histophotometrical measurement. Follicle cells might be used quite conveniently as a standard to determine the relative amount of ploidy since most of them are either diploid or tetraploid.

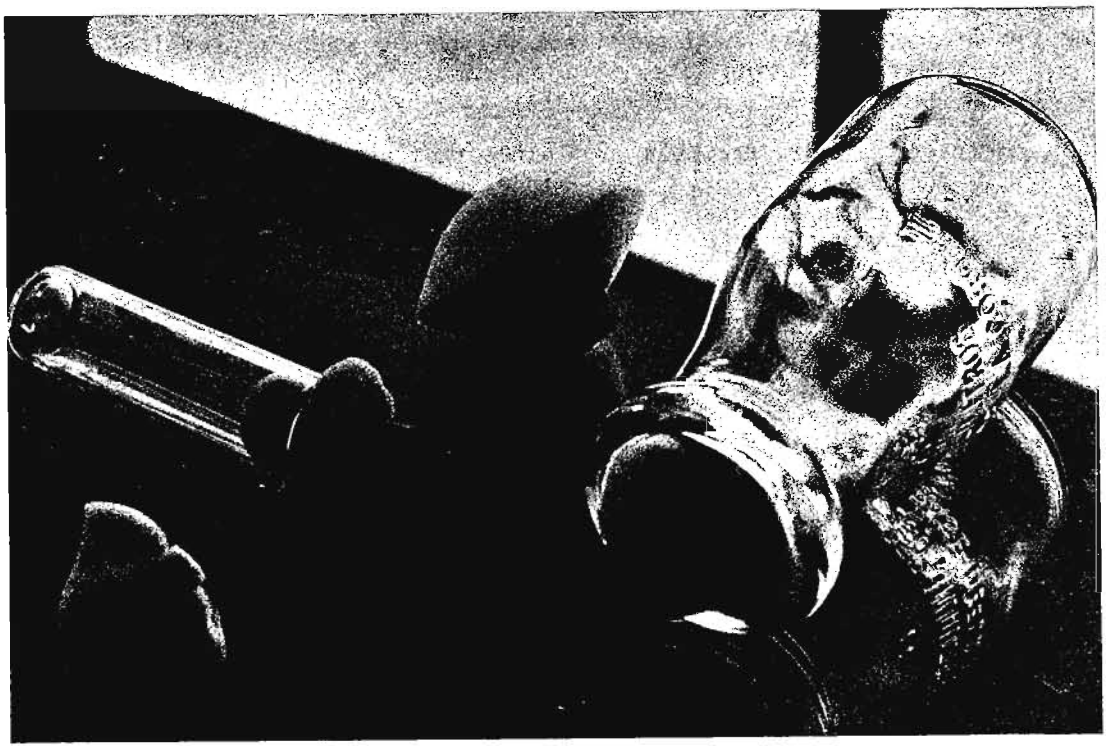
Reference: Ristow, H., and S. Arends. A system in vitro for the synthesis of RNA and protein by isolated salivary glands and nuclei from Chironomus larvae. BBA, 157: 178-186 (1968).

Félix, R. Programa de Genética y Radiobiologia. Comisión Nacional de Energía Nuclear. México City, México. Durable plastic foam plugs used as stoppers for bottles and vials.

Polyurethane foam plugs have been largely employed in this laboratory in place of cotton plugs, since they may be re-used for more than a year without losing their resiliency. The plugs were manufactured at the laboratory, cementing together a piece of rubber tubing with a polyurethane disc. The size of the two pieces are as follows:

	For half-pint bottles	For vials of 1x3½ inches
Polyurethane disc		
Diameter, mm.	70	60
Thickness, mm.	20	5
Rubber tube		
External diameter, mm.	20	15
Length, mm.	40	30

The 20 mm. (diam.) tubes may be obtained cutting to pieces commercial hose tubing. To cement the two pieces together we have used an adhesive employed for the cementing of sole-leather. A first coat of the adhesive is applied to both pieces leaving a circle in the middle of the foam disc without covering to assure proper ventilation of the cultures. An hour later a second coat is adhered to the first one and the two pieces are forced into the container. After a few days, when the cement is dry, the plugs are taken away and autoclaved at 120°C (248°F) when necessary. The used plugs are washed and dried in a dry oven at 75°C. They are handled more easily with repeated use.



Polyurethane foam plugs allow proper ventilation of culture bottles and vials.

The bottles and vials are sealed perfectly without excluding air and evaporation. Another advantage is the economy of re-use which makes then cheaper than cotton, and elimination of allergic responses to the irritation of cotton fibres. However, the economy of time which comes from the repeated use is the main quality that makes the fabrication of these hand-made plugs highly advisable.

Carlson, P., and M. Tsukada.** Yale University, New Haven, Connecticut.

A replica technique for the electron microscopic analysis of the wing surface of Drosophila.

A two stage replica procedure has been devised to allow fine structure observation of the wing surface. The technique would be valuable for studies on speciation, phenogenetics, and cuticle development. The method is as follows: 1. Rinse entire wings in 70% ethanol and dry. 2. Place wings on a methyl methacrylate plate* (methyl methacrylate, 98%; benzyl peroxide, 2%; heat at 80°C. until polymerized), sandwich between two clean glass slides, and fix in place with an even, firm pressure (two ordinary paper clamps will do). 3. Heat to 100°C. for 30 to 40 mins. and cool to room temperature. 4. Remove the glass slides and cover the methyl methacrylate with 10% polyvinyl alcohol (dissolved in double distilled water at 80°C). Allow the alcohol to dry and strip off the residue with forceps. All wing remains should be removed by 4 to 5 repetitions of this procedure. 5. Shadow the methyl methacrylate cast with carbon (200Å) and chromium (50Å). 6. Using a sharp razor, cut the metal film into squares small enough to fit on a grid (3mm. x 3mm.). 7. Place the shadowed methyl methacrylate into a bath of 1:1 chloroform-benzene which slowly dissolves the plastic and releases the metal film. Pick up the film on a grid and wash 3 to 4 times in a fresh preparation of the same solution. 8. Observe in the electron microscope.

Fig.1. Upper surface of the wing of a Canton S female. Magnification 10,000X black line = 1 micron. Note the regular granular structures on the wing surface. Each granule is marked by a center pore. The base and shaft of wing hairs are evident.

The photograph illustrates some of the regular detail visible in such a preparation.

*Commercially available from Oken-Shoji Co.,Ltd., Katagiri Bldg., Ginza Higashi, Chuo-ku, Tokyo, Japan.

**Present address: Department of Botany, University of Washington, Seattle, Washington 98105.

Alleaume, Nadine. Division of Biology, California Institute of Technology, Pasadena, California. Vital staining of Drosophila eggs.

Flies injected with trypan blue in 0.4% Drosophila ringers solution lay eggs with blue yolk (K. Sander and H. Vollmar). We found feeding to be more convenient than injection for producing stained eggs. Adult wild-type flies were fed for three days on medium containing either Nile blue (0.01%), Toluidine blue (0.02%), Bismark brown (0.5%) or N Phenyl Nile blue chloride (0.05%). The flies were transferred to dye-free medium and about half of the eggs they laid in the following two days were found to contain dye. The first three dyes colored only the yolk. N Phenyl Nile blue chloride led to light pink eggs with pink fat bodies in the developing embryos. With these four dyes the embryos in stained eggs develop normally.

Granholm, N.A. Department of Biology, University of Oregon. Studies of selected spermatocytes in the light and electron microscope.

A technique is available which enables one to observe living Drosophila spermatocytes in vitro and to recover the observed cells for electron microscopy. The technique is patterned closely after one reported by Brinkley and Nicklas for grasshopper spermatocytes (1968).

Pupal testes are dissected under series 11-14 Halocarbon Oil (Halocarbon Products Corp., 82 Burlews Court, Hackensack, N.J.). An intact testis is freed of adhering fat and transfered to a drop of Halocarbon Oil on a clean but otherwise untreated #1 coverslip. The testis is cut using small dissecting knives and smeared over an area of the coverslip, thereby expelling the cells from the testis and spreading the cells into a single-cell layer. If care is taken to spread the cells evenly, keeping the cell layer intact, the cells will adhere to the coverslip. Enough oil is then added such that the coverslip will remain slightly above the surface of the glass slide onto which it is inverted. The coverslip is ringed with VALAP (vaseline + lanoline + paraffin wax with a 50°C melting point, in the proportions 1:1:1) (Mole-Bajer and Bajer, 1968) to hold it in place. Cells are selected and photographed. Cells in such a preparation have been followed from diakinesis through completion of the first meiotic division.

It is necessary to separate the cells from the oil for fixation. This is accomplished through quick-freezing the slide-coverslip preparation in pentane-isopentane (ca. 1:1) cooled in a liquid nitrogen bath. The preparation is then quickly transfered from the pentane-isopentane into the liquid nitrogen. The coverslip is carefully lifted vertically from the slide by prying one corner with a scapel; this is done while the preparation is immersed in liquid nitrogen. It is very important that the transfer of the coverslip into the fixative (3% glutaraldehyde in 0.1 M phosphate buffer, pH 7.2) is done as quickly as possible. The remainder of the schedule is routine: fix 30 min. in glutaraldehyde; wash in buffer 30 min.; postfix in 2% OsO_4 in 0.1 M phosphate buffer, pH 7.2; rapid dehydration in an alcohol series followed by propylene oxide (PO). The coverslip is removed from PO and the surface bearing the cells is quickly flooded with epoxy which has been mixed with PO (ca. 2 epoxy:1 PO). The coverlip is kept at room temperature overnight and then cured at 60°C for at least 24 hours.

The cured plastic with coverslip can now be cemented to a glass slide (Eastman 910 Adhesive; Eastman Chemical Products, Inc., Kingsport, Tenn.). The preparation is examined and the desired cells located. The coverslip is removed by placing the cemented preparation on a block of dry ice for ca. 10 min. and carefully prying the coverslip with a razor blade. In general, the coverslip will be removed in a few large fragments. The plastic may now be divided, while cemented to the slide, to recover the cells individually. It is technically difficult to separate more than 3-4 cells. Initial rough trimming is easily done at this time also. Desired portions of the plastic are removed from the slide and cemented to clear plastic pegs for final trimming and sectioning. It is possible to examine, using either a high power dissecting scope or a compound microscope, the plastic during sectioning in order to determine when one has sectioned the cell completely.

The technique can be used to recover intact, fixed cells for further histochemical studies, also.

References: Brinkley, B.R. and R. Bruce Nicklas. Ultrastructure of the meiotic spindle of grasshopper spermatocytes after chromosome micromanipulation. J. Cell Biol., 39 (2) part 2, 16A (1968). Mole-Bajer, J. and A. Bajer. Studies of selected endosperm cells with the light and electron microscope: The technique. La Cellule 67, 257 (1968).

This work was supported by P.H.S. Health Science Advancement Award 1 SO4-FR 06027-02.

Kirschbaum, Werner F. Argentine Catholic University, Faculty of Agrarian Sciences, Buenos Aires, Argentina. Fast sexing of larvae.

A successful method for sexing a high number of Drosophila larvae and of other diptera consists in placing them in a drop of water between two slides and looking at them in this motionless condition with a stereoscopic microscope using transparent illumination. Several larvae can be placed in a row on each slide and the right position to inspect the gonads of each larvae can be accomplished by moving gently the upper slide to the sides.

Grant, B.S., G. Bean and W.L. Harrison. College of William and Mary, Williamsburg, Virginia. A Drosophila eclosion fraction collector (DEFC).

Eclosion rhythm studies require an around-the-clock method for recording the time-of-day when flies emerge from the puparium. Periodic manual transfer from cultures proves far too inconvenient and exhaustive to personnel, particularly in long term experiments. An automated method employed by Pittendrigh and his associates at Princeton (see Zimmerman, et al., J. Insect Physiol., 14) results in the death of harvested adults. Since our experiments require living flies, a different method of collection had to be devised.

The system we have developed provides for the automatic hourly collection of newly emerged live flies without the necessity of handling individual pupae. Eggs are deposited on completely filled 1-1/4 X 1-3/4 inch plastic food cups. An open ended 1-1/4 X 4 inch clear plastic chimney is taped to the food cup to form a large vial. After numerous dark pupae appear on the inner surface of the chimney, it is removed from the food cup and attached to the DEFC (see e in Figure). (In order to avoid sampling only early developers additional clean chimneys should be subsequently placed on the food cups until the larvae are depleted.) Adults are continuously removed from the chimney once they have freed themselves from their pupae cases. This is

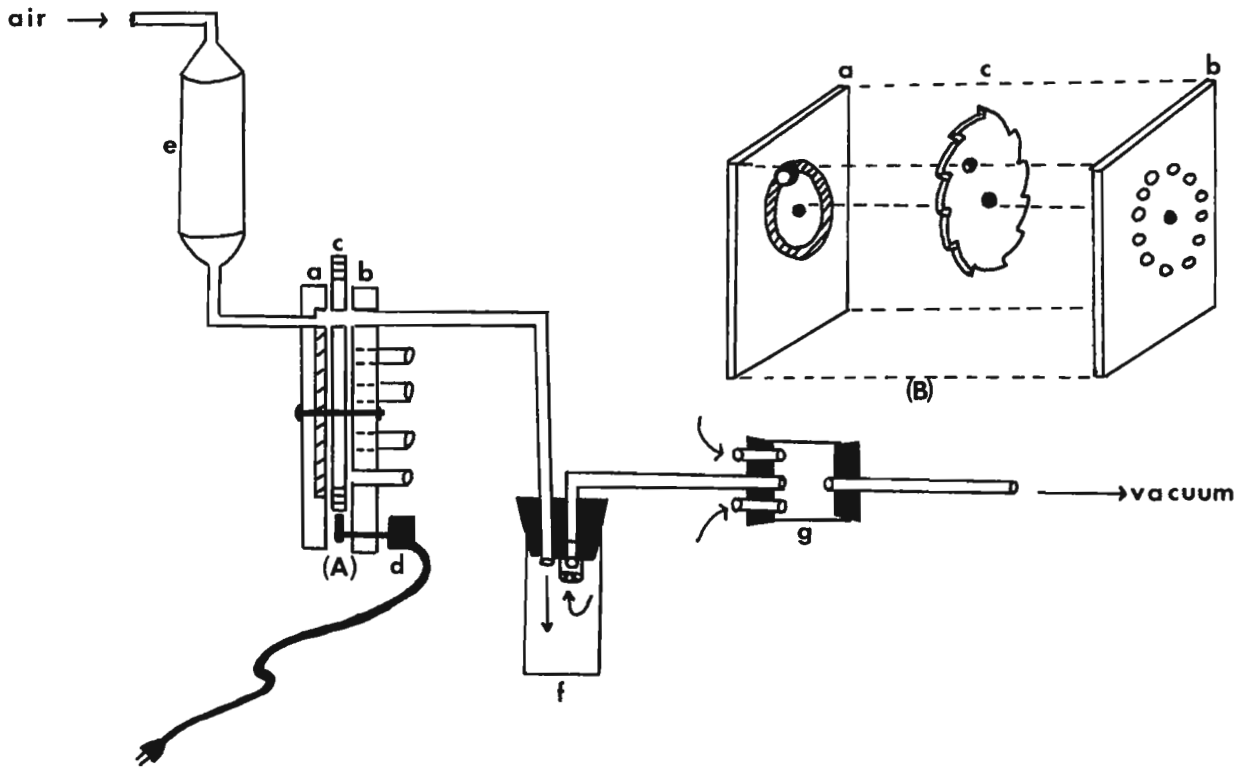
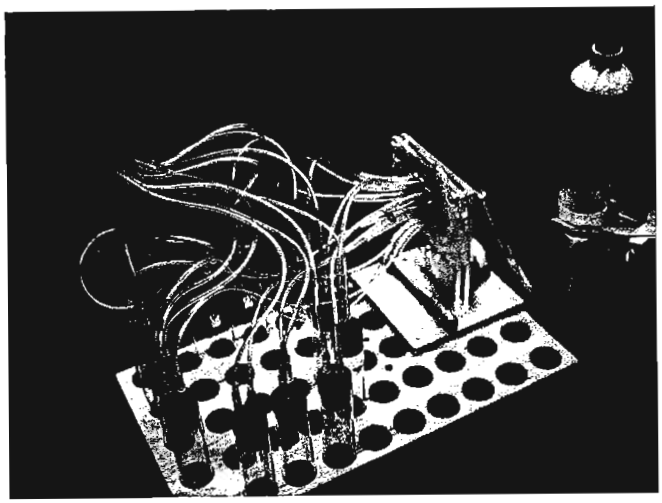


Figure 1. Drosophila eclosion fraction collector. (A) Cross-sectional diagram of sorter (B) Exploded view of sorter. Explanation in text.

accomplished by an air current generated from a carefully balanced combination of air being pumped into the upper end of the chimney and withdrawn at the lower end via a vacuum pump. This aspiration should be fairly gentle lest the flies be injured in transit, but the air stream within the chimney must be of sufficient force to effectively remove adults after eclosion. The flies are then drawn through an exhaust tube to the sorter.

The heart of the DEFC is the sorter. It is constructed from Lucite and consists of a 3-1/4 inch geared wheel (c) sandwiched between two 4 X 5-1/2 inch plates. Lightly greased paper gaskets actually separate these three pieces for the purpose of lubricating the wheel and to insure a reasonable air seal. The front plate (a) has a single 1/4 inch hole drilled through it which intersects a circular grooved track on its inner surface. The back plate (b)

has a ring of twelve 1/4 inch equally spaced holes, each of which is connected by tubing (1/8 inch inside diameter) to one of twelve collecting vials (f). The holes in plate b communicate with the track in plate a by way of a single hole through the geared wheel which permits only one hole in plate b to be open at any given time. Flies from the chimney enter the track in plate a through the single hole in that plate and then are drawn through the opening in the gear wheel to be shunted down one of the collection holes in plate b. There is only one route open at any given hour to a collection vial. The wheel is turned 1/12th revolution every hour by a lever extending from the axle of a small one-RPH electric motor (d). This turning time takes about seven minutes which allows a given vial to be open for the major portion of the hour. All twelve vials are connected to a single vacuum block, but appreciable pressure is present only in the particular vial open to traffic.



Our machines were designed to run unattended for twelve hours. At the end of this period, the collection vials are replaced with an empty set. The flies collected from the preceding block of time can then be counted and sexed for use in prescribed matings. The machine, of course, can be modified to run for longer periods of time by altering the size and numbers of teeth on the geared wheel and the corresponding number of collecting holes in plate b to accommodate the grace period of virginity for the species under investigation or for, perhaps, a specified photocycle in the laboratory.

Supported by NSF-GU-3111-M.

Paika, Inder J. University of Nebraska, Lincoln, Nebraska. Application of air drying technique to the preparation of chromosomes in testes of adult males.

Crozier, 1968 (Stain Technology, 43: 171-173) has described an air drying technique applied to the preparation of chromosomes of Drosophila larval ganglion cells.

His method has been extended to testicular material of adult flies of D. affinis. Adult males were injected with a small quantity of 0.1% colcemide in Bodenstein's solution and after 1½ to 2 hours the testes were dissected out in 1% sodium citrate solution and left there for 15 to 20 min., after which the testes were fixed in freshly prepared acetic-methanol (1:3) for about 30 mins. After fixation the material was put in a drop of 60% acetic acid on a clean warm slide for about 30 seconds. A very small drop of acetic-methanol (1:3) was then added to the dissociated tissue and the slide tilted to regulate spreading and allowed to dry in the air. Staining was done with lactic-acetic-orcein (2gm. synthetic orcein in 50 ml. glacial acetic acid and 50 ml. 85% lactic acid) at 45°C. for about one hour. The slide was then placed vertically in acetic-ethanol (1:3) until the coverslip dropped off. After de-hydration with 95% and absolute ethanol the preparation was mounted in euparal.

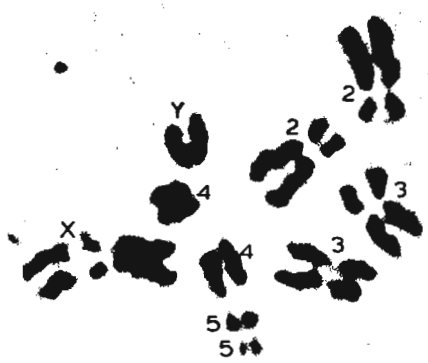


Fig.1. Phase contrast photomicrograph of primary spermatocyte of D.affinis (Chadron State Park, Nebraska) prepared by air drying technique applied to adult testes.

Félix, R. Programa de Genética y Radiobiología, Comisión Nacional de Energía Nuclear, México City, México. A food medium dispenser device for filling vials.

A very inexpensive to build device for rapid dispensing of food medium to vials, with complete control of the operator is described in the present technical note. The unit is waterjacketed to insure even heating which avoids the clogging of the delivery tube. The liquid medium is poured into the unit after filling the space between the double wall of the dispenser with hot water with the help of a funnel inserted through a hole in the top of the unit. It is not necessary to repeat this operation, as the unit can be refilled several times with food medium without renewing the hot water.

Flow is stopped by pressing the latex or rubber delivery tube with a Mohr pinchcock. The medium is quickly poured into the vials actioning the pinchcock with the right hand, at the same time the row of vials is moved along a track by pushing the first one in the row with the left hand.

Rarely the devlivery tube gets clogged, in such a case a thin glass rod pushed from above may be used to remove the plug through the outlet. Most of the dimentions of the apparatus shown in the figure are not critical because the temperature of the food medium is easily maintained by hot water in a waterjacketed unit. This low cost apparatus is easily constructed and useful when hundreds or thousands of vials are used in a Drosophila laboratory.

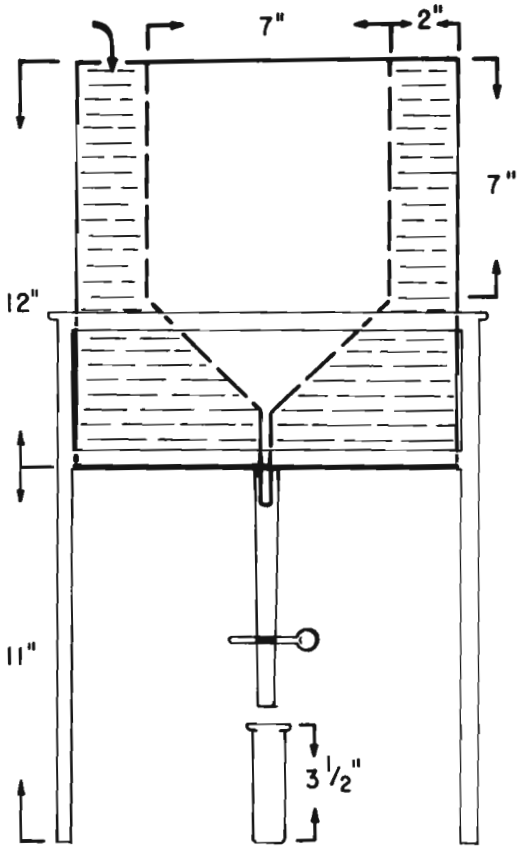
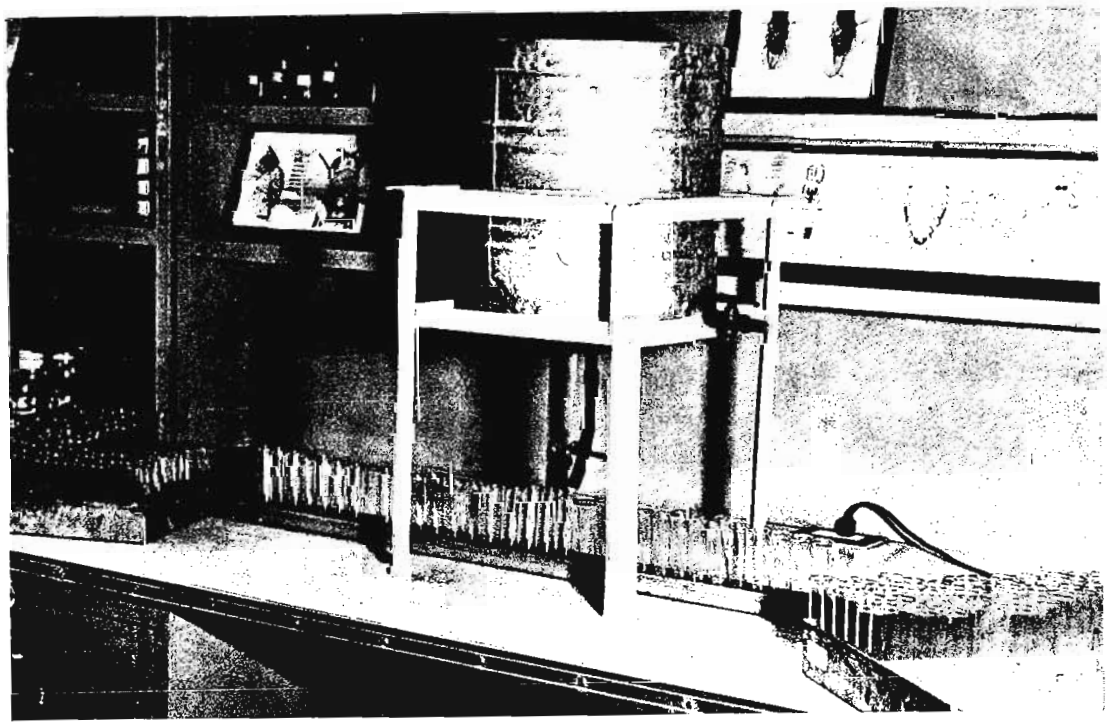


Diagram of the dispenser device.

The dispenser with a row of vials on a track during the filling operation



Shivertaker, L.W. Department of Pathology, West Virginia University School of Medicine, Morgantown, West Virginia. The microinjection of Drosophila larvae.

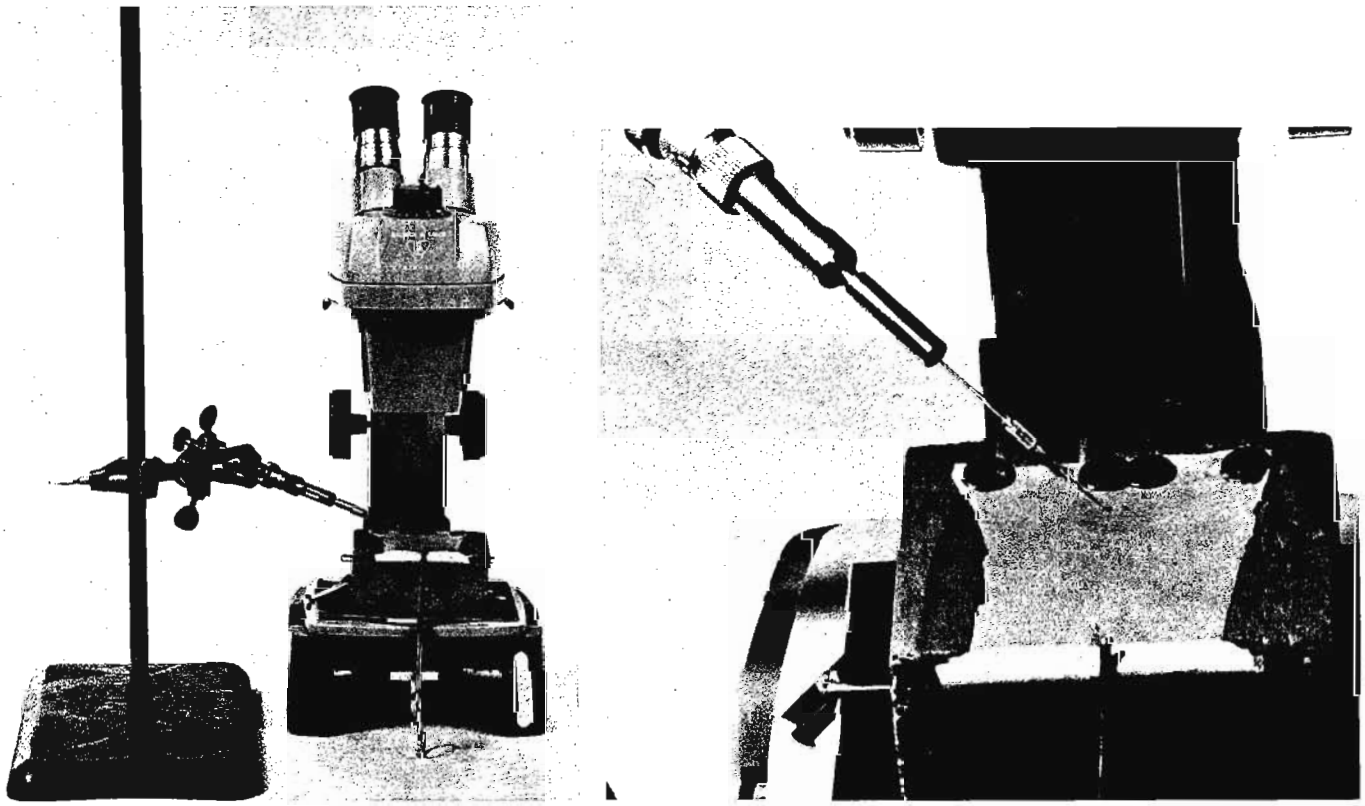
A simple, accurate, microinjection technique is described for the injection of small species such as larvae. This method renders a considerably higher survival than previously described techniques.

Several lines of evidence suggest that the overall pattern of metabolism, growth, and differentiation are greatly influenced by a variety of exogenous agents. In the use of small species, the introduction of experimental compounds by injection methods has been fraught with many technical difficulties. This investigation was undertaken to develop a simple, accurate microinjection technique. The method has been used for the larvae of D. melanogaster, but can be adapted for any small species.

Prepupal larvae of D. melanogaster grown on a banana-agar-yeast medium at 27°C. were utilized. The apparatus consisted basically of three parts: (1) a wooden stand, (2) a microinjecting syringe, (3) a dissecting microscope.

A modification of Burdette's method (1965) is used to steady the larvae during injection. A small piece of rubber sheeting is stretched across a hole in a wooden stand. A small perforation of selected size made in the rubber sheeting serves to hold the larva firmly but without injury. The rubber is fastened by one end to the wooden stand by thumbtacks. The loose end is clamped by a hemostat to allow easy stretching of the rubber sheeting. The larva is placed vertically through the rubber sheeting with its ventral surface toward the needle. This method allows adequate manipulation of the larva.

Figure (1): The relationship of the injecting needle to the wooden stand



The injection apparatus consists of two parts: (1) a glass needle, and (2) a spring-loaded microinjecting syringe. The needle is made by drawing a pyrex capillary tube, 1 mm in diameter, to a fine point by the use of an automatic pipette puller (Dave Kopf Vertical Pipette Puller - model 700 B using an electric coil). If the needles are siliconized (Siliclad: Clay-Adams Co.) before bevelling, penetration appears to be less traumatic. The point is bevelled and sharpened with fingernail clippers or similar bitting instruments,

which give a clean sharp break. The external diameter at the tip ranges from 40-78 micra. This type of needle is much less traumatic than the 30 gauge needle used by Burdette (1965). With a series of polyethylene adapters, the needle is connected to the spring-loaded microinjecting syringe (Hamilton Co., Whittier, California) which delivers from 0.1 lambda to 10 lambda of solution. The apparatus is positioned as shown in Figure 1. The wooden stand is placed on the stage of the dissecting microscope. The microinjecting syringe is then fastened securely to a ring stand by a clamp. The larva is manipulated by teasing needles and positioned by moving the wooden stand instead of the syringe. The larva is then brought to the needle and the tip of the needle is allowed to enter the posterior ventrolateral portion of the larva. Care must be taken to avoid breaking the injecting needle by keeping it straight with the teasing needles. After the tip of the injecting needle enters the larva, a spring loaded plunger of the syringe is released and the required amount of solution is injected. Effectiveness of the injections can be demonstrated by the use of methylene blue. Rapid diffusion throughout the larva is observed with little or no leakage of the dye. With practice, injections can be done every three to five minutes.

Mortality of the injection procedure occurs within the first four hours after injection. Once the larva enters full pupation after injection, death, if it occurs, is probably a result of the solution injected and not the technique employed. In one series, 135 larvae were injected of which 104 larvae entered pupation. This method, therefore, renders a survival rate of 77%.

Reference: Burdette, W. and R. Anderson, Genetics, 51: 625 (1965). I appreciate the assistance of Enid Gilbert, M.D., Warren Pistey, M.D., and Judith R. Hildebrandt, Ph.D. The work was supported by an Institutional Research Grant, School of Medicine and Dentistry, West Virginia University, and the Lederle Foundation.

New address as of June 1, 1969: Meadowbrook Hospital, East Meadow, N.Y. 11554.

Doane, W.W. Yale University, New Haven, Connecticut. A quick and easy method for rearing large quantities of 'clean' larvae.

When handling large numbers of larvae for biochemical studies or genetic screening programs, it is desirable for their ages to be synchronized and for them to be free of food debris at harvest time. We have been using a quick, easy method for rearing 'clean' larvae that eliminates the need for cooked food medium or agar and yet provides an adequate diet, judging from larval weight and the minimum in its variability when larval ages are properly timed.

Larvae are simply reared on an aqueous paste of brewer's yeast (20%) and cane sugar (10%) that is spread over a 1/4 to 1/2 inch thick foam plastic pad ('plastafoam', or polyurethane of the sort sold for mattress pads). This sits in the bottom of a plastic box or crisper. A ventilation hole at one end of the box is closed with a cotton plug. The paste is made by mixing sterile, aqueous stocks of yeast and sugar. To 100 ml of paste, 1.1 ml of stock Tegasept (10 g/100 ml 95% ETOH) may then be added. However, putrefaction of the yeast occurs when Tegasept is used as inhibitor. Therefore, to prevent an unpleasant odor, use of the mixture of phosphoric and propionic acids described by Lewis (DIS 34: 117) is recommended. Stock food paste stores well under refrigeration; plastic pads may be cleaned, autoclaved and reused. For sterile rearing conditions, additional nutrients may be added to supplement the partially degraded yeast.

Pairs of mature, well-fed adults are released into food boxes for egg deposition over given time intervals; the number depends on the box size and species used. They are removed through the hole in the container by means of a tube attached to a vacuum cleaner and equipped with a cotton gauze bag for catching flies. The same adults may be used repeatedly. The number of larvae that develop cannot be precisely predicted, but should the food appear to be running out, more paste may be poured or spread onto the foam pad.

This method is particularly successful for rearing larvae of D. hydei which tend to restrict themselves to the upper layers of ordinary culture media. The larvae feed mostly on the surfaces of the plastic foam and are easily washed off with water at the time of sacrifice into a collecting vessel. Excess food and waste products are removed by repeated rinses with water which are decanted off, leaving the clean larvae in the bottom of the vessel. Pouring the last rinse water with larvae through a mesh may prove useful before blotting them dry. For D. melanogaster, whose larvae are considerably smaller than those of D. hydei, the method should prove most useful in harvesting late third instar larvae on the verge of puparium formation. The plastic box may be inverted with the food pad placed on the lid and the larvae collected from the walls of the bottom section as they climb up.

(Supported by NSF Grant GB 7106.)

Finnerty, V., D.L. Baillie and A. Chovnick. University of Connecticut, Storrs. A chemical system for mass collection of virgin females or males.

A purine selector system has been devised to kill flies lacking xanthine dehydrogenase (XDH) activity (Glassman, E., Fed. Proc., 24: 1243, 1965). The purine (Sigma Chem. Co., P6880) is used as an aqueous solution, generally 0.2%. Parental flies are allowed to remain in fresh culture bottles for 2-3 days. Immediately after transfer, 1-2 ml. of 0.2% purine is evenly distributed over the surface of the already formed culture. This method is useful since it allows stocks to be maintained indefinitely by the usual transferring. When one sex is required the chemical selector is simply applied to the required number of cultures in the manner just described.

1. For attached-X virgin females: such females, with any desired combination of markers (except those with drastically reduced or no XDH activity) are kept with ma-1 males. The purine system will kill ma-1 males before eclosion leaving only virgin females.

2. For virgin males: method 1 is reversed so that desired males are kept with homozygous ma-1 attached-X females.

3. For free-X heterozygous virgin females: a variation of method 1 is potentially useful where virgin females are needed for (X or autosomal) fine structure analysis. Where the heterozygous female, a^X/a^Y, are required, virgin females of the type a^X/a^X, homozygous for ma-1, are crossed to a^Y males. After treatment, the daughters, being ma-1/ma-1+ having normal levels of XDH activity, will survive. The sons, being ma-1, will be eliminated.

Similar selector systems employing ry with X-translocations may be utilized in situations where ma-1 would be undesirable.

Since the purine system may be used for a variety of genotypes and culture conditions, the concentration of purine may have to be adjusted to maximize the results. We have noted that dilute aqueous purine is subject to destruction by mold and therefore make up fresh solution with clean glassware as required. Any unused solution is kept refrigerated. The purine concentrations described have been successful with our medium (cornmeal, agar, molasses, karo, brewers yeast, tegosept) used in half-pint creamers, but may well need adjusting when used with different media or with different volumes of media.

Leuthold, U. and Würgler, F.E. Swiss Federal Institute of Technology, Zürich, Switzerland. Egg collection from individual females of D. melanogaster.

As a standard procedure for the registration of X-ray induced damage in stage 14 oocytes virgin females are irradiated. The females are then mated with unirradiated males and allowed to lay eggs for 24 hours. With this procedure two difficulties arise: (a) variation in control mortality resulting from eggs deposited by non-inseminated females and (b) heterogeneity of oocyte stages tested if some females deposit large numbers of eggs. To avoid these difficulties the following modified method is used:

Females from uncrowded standard cultures[1] are collected as virgins and kept for 4 days in "feeding bottles" with well-yeasted medium[1]. On the 5th day the females are irradiated and mass mated with about 2 days old males in empty bottles in a dark room. About twice as many males as females are used. After 2 to 3 hours the females (which do not lay eggs in the empty bottles) are separated from the males and put individually into special egg collection arrangements in a room of 25°C. and 96% relative humidity. Each egg collection arrangement consists of a glass beaker (5 cm diameter, 9 cm high) standing upside down on a thick blotting paper and a small plastic bowl (1.5 cm diameter, 1 cm high) placed in a central position beneath the beaker. The bowl is two-thirds filled with fermenting egg laying medium[1]. A large area of the smoothed surface of this medium is covered with black paper soaked previously in 1% acetic acid. Since most of the liquid will be absorbed by the medium, more acetic acid is dropped into the paper. Females anaesthetized by CO_2 are brought individually under each beaker. As the black paper in the bowl is the only wet place in the arrangement the flies will deposit most of the eggs on it. Occasionally some eggs may be found on the free surface of the medium or on the wall of the bowl. During the egg collection period groups of 24 of these arrangements are brought under a light-tight cover which prevents the flies from disturbance by light changes in the experimental room. With this method an average of about 20 eggs per female are deposited within 3 hours. From

these eggs the rate of radiation induced dominant lethals can be determined.[2]

A test for insemination of the females is done in the following way: At the end of the collection period the females are put individually into small culture tubes with standard medium. Because younger stages of oogenesis are much less sensitive to the induction of dominant lethals by X-rays, even strongly irradiated females, which have been inseminated, will deposit viable eggs after some time. Examining these cultures for progeny after 6 to 7 days allows for the detection of non-inseminated females by the lack of larvae or pupae in the tubes.

This egg collection method initially developed for the stock "Berlin wild" has been successfully adapted to a strain (XY/XY) with retarded maturation of the flies and reduced rate of oviposition. In this case 6 days old virgin females were used and the mating period as well as the egg collection period have been prolonged to 4 hours. With similar modifications the method has been used for experiments with a triploid strain and for tests where inseminated females were irradiated (Lütolf, Graf, unpublished).

The method can also be adopted for dominant lethal tests after irradiation of mature sperms in males. In this case single irradiated males are mated for a few hours with single females in small empty tubes. Then the females are put individually into the egg collection arrangements. Egg collection can be extended to many hours (e.g. overnight) since the cells to be tested have been transferred by a single copulation to the females, and no difficulties from differential radiosensitivity of various cell stages can appear.

Work supported by Schweizerischer Nationalfonds zur Förderung der wissenschaftlichen Forschung und Jubiläums-Fonds 1930 der ETH.

References: 1. Würgler, F.E., Ulrich, H. and Spring, H.W. Experientia 24: 1082, 1968. 2. Würgler, F.E., Petermann, U. and Ulrich, H. Experientia 24: 1293, 1968.

Williamson, J.H. and P. Stubblefield. University of California, Riverside, California. An efficient method of collecting homogeneous samples of stage 14 oocytes.

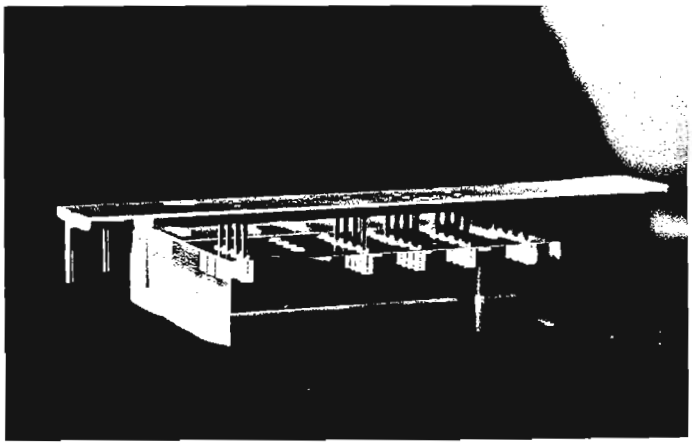
A cursory review of the pertinent literature will convince anyone that previously used methods of collecting samples of stage 14 oocytes are based on hearsay. Usually females are aged several days, mated, and the number of eggs per female limited to a maximum of 24. This practice is based on the assumption of twelve ovarioles per ovary and one stage 14 oocyte per ovariole. Our technique (borrowed from D.R. Parker) is to rear females in uncrowded cultures, collect virgins at twelve-hour intervals, and to store females for four days on new culture medium sprinkled with live dry yeast. Females are then lightly etherized, put into gelatin capsules, allowed to recover, irradiated and mated without etherization to males that had also been aged on yeasted medium. Matings were made on food warmed to room temperature and held at 25° C. with lights for twelve hours at which time all flies are discarded. Egg counts from individual females revealed that many produced more than 24 eggs in twelve hours. Subsequently two samples of 30 C(1)RM, y v bb / B^{S}Yy+ females, one group aged for four days, the other five days, were dissected and the number of stage 14 oocytes per female determined. The 4-day old females averaged 43.4 stage 14's (range: 22-68) and the 5-day old females averaged 45.8 stage 14's (range: 24-74). In most cases each ovariole contained two or three stage 14's and all ovaries were made up of 16 or 18 ovarioles. Ovarioles with three stage 14's contained no additional oocytes of intermediate stages, and only a few very early stages. A third group of thirty females of the same genotype, 4 days old, produced an average of 25 eggs per female in a twelve-hour interval (range:0-68).

Wild type females from a cross of Canton-S and Guasti-36-10 were collected and aged 4 days as described above. Fifty-nine females were dissected and averaged 84.4 stage 14's per female (range: 52-111). Sixty-three females from a cross of Oregon-R and Guasti-36-10 averaged 70.2 stage 14's per females (range: 39-104). Apparently strains differ in the rate of egg production and each experimental strain should be analyzed accordingly. It seems reasonable that as long as the number of eggs laid in a twelve-hour interval does not exceed the number of "stored" stage 14 oocytes, one can assume that a homogeneous sample is obtained.

Adamkewicz, S.L. and R. Milkman. The University of Iowa, Iowa City, Iowa. A multiple sample homogenizer/applicator for cellulose acetate electrophoresis.

Up to 20 samples per 1" x 6" cellulose acetate strip are permitted by the use of the following device, which is essentially a multiple-well lucite block and a brass strip fitted with 20 complementary stainless steel rods and 3 guide rods. All rods are flat ended.

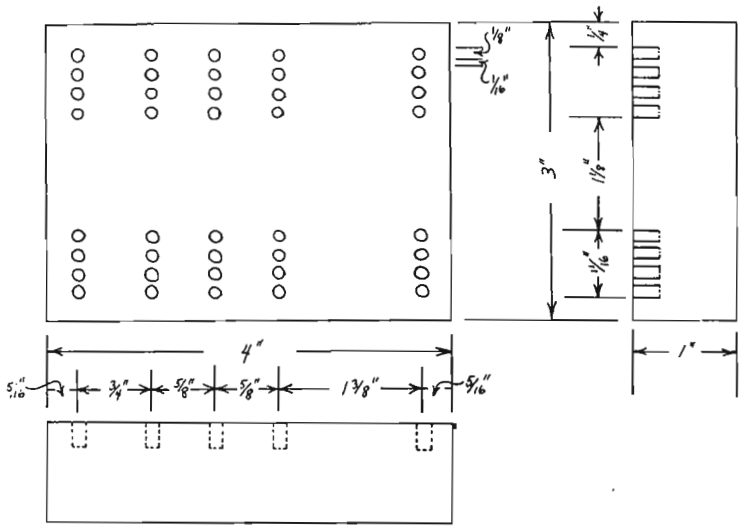
Flies are placed in the wells with buffer and homogenized by the rods. The homogenate is applied to the strip by resting the rods on it. No additional pressure is used. The strip is then electrophoresed and stained according into common procedures. We use the Gelman system.



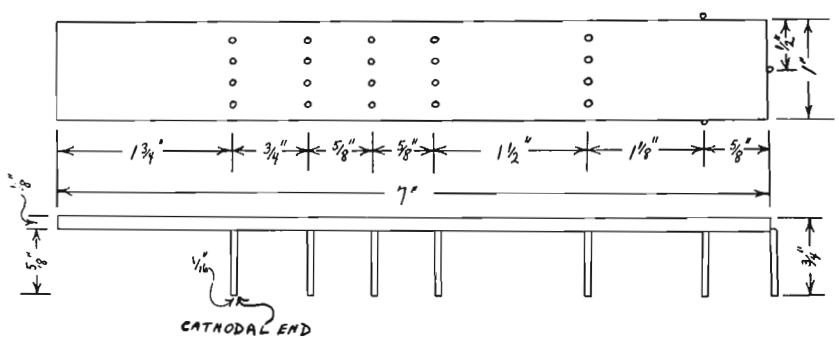
Each lucite base has two sets of twenty holes and thus fits two brass applicators at once. The arrangement compensates for net migration differences due to the unavoidable evaporation that occurs even at low temperatures and causes bulk flow toward the center. Resolution is not impaired.

Alcohol dehydrogenase, α-glycerophosphate dehydrogenase, and hemoglobin have been run successfully; a variety of animals (12 major classes) have been used in addition to Drosophila. One person, unassisted, can run hundreds of samples per day. The ease of application is emphasized.

In a double blind test, using D. melanogaster α-glycerophosphate dehydrogenase, 391 of 398 spots were scored correctly.



Lucite Base Above
Brass Applicator Below



EDITOR'S NOTE: Starting on the next page the reader will find a new account of the details of meiosis in the amphibians. This is such an extraordinary story, with universal appeal, that the reader will quickly understand why the editor did not hesitate to include this non-Drosophila work. This does not, however, represent a permanent change of policy.

Basden, E.B. Institute of Animal Genetics, Edinburgh, Scotland.
Attitude of killed melanogaster adults.

Specimens after etherisation, or after killing, pinning and setting, usually do not expose the minute discal sternopleural hairs, these being overlaid by the drawn-up first coxae, and the wings are variously positioned. If specimens are required for pinning, or for scoring or measuring, it will save time if they die (or are anaesthetised) with appendages conveniently extended to allow maximum easy examination. Therefore a few reagents were tested for attitude of killed flies.

Live adults from a selected (da Silva) Kaduna strain were sucked into 3" x 1" corked vials, the corks dampened inside with the reagent, and the flies allowed to die with the vial on its side.

Acetone: Legs half-stretched, rarely drawn up. 1st coxae raised. Wings flat, extended.
Amyl acetate: Hind legs semi-stretched, 2nd legs sometimes drawn up. 1st coxae 50% raised. Wings up or flat.
Carbon tetrachloride: Mid and hind legs stretched. 1st coxae rarely raised. Wings up.
Chloroform: Legs usually drawn up. 1st coxae rarely raised. Wings 50% up, 50% down.
Ether: Legs mostly drawn up. 1st coxae rarely raised. Wings up or flat.
Ethyl acetate: Mid legs stretched, sometimes upraised. 1st coxae 50% raised. Wings up.
Xylol: Hind legs stretched, often up-raised, 2nd legs stretched. 1st coxae sometimes raised. Wings up.
75% Ethyl alcohol + 5% glycerine (total submersion): Legs various, usually drawn up. 1st coxae 50% raised. Wings up, flat or down.

Acetone allowed best examination and the wings did not hide the scutellum. The effects of the same reagents on different species have not been studied but with xylol D. subobscura and D. obscura adults usually had the wings down (thus obscuring some pleura and legs) and the male genitalia were well extruded.

Ward, Calvin L. Department of Zoology, Duke University, Durham, North Carolina.
Handling of single fly homogenates for acrylamide gel disc electrophoresis.

Acrylamide gel disc electrophoresis of single Drosophila poses several technical problems. The inclusion of the small amount of material in a sample gel according to the technique of Davis (1964) is time consuming, and the layering of the sucrose homogenate through the buffer (Wrigley, 1968) on the surface of the stacking gel is tedious and difficult with such small amounts of material. We found the following technique to be fast and effective. Glass tubes 8 1/2 cm. long are filled to 7 cm. with the separating gel and layered with water. After polymerization, the water is removed and 1/2 cm. of stacking gel is added and water layered. Upon the completion of photo-polymerization of the stacking gel, the water is removed and the tubes inserted into the upper bath. Each stacking gel is immediately layered with 25 lambda of 40% sucrose. Homogenates are prepared by the technique of Johnson (1966); however, we use individual Lucite slides, 3" x 1" x 3/8", each drilled with a single hole, 1/4" in diameter and 1/4" deep. A small amount of powdered glass and 25 lambda of 40% sucrose are placed in each cavity. To facilitate homogenization the larva is first torn open on the surface of the slide. At this stage the salivary gland may be removed for cytological analysis if desired. The larva along with hemolymph is wiped into the cavity with a single layer of Kimwipe handled with jewelers forceps: the material is homogenized with a Pyrex rod driven by a variable speed motor. The sucrose homogenate plus powdered glass and chitinous remains are absorbed by a pad of four layers of Kimwipes (punching several layers at a time causes the edges to adhere) handled by jewelers forceps: filtration is unnecessary. The homogenate saturated paper is inserted into the sucrose layered on the stacking gel. Sufficient buffer is added with a Pasteur pipette to fill the tube and finally the upper bath is carefully filled. This method of sample application has given good reproducible results in esterase studies on Drosophila melanica.

References: Davis, B.J. Ann. Acad., Sci. 121: 404. Johnson, F.M. D.I.S. 41: 193. Wrigley, C. Sci. Tools 15.

Kezer, J. University of Oregon, Eugene, Oregon. Observations on salamander spermatocyte chromosomes during the first meiotic division.

Introduction: Salamanders have long been known as a source of superb material for the study of meiosis. This paper will be devoted to a presentation of the behavior of the chromosomes of salamander spermatocytes during the first meiotic division from leptotene through prometaphase, as seen in longitudinal sections through the testis and in squash preparations of spermatocytes. The chromosome preparations have been obtained from various species of the Plethodontidae, a family of salamanders widely distributed throughout the United States and Latin America.

The structure of the testis of plethodontid salamanders has been described by Kingsbury (1902) and Burger (1937). The latter paper contains an excellent description that should be consulted for detailed information. A typical plethodontid testis has a cylindrical structure and consists of a longitudinal duct surrounded by ampullae which are connected by short ducts to the main longitudinal duct. Primary spermatogonia are clustered about the short ducts of the ampullae and these cells, along with the duct system, constitute the persistent structures of the testis. The reproductive cycle is an annual event in the temperate zone plethodonts. After the ampullae have been emptied of their sperm, the testis is built up by proliferation from the persistent primary spermatogonia, so that at a particular time during the year, the ampullae become filled with secondary spermatogonia that are available for transformation into spermatocytes.

The meiotic divisions appear first at the posterior end of the testis and spread through the gonad during a period of about two months as a caudocephalic "wave". As a consequence, it is possible to secure salamanders in which the meiotic events are present in the testis as a sequentially ordered series: the ampullae at the extreme anterior end will contain spermatocytes in leptotene, slightly more posterior ampullae will be filled with zygotene spermatocytes, and all other stages of meiosis will follow in sequential order through spermatids to developing sperm in the extreme posterior ampullae. This orderly progression of events within the gonad makes possible interpretations and identifications that are difficult in the mixed-up cells of a squash preparation. To exploit such a situation, longitudinal sections of entire testes can be used to determine the sequence of meiotic events, supplemented with squash preparations of spermatocytes to reveal the contents of entire nuclei in a spread-out condition.

The accompanying plates of photomicrographs have been prepared with the above ideas in mind. Photos taken from longitudinal sections of the testis of Batrachoseps attenuatus (California slender salamander) are presented along the left margins of the first three plates. Extending from these to the right, are photomicrographs of squash preparations of spermatocytes corresponding in stage of development to those seen in the sections. The squashes were obtained from a variety of plethodontid species, identified in the legends that accompany the plates. The photos of sections illustrate the sequence of meiotic events present in the Batrachoseps testis beginning with the leptotene spermatocytes in the extreme anterior ampullae and ending with the ampullae about midway in the testis, in which the latest meiotic cells are in prophase of the second meiotic divisions.

Materials and Methods: Longitudinal sections were obtained from testes fixed in Sanfelice's fixative, paraffin embedded, sectioned at 10 micra and stained with iron hematoxylin. Acetic-orcein squashes were prepared by a technique modified from LaCour (1941). A small piece of fresh testis (about 2 mm in diameter) was dismembered in a drop of 2% orcein dissolved in 45% acetic acid. A coverglass coated with dried Mayer's egg albumen was placed over this suspension of stained cells, the slide inverted over absorbent paper and the cells forcefully squashed by pressure on the back of the slide. The coverglass, with cells embedded in the film of albumen, was soaked free from the slide in 15% acetic acid, dehydrated in absolute ethanol and mounted in Euparal. Testes for the Feulgen squashes were fixed in Clarke's fluid (3 parts absolute ethanol and 1 part glacial acetic acid), hydrolyzed for 10 minutes in N HCl at 60°C, stained for 30 minutes in the Feulgen reagent, dismembered into small pieces and gently squashed in 45% acetic acid. The Feulgen preparations were made permanent by the quick-freeze method of Conger and Fairchild (1953).

Observations and Discussion: Leptotene nuclei, as they appear in sectioned material, are shown in Fig. 1. Figs. 2 and 3 are Feulgen squash preparations of nuclei at this same stage. The severely squashed nucleus of Fig. 3 illustrates the leptotene chromosomes as elongate strands with a chromomeric structure. Centromeric heterochromatin is seen as deeply staining material in some of the sectioned spermatocytes.

Synapsis of the homologous chromosomes is illustrated with remarkable clarity in the zygotene nuclei of Figs. 4, 5, 6, 7, 8 and 9. Synapsis begins simultaneously at both ends of a pair of homologues and proceeds inward toward the middle of the pair, bringing homologous chromomeres together. This mode of synapsis has been observed in amphibians by Beçak, Beçak and Rabello (1967), in connection with their studies of polyploid frogs. The chromosome ends, at which the synapsis begins, are directed approximately toward a part of the cytoplasm in which the centrioles are located, producing the so-called bouquet arrangement of the bivalents that will persist through pachytene. These ideas are illustrated by the isolated zygotene bivalent shown in Fig. 9, in which synapsis has been completed along about two thirds of the length of the pair of homologues. In Fig. 5, synapsis is just beginning, as indicated by the short stretches of more deeply staining bivalent ends along the lower right where homologous pairing has been completed. Fig. 6 illustrates a nucleus in which synapsis is about half completed, and Fig. 8 is a zygotene nucleus so severely squashed that the contrast between the paired and unpaired portions of the homologues is accentuated. Exactly these same events can be seen in the sectioned material of Figs. 4 and 7, in which synapsed portions of the homologous pairs appear as the more deeply staining strands at a particular position within a zygotene nucleus. In Fig. 7, the five nuclei along the right margin of the photomicrograph have completed synapsis and are now in pachytene.

Pachytene nuclei are illustrated in Figs. 10, 11 and 22. Synapsis has brought the two members of a homologous pair into such an intimate association that they appear as a single strand. A salamander pachytene nucleus thus contains the haploid number of bivalent U-shaped loops with their ends oriented toward the portion of the nuclear envelope that is adjacent to the centrioles.

Pachytene is followed by a diffuse stage, illustrated in Figs. 12, 13 and 23. The chromomeres of the pachytene bivalents spin out into Feulgen-positive loops, well shown in Fig. 23, and this process continues until the bivalents disappear, producing a nucleus lacking clearly defined chromosomal strands. It seems that this is a stage during which the DNA that was folded into the chromomeres of the pachytene bivalents is completely spun out into the nuclear sap. Indeed, the spermatocyte diffuse stage may represent a situation comparable to the lampbrush condition of young amphibian oocytes, in which the nucleus is filled with pairs of elongate lateral loops that extend out from the chromomeres of bivalent axes (Callan and Lloyd, 1960).

Feulgen-positive loops can be seen protruding from the chromomeric axes of the zygotene bivalents of Figs. 5, 6, 8 and 9. Moreover, Donnelly and Sparrow (1965) have observed Feulgen-positive lateral loops at zygotene and pachytene in spermatocytes of the salamander, Amphiuma means tridactylum. Thus, loops of DNA extending out from the chromomeres of bivalent axes are present in salamander spermatocytes at least as early as zygotene. It is during the diffuse stage following pachytene that these loops reach their maximum extension, and because of this diffuse distribution of the DNA, it becomes impossible to resolve the bivalents as clearly defined structures. During the diffuse stage, centromeric heterochromatin remains condensed. In the diffuse nucleus of Fig. 23, there are 13 masses of centromeric heterochromatin, two of which are fused, and in the salamander from which the nucleus was obtained, n=13.

The existence of a diffuse stage between pachytene and diplotene would be difficult to determine if one had available only squash preparations with their mixtures of unordered nuclei. The tendency would be to classify as leptotene or pre-leptotene all large spermatocyte nuclei that lack clearly defined chromosome strands. It is when these diffuse nuclei are seen occupying a position in the testis just posterior to pachytene nuclei and just anterior to early diplotene nuclei that the reality of the diffuse stage becomes clearly apparent. And the existence of this stage becomes even more convincing when one can observe, in longitudinal sections, the gradual change from pachytene to diffuse and the gradual appearance of diplotene bivalents from the diffuse nuclei, as shown in Fig. 14, in which diffuse nuclei are at the top of the photo, and early diplotene nuclei in the lower half. A diffuse stage between pachytene and diplotene has been identified in the meiosis of many organisms. A review of the literature is given by Barry (1969).

To produce the diplotene nuclei shown in Figs. 14, 15, 16, 17 and 19, the greatly extended loops of DNA must again become folded into the chromomeres of the bivalent axes. As the diplotene bivalents emerge from the diffuse nuclei, the component bivalent halves, so closely associated at pachytene, now appear separated except at the positions of chiasmata. And as the diplotene stage progresses, the chromosomal duplication that took place during the interphase prior to the first meiotic prophase becomes visible, producing four-strand bivalents such as those shown in Fig. 21.

In Fig. 18, late diplotene nuclei are present at the upper left. The smaller nuclei in the lower portion of the photo are in prophase of the second meiotic division. In Fig. 20, late diplotene nuclei are shown in the upper left, nuclei in interphase between the first and second meiotic divisions are at the upper right and first meiotic metaphase nuclei are in the lower half of the photo.

Fig. 24 is a photomicrograph printed with high contrast to accentuate the short loops that invest the axes of these prometaphase bivalents. This situation is interpreted as indicating that the DNA loops of the diffuse stage are not completely folded back into their respective chromomeres, but remain as short lateral extensions from bivalent axes. Indeed, loops approximately similar to those seen in this photomicrograph can be demonstrated at diplotene, first meiotic metaphase and anaphase and at all stages of the second meiotic division. They appear to be a characteristic feature of salamander meiotic chromosomes.

The "fuzzy" and "whiskery" condition of animal spermatocyte chromosomes has been noted by many investigators, and this condition has been cited as possible evidence for a lampbrush stage in spermatogenesis. Moreover, in a study of pigeon spermatocyte chromosomes, using electron microscopy, Nebel and Coulon (1963) demonstrated lateral loops at pachytene and first meiotic metaphase. Thus, it is possible that the Feulgen-positive loops of salamander meiotic chromosomes are of general occurrence in animal spermatocytes. The big question that remains concerns the meaning of these lateral loops of DNA relative to the overall structure of meiotic chromosomes.

Summary: In salamander spermatocytes, synapsis of the homologous chromosomes begins at their ends and proceeds inwards, bringing the homologous chromomeres into such an intimate association that the resulting pachytene bivalents appear as single strands. The arrangement of the bivalents as U-shaped loops with their ends directed approximately toward the centrioles occurs at the very beginning of zygotene and persists through pachytene. Pachytene is followed by a diffuse stage in which the DNA of the bivalent chromomeres spins out so completely into enormously long lateral loops that the bivalent axes disappear. It is from these diffuse nuclei that the diplotene bivalents emerge.

Feulgen positive lateral loops, springing from chromomeres, can be seen in salamander spermatocytes at least as early as zygotene. The loops reach their maximum extension during the diffuse stage. The formation of diplotene bivalents involves a return of this extended DNA back into the chromomeres of the bivalent axes, but it does not fold in completely, since Feulgen-positive lateral loops can be demonstrated along the axes of salamander meiotic chromosomes until the conclusion of the meiotic divisions.

Acknowledgements: Many of the observations on which this paper is based were made at St. Andrews University, Scotland, during a 1968-69 sabbatical year, working as the guest of Prof. H.G. Callan and Dr. Herbert C. Macgregor. The photomicrographs of Batrachoseps testis sections were made from slides prepared by Prof. Callan for use in the general zoology course at St. Andrews University. The ideas presented here regarding synapsis and the diffuse stage resulted from conferences with Prof. Callan, who has generously given me permission to use them in this paper. The Feulgen squash preparations of Oedipina uniformis were prepared by Dr. Kathleen Church. I am indebted to Mr. Harrison M. Howard for the photomicrograph of Fig. 24.

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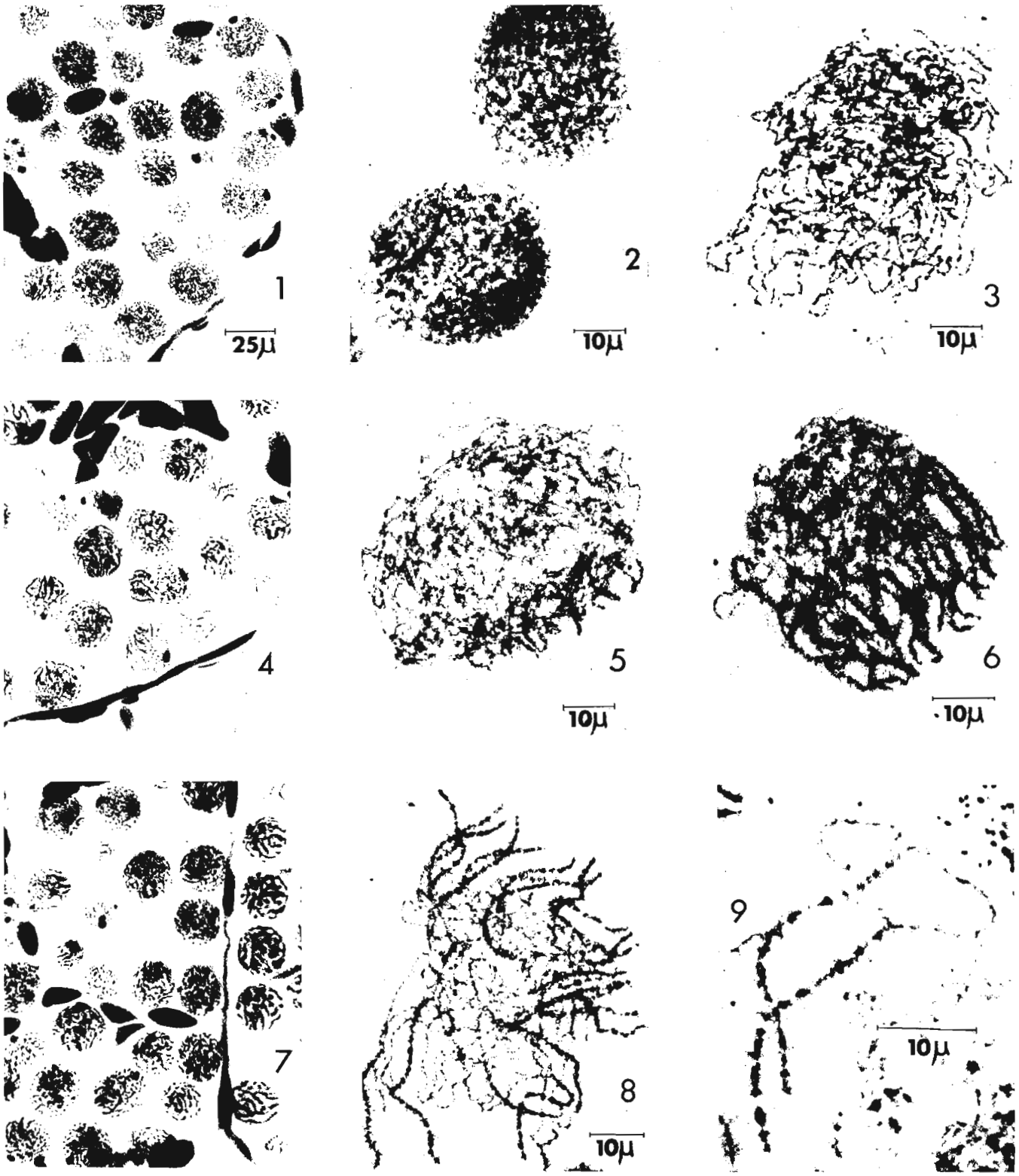

Figs. 1, 4, 7, 10, 12, 14, 16, 18 and 20. Batrachoseps attenuatus. 10μ sections. Iron hematoxylin stain. The scale on Fig. 1 applies to all the photos of this series.
Figs. 2, 3, 5, 6, 8 and 9. Oedipina uniformis. Feulgen squash.
Figs. 11 and 13. Batrachoseps attenuatus. Acetic-orcein squash.
Figs. 15, 17 and 19. Plethodon cinereus. Acetic-orcein squash.
Fig. 21. Oedipina uniformis. Acetic-orcein squash. Fig. 22. Thorius dubitus. Feulgen squash. Fig. 23. Pseudoeurycea werleri. Acetic-orcein squash. Fig. 24. Hydromantes platycephalus. Acetic-orcein squash.


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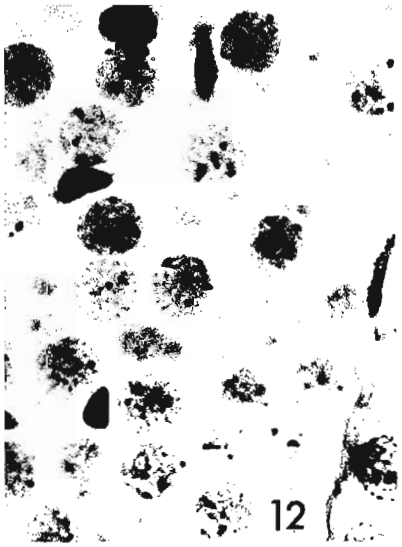
10μ
11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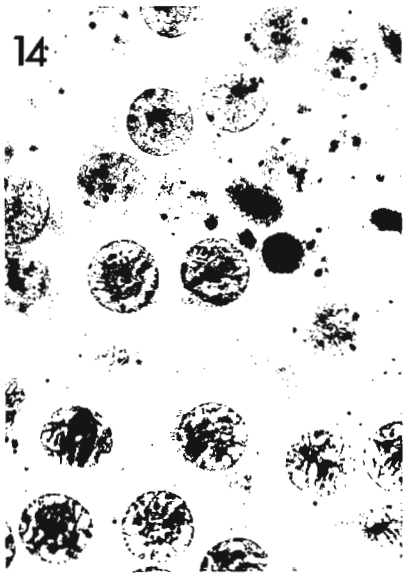
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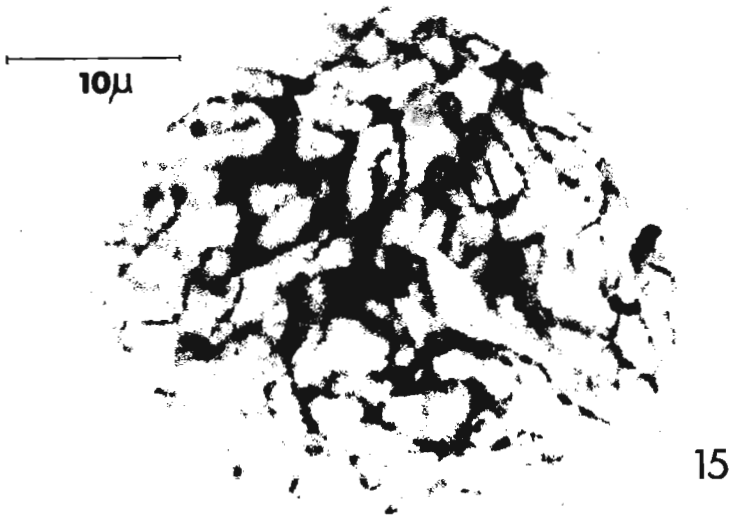
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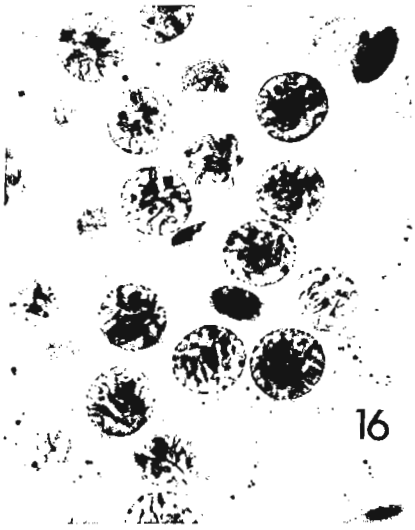
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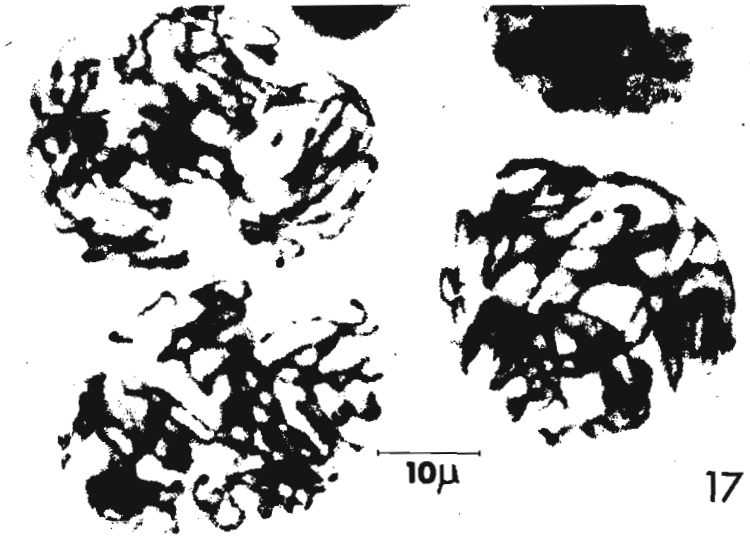

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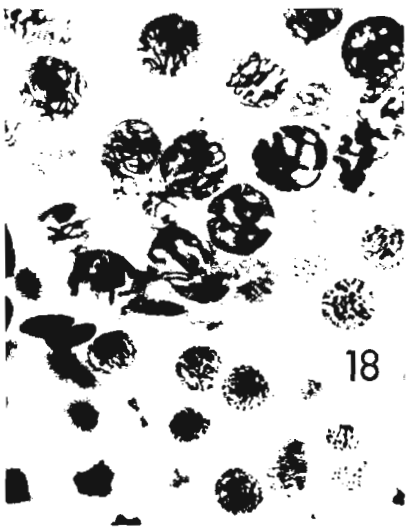
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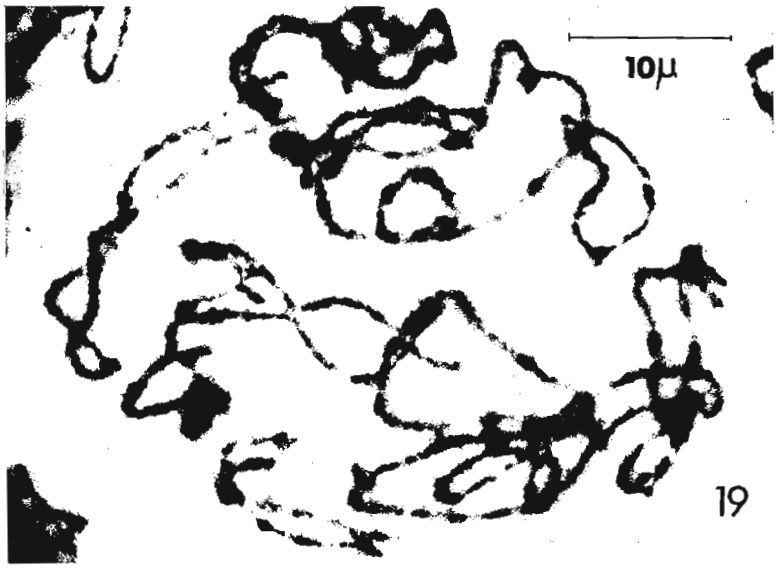
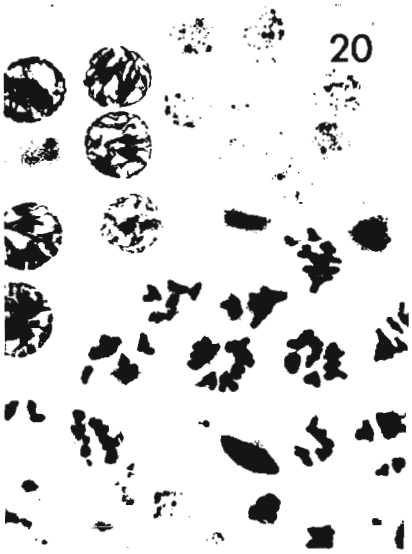


19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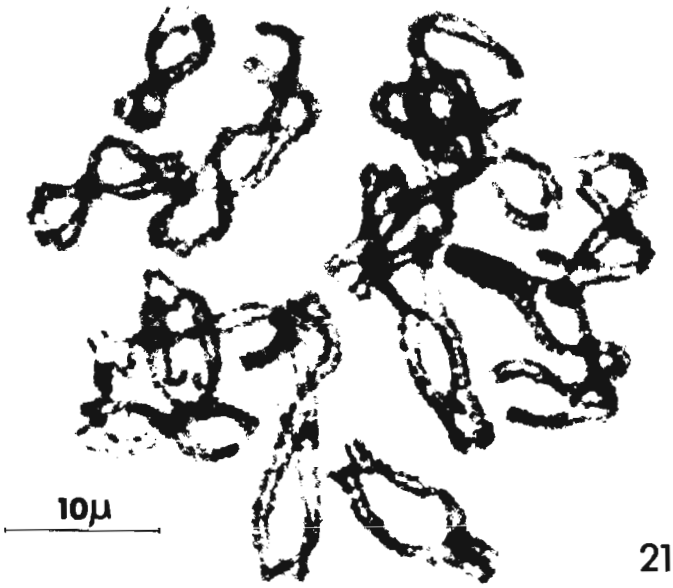

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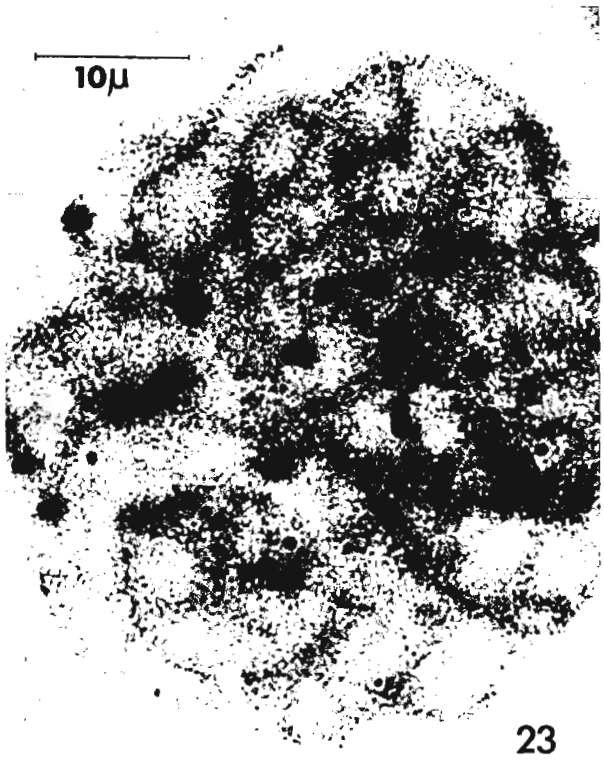
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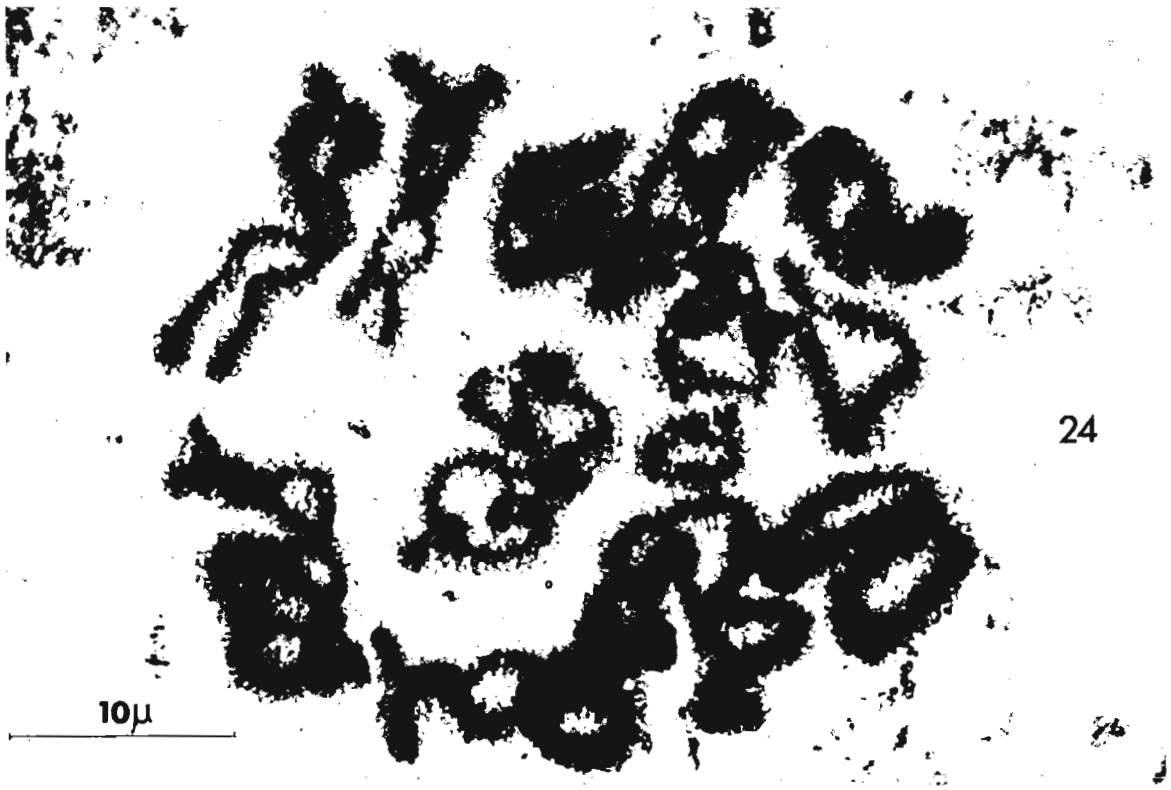
24
10μ

Jungen, H. and R. Locher. Zoological Museum of the University, Zürich, Switzerland. Apparatus for the determination of the egg laying time of single females of D. subobscura.

This apparatus allows one to study the egg laying pattern over 24 hours of single D. females. The principle idea is to have a glass plate covered with a food medium over which a vial containing a female is moved.

Fig. 1 shows the apparatus composed of two floors. The two plates bearing the food medium (p) measure 75 x 28 x 0.5 cm. A mobile vehicle (v) holds eight glass tubes (t) on each of the two exchangeable arms (a). Each vial is 22 mm in diameter. The tubes are held in position by

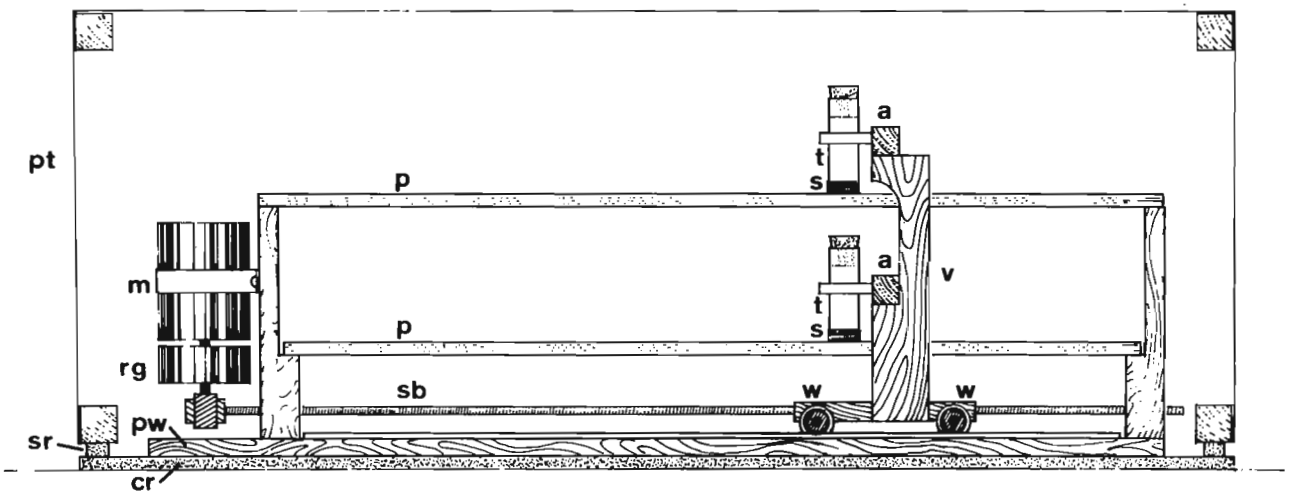

Figure 1

metal clasps and are closed with a plug. Their lower end is held 2 mm over the surface of the food medium. A silk band (s) with a fringed border (b) 2-3 mm wide is wrapped three times around the lower end of the tube in a way that the fringed border overlaps preventing the flies from escaping (fig. 2). The band is changed after every running. A screw bar (sb) goes through a nut fixed on the vehicle. A synchronous motor (m) from Philips (AU 5100/22) drives by a reduction gear (rg) of 3:1250 (Philips, BA/UR 3/1250 L) the screw bar and moves therewith the vehicle. The wheels (w) of the vehicle roll on a metal band. The apparatus is fixed on a non-warping plate of wood (pw) which lies on crepe rubber (cr). A switch changes the direction of rotation of the motor.

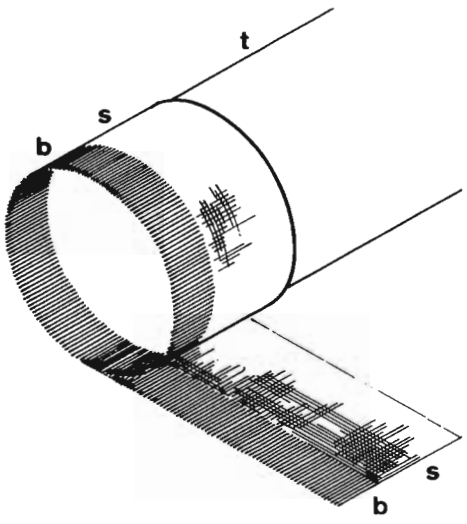

Figure 2

A transparent plastic tent (pt) serves to cover the whole apparatus. Strips of crepe rubber (sr) make it close tightly. The tent retains the humidity and prevents the invasion of other flies.

The agar and maize food is stained by molasses to facilitate counting of eggs. The food had to be cast on to the pre-heated glasses. A thin layer of yeast suspension is sprayed on the food plates by means of an atomizer and compressed air.

An additional technical note, "Fruit fly fractionation," by Oliver and Phillips, will be found on page 58.

Irwin H. Herskowitz, Editor

D. = Drosophila D.m. = Drosophila melanogaster

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PERSONAL AND LABORATORY NEWS I

Michael R. Cummings now Assistant Professor at Dept. of Biogical Sciences, Univ. of Illinois at Chicago Circle (from Northwestern Univ., Evanston, Ill.)
W. van Delden now Head of the Population Genetics Group in the Genetics Institute, Univ. of Groningen, Netherlands (after a year at the Univ. of Chicago)
J. James Donady now an NIH Postdoctoral Fellow in the Dept. of Biol., City of Hope Medical Center, Duarte, Calif. (from Dept. of Zoology, University of Iowa)
R. Fahrig now at the Zentrallaboratorium für Mutagenitätsprüfung, 7800 Freiburg/Breisgau, Breisacherstrasse 33 in the new laboratory of E. Vogel
Eleanor Markowitz now at the Dept. of Anatomy, Univ. of Wisconsin in Madison (from Iowa City)
Daigoro Moriwaki now Director of the National Institute of Genetics, Misima, Sizuoka-ken, Japan (from the Dept. of Biol., Tokyo Metropolitan Univ.)
M. Pelecanos now head of the new Department of Genetics at the University of Patras, Greece
W. Scharloo now occupies the second chair in Genetics at the Genetisch Institut, Rijksuniversiteit, Opaalweg 20, Utrecht, Netherlands (from the Univ. of Groningen)
Forbes W. Robertson now Head of the Department of Genetics, Univ. of Aberdeen, Scotland (from Edinburgh University, Scotland)
Bungo Sakaguchi now with the Faculty of Agriculture, Kushu Univ., Fukuoka, Japan (from the National Institute of Genetics, Misima, Japan)
B. Shorrocks now at the Dept. of Zoology, The University, Leeds, Yorkshire, England (from Newcastle-upon-Tyne)
Heinrich Ursprung now at the Laboratory for Developmental Biology, Swiss Federal Institute of Technology, Zürich, Switzerland (from Johns Hopkins Univ., Baltimore, Maryland)
Armon F. Yanders now Assistant Dean, Research and Graduate Programs, Univ. of Missouri, Columbia, Mo. (from Michigan State Univ.)

J.K. Lim. A new genetics laboratory, aimed primarily for undergraduate training, consisting of the following three rooms has been in operation since the Fall semester, 1969, at the Wisconsin State University, Eau Claire, Wisconsin:

air conditioned teaching laboratory with 16 stations....ca. 900 sq.ft.
air conditioned preparation room.....ca. 280 sq.ft.
air conditioned faculty research laboratory .....ca. 180 sq.ft.

At present, these rooms are equipped with necessary optic systems including three Carl Zeiss phase contrast microscopes, five incubators, hoods, medium pumps, an autoclave, and other minimum essential equipment for study of Drosophila and T4 phage. Approximately three-quarters of the laboratory exercises are with Drosophila and the remaining exercises are devoted to deletion mapping, complementation tests, and intragenic recombination studies using the rII mutants of T4 phage.

The Duke University Program in Genetics, an interdisciplinary program involving faculty members from the departments of Anatomy, Biochemistry, Botany, Medicine and Zoology, is offering a number of NIH Predoctoral Traineeships. Further information may be obtained by writing to the Director of the Genetics Program, Nanaline H. Duke Building 151, Duke University, Durham, North Carolina 27706.

A New Genetical Society: A new Genetical Society was established in the UAR which includes about 150 members. Professor A. Azim O. Tantawy, Professor of Population Genetics, Dept. of Genetics, Faculty of Agriculture, Alexandria University, Alexandria, UAR, was elected Chairman. The Society intends to establish its own library. Contributions of reprints (old or new), journals and books to the Society will be very much appreciated. The first symposium of the Society was held in Cairo in January 1970. Proceedings of the symposium will be published and institutions interested in purchasing the proceedings should get in touch with the Chairman.

J.F. Barker, University of Ibadan, Dept. of Zoology, Ibadan, Nigeria: If any suitably qualified person from abroad is interested in coming here as my research student to study the population genetics of African Drosophilids, it may be possible to arrange this.

From a letter from Mrs. H.J. Muller: A list of publications of H.J. Muller, up to 1961, is found in "Studies in Genetics" by H.J. Muller. A complete list will be found in Pontecorvo's "Biographical Memoirs of Fellows of the Royal Society," vol. 14, 1968.

DIS Job Information Service was not sufficiently widely subscribed to by either job seekers or (especially) potential employers to merit its continuation. If demands for such a service develop in the future, DIS will once again consider the possibility of re-opening that department.

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J. Erickson, Western Washington State College, Bellingham, Washington, is responsible for the suggestion that stock list page headings should include the names of the main stock centers, an idea utilized this time for the Pasadena Stock Center list. Practical suggestions from DIS subscribers are always welcome and will be used when possible.

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- See D.I.S.43:205

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See D.I.S.44:147

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see DIS 42:169

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See D.I.S.44:154

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See D.I.S. 44:171
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See D.I.S. 43:241
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See D.I.S. 43:244
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See D.I.S. 44:174

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Hewitt, Nathaniel, B.A. Graduate student. Gene dosage studies of ADH variants in triploid D. melanogaster
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Pipkin, Sarah B. Ph.D. Professor Regulation of activity of ODH structural genes during development in D. metzii, D. pellewae, and D. leticiae and in interspecific hybrids of these species
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PERSONAL AND LABORATORY NEWS II

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Choi, Y. Austria, Vienna
Choo, J.K. Korea, Seoul
Chornoma, R. Upton, New York
Chovnick, A. Storrs, Connecticut
Christmas, J. Canada, Toronto
Christopher, W. Chapel Hill, North Carolina
Chung, C.U. Korea, Kwangju
Chung, J.Y. Korea, Seoul
Chung, O.K. Korea, Seoul
Clancy, C.W. Eugene, Oregon
Clancy, E. Eugene, Oregon
Clark, A. Stony Brook, New York
Clark, A.M. Australia, Adelaide
Clark, A.M. Newark, Delaware
Clawson, M.G. Knoxville, Tennessee
Claxton, J.H. Australia, Armidale
Clayton, F.E. Fayetteville, Arkansas
Clise, R. Cleveland, Ohio
Clower, K. Gary, Indiana
Coe, E.H.Jr. Columbia, Missouri
Cohn, R.H. Urbana, Illinois
Colaianne, J.J. Lafayette, Indiana
Colin, C. Duarte, California
Conine, D. Austin, Texas
Connolly, K.J. England, Sheffield
Cook, R.M. England, Sheffield
Cooper, K.W. Riverside, California

Cordeiro, A.R. Brasil, Pôrto Alegre
Cordle, M.S. Chapel Hill, North Carolina
Coughlin, J.M. St. Louis, Missouri
Coulter, G. Australia, Brisbane
Counce, S.J. Durham, North Carolina
Crépin-de Jong, T. Netherlands, Leiden
Critelli, G. Buffalo, New York
Croft, J.H. England, Birmingham
Crossley (née Pearce), S.A. Australia, Clayton
Crow, J.F. Madison, Wisconsin
Cuba, R. Houston, Texas
Cullen, M.U. New Haven, Connecticut
Daillie, J. France, Lyon
Danieli, G.A. Italy, Padova
Dapples, C.C. Billings, Montana
Darst, P.R. Chapel Hill, North Carolina
Das, A.K. India, Calcutta
Datta, R.K. India, Calcutta
Daugherty, P.A. Greenville, North Carolina
David, J. France, Lyon
Davis, B.K. Seattle, Washington
Davis, C.S. Columbus, Ohio
Dawood, M.M. U.A.R., Alexandria
Day, J.W. Ames, Iowa
Dean, M. Ithaca, New York
De Garay, A.L. Mexico, Mexico City
Deland, M.C. Storrs, Connecticut
De la Rosa, E. Mexico, Mexico City
Delden, W. van Netherlands, Haren
del Solar, E. Chile, Santiago
DeMarinis, F. Cleveland, Ohio
Dene, H.T. Rochester, Michigan
Denell, M.T. Austin, Texas
Denell, R.E. Austin, Texas
Dennis, L. England, Sheffield
Dennison, D. Lubbock, Texas
De Reggi-Mourgues, C. France, Lyon
De Remer, J. Rochester, New York
Derridj, S. France, Gif-sur-Yvette
Devaux, J. France, Gif-sur-Yvette
de Witt, P. Chapel Hill, North Carolina
Dews, D.L. Eugene, Oregon
Diamantopoulou, E. Greece, Athens
Dickerman, R. Cleveland, Ohio
Dickinson, W.J. Portland, Oregon
Dijken, F.R. van Netherlands, Haren
Doane, W.W. New Haven, Connecticut
Dobzhansky, Th. New York, New York
Doira, H. Japan, Fukuoka
Dolfini, S. Italy, Milan
Dollar, J. Lexington, Kentucky
Donaldson, D.W. Columbus, Ohio
Driskell, W.J. Pasadena, California
Dübendorfer, A. Switzerland, Zürich
Duck, P.D. Storrs, Connecticut
Duckenfield, J.E. Baton Rouge, Louisiana
Dunn, A.J. Chapel Hill, North Carolina
Dunn, B.K. Madison, Wisconsin
Dunn, M. Ithaca, New York
Du Pui, M.L.L. Netherlands, Haren
Durrett, S. Lexington, Kentucky
Dutta Gupta, A.K. India, Calcutta
Duyn-Goedhardt, A. van Netherlands, Leiden
Dykhuizen, D.E. Chicago, Illinois
Eames, A. Logan, Utah
Early, D. Lafayette, Indiana
Easwaran, B.M. India, Madras
Ebersole, L.A. Columbus, Ohio
Ebisuzaki, M.M. Canada, Regina
Ebitani, N. Japan, Tokyo
Edington, C.W. Washington D.C.
Edney, E.B. Riverside, California
Eggert, R. Davis, California
Ehrlich, E. Eugene, Oregon
Ehrman, L. New York, New York
Eiche, A. Sweden, Stockholm
Eichenberger, E. Pasadena, California
Elens, A. Belgium, Namur
El-Helw, A. U.A.R., Alexandria
El-Helw, M.R. U.A.R., Cairo
Ellis, J.F. Clinton, New York
El-Kawolgy, T.A. U.A.R., Cairo
Ellison, J.R. Eugene, Oregon
Elmore, E.L. Logan, Utah
Emara, M.K. U.A.R., Alexandria
Emison, M. Austin, Texas
Emmens-Pieters, J. Netherlands, Haren
Engelking, A.B. San Marcos, Texas
Engle, J.B. Madison, Wisconsin
Englert, D.C. Carbondale, Illinois
Engstrom, L.E. Urbana, Illinois
Enns, R.E. Eugene, Oregon
Epler, J.L. Oak Ridge, Tennessee
Erickson, J. Bellingham, Washington
Erk, F.C. Stony Brook, New York
Escott, C. Australia, Bundoora
Esposito, V.M. Bethesda, Maryland
Ezell, D. Canada, Vancouver
Faber, J. Netherlands, Utrecht
Fabergé, A.C. Austin, Texas
Fahmy, A.M. U.A.R., Alexandria
Fahmy, M.J. England, Chalfont St. Giles
Fahmy, O.G. England, Chalfont St. Giles
Falk, R. Israel, Jerusalem
Falke, E. Charlottesville, Virginia
Faller, E. Switzerland, Zürich
Farrow, P. Ithaca, New York
Faulkner, B.M. England, Liverpool
Faust, C.C. Baton Rouge, Louisiana
Fausto, A. Providence, Rhode Island
Félix, R. Mexico, Mexico City
Fellows, D. Tucson, Arizona
Femino, J. Providence, Rhode Island
Fenner, H. Chile, Santiago
Fernandes, N. Brasil, São Paulo
Feuerbach-Mravlag, H. Austria, Vienna
Fields, P. Philadelphia, Pennsylvania
Figueroa, J.A. Chicago, Illinois
Finlay, D. Australia, Sydney
Finnerty, V.G. Storrs, Connecticut
Fisher, L. South Orange, New Jersey
Fitz-Earle, M. Canada, Toronto

Fletcher, R.A. Spokane, Washington
Florian, M.L.E. Canada, Ottawa
Fontdevila, A. Spain, Barcelona
Fontyne, M.C. France, Orsay
Forbes, C. Moscow, Idaho
Forrest, H.S. Austin, Texas
Fort, C. Spain, Barcelona
Foster, G. Canada, Vancouver
Fouillet, P. France, Lyon
Fourche, J. France, Lyon
Fowler, G.L. Eugene, Oregon
Fox, A.S. Madison, Wisconsin
Fox, D. Switzerland, Zürich
Fox, D.P. Scotland, Aberdeen
Frankham, R. Chicago, Illinois
Franklin, R. Notre Dame, Indiana
Fredline, D.F. Australia, Armidale
French, W.L. Baton Rouge, Louisiana
Frey, F. France, Gif-sur-Yvette
Frias, D. Chile, Santiago
Friedberg, E. Israel, Jerusalem
Friedman, L.D. St. Louis, Missouri
Friedman, T.B. Ann Arbor, Michigan
Fritz-Niggli, H. Switzerland, Zürich
Frutos, R. de Spain, Barcelona
Frydenberg, O. Denmark, Aarhus
Fuchs, M.S. Notre Dame, Indiana
Fujii, H. Japan, Fukuoka
Fujii, S. Japan, Kobe
Fujimoto, M. Japan, Tokyo
Fujio, Y. Japan, Nagoya
Fukatami, A. Japan, Tokyo
Funk, C. Germany, Saarbrücken
Fuscaldo, K.E. Philadelphia, Pennsylvania
Futch, D.G. San Diego, California
Gabay, S.J. Urbana, Illinois
Gale, J.S. England, Birmingham
Ganguly, R. India, Calcutta
Gans, M. France, Gif-sur-Yvette
García-Bellido, A. Spain, Madrid
García, P. Spain, Barcelona
Gardner, E.J. Logan, Utah
Garen, A. New Haven, Connecticut
Garnett, P. Baton Rouge, Louisiana
Gay, H. Ann Arbor, Michigan
Gazal, M. U.A.R., Alexandria
Gearhart, J. Ithaca, New York
Gee, B. Ithaca, New York
Gee, P. Baton Rouge, Louisiana
Geer, B.W. Galesburg, Illinois
Gehring, W. New Haven, Connecticut
Gelbart, W.M. Madison, Wisconsin
Geltosky, J.E. Pasadena, California
Gerdes, R.A. Denton, Texas
Gersh, E.S. Philadelphia, Pennsylvania
Gethmann, R.C. Chicago, Illinois
Gibbs, E.M. England, Chalfont St. Giles
Gibson, D. Canada, Vancouver
• Gibson, J.B. England, Cambridge
Giebelhausen, E. Urbana, Illinois
Giesel, J.T. Chicago, Illinois
Giessman, B. St. Louis, Missouri
Gill, J.J.B. England, Liverpool
Gillespie, J.H. Austin, Texas
Glaser, P. Providence, Rhode Island
Glass, B. Stony Brook, New York
Glassman, E. Chapel Hill, North Carolina
Glover, T.J. Columbus, Ohio
Gnes, A. Italy, Padova
Gochberg, C. Madison, Wisconsin
Godbole, N.N. India, Poona
Godoy, R. Chile, Santiago
Gold, E.E. Urbana, Illinois
Golden, B. Providence, Rhode Island
Goldschmidt, E. Israel, Jerusalem
Goldstein, M.A. Houston, Texas
González, C. Spain, Barcelona
Gonzalez, F.W. Upton, New York
González, R. Spain, Barcelona
Gooch, J.L. Huntingdon, Pennsylvania
Goodnight, K. Tucson, Arizona
Gorospe, J. Spain, Madrid
Gossi, S.J. Pullman, Washington
Gotborn, L. Sweden, Uppsala
Götz, K.G. Germany, Tübingen
Gould, A.B. Newark, Delaware
Gouldbourn, J.M. England, Sheffield
Grabicki, E. New Haven, Connecticut
Grace, D. Stony Brook, New York
Graf, U. Switzerland, Zürich
Graham, M. Philadelphia, Pennsylvania
Grainger, J. England, Heslington
Grant, B.S. Williamsburg, Virginia
Greco, J. Morgantown, West Virginia
Green, M.M. Davis, California
Green, P. Madison, Wisconsin
Greg, M. Tucson, Arizona
Gregg, T.L. Berkeley, California
Greisen, K.S. Pasadena, California
Grell, E.H. Oak Ridge, Tennessee
Grell, R.F. Oak Ridge, Tennessee
Griffin, J.D. Albuquerque, New Mexico
Grigliatti, T. Canada, Vancouver
Grimes, W.P. Dallas, Texas
Gritzmacher, C. Eau Claire, Wisconsin
Grivet, F. France, Gif-sur-Yvette
Groot-van Stralen, C.Th. de Netherlands, Leider
Grossberg, R.D. Carbondale, Illinois
Grossfield, J. Lafayette, Indiana
Grüneberg, H. England, London
Gsell, R. Switzerland, Zürich
Guest, W.C. Fayetteville, Arkansas
Gupta, J.P. India, Varanasi
Gupta, R. India, Calcutta
Guterman, H. Ithaca, New York
Guzmán, J. Mexico, Mexico City
Haapala, O. Finland, Turku
Hablas, A.A. U.A.R., Alexandria
Hackman, W. Finland, Helsinki
Hadorn, E. Switzerland, Zürich
Halfer, C. Italy, Milan
Hall, J. Seattle, Washington

Hama, H. Japan, Chiba
Hammoda, M.H. U.A.R., Giza
Hammond, K. Australia, Sydney
Hanks, G.D. Gary, Indiana
Hannah-Alava, A. Finland, Turku
Hanson, T.E. Pasadena, California
Hardjosworo, P. Ann Arbor, Michigan
Hardy, R. San Diego, California
Harlander, S. Eau Claire, Wisconsin
Harman, J.W. England, Chalfont St. Giles
Harrison, B.J. England, Norwich
Harrison, W.L. Williamsburg, Virginia
Harrod, M.J. Dallas, Texas
Hartman, D. New Haven, Connecticut
Hartmann-Goldstein, I.J. England, Sheffield
Hashem, Y.D. U.A.R., Alexandria
Hassan, A.L. U.A.R., Giza
Hathaway, D. St. Louis, Missouri
Hauri, H.-P. Switzerland, Zürich
Hawkins, E. Stony Brook, New York
Hay, D.A. England, Birmingham
Hayashi, K. Japan, Osaka
Hayman, D.L. Australia, Adelaide
Hebert, P. England, Cambridge
Hedges, J. Riverside, California
Heed, W. Tucson, Arizona
Heisenberg, M. Germany, Tübingen
Hengstenberg, R. Germany, Tübingen
Hennig, I. Germany, Tübingen
Hennig, W. Germany, Tübingen
Hensen, A.E. Netherlands, Leiden
Herbert, G. Providence, Rhode Island
Hess, O. Germany, Freiburg
Hewitt, J. England, Leeds
Hewitt, N. Washington D.C.
Hewlett, J.M. Evanston, Illinois
Heystek, A.G. Netherlands, Leiden
Hihara, F. Japan, Tokyo
Hihara, Y.K. Japan, Tokyo
Hinton, C.W. Wooster, Ohio
Hiraizumi, Y. Austin, Texas
Hirose, Y. Japan, Kobe
Hiroyoshi, T. Japan, Osaka
Hiss, E.A. Notre Dame, Indiana
Hittle, M. Duarte, California
Hjorth, P.J. Denmark, Aarhus
Hochman, B. Knoxville, Tennessee
Hodgetts, R.B. Pasadena, California
Hoff, D. Madison, Wisconsin
Hogness, D.S. Stanford, California
Holden, J. Canada, Vancouver
Hollander, W.F. Ames, Iowa
Hollingsworth, M.J. England, London
Holm, D. Canada, Vancouver
Holmquist, G.P. Urbana, Illinois
Holt, A.C.E. England, Reading
Holzman, H.E. Chambersburg, Pannsylvania
Honda, Y. Japan, Nagasaki
Hooker, W.J. Wooster, Ohio
Hooper, G.B. Poughkeepsie, New York
Hotta, Y. Pasadena, California
House, V.L. Columbus, Ohio
Howard, L.A. Chapel Hill, North Carolina
Huang, S.L. Austin, Texas
Hubby, J.L. Chicago, Illinois
Hudson, G.J. England, Cambridge
Hughes, M. Cambridge, Massachusetts
Hunt, D.M. England, London
Hunt, J.P. Wales, Glamorgan, Swansea
Hunt, P. Berkeley, California
Huntress, G. Philadelphia, Pennsylvania
Hürlimann, R. Switzerland, Zürich
Ikeda, H. St. Louis, Missouri
Ikeda, H. Japan, Tokyo
Imaizumi, Y. Japan, Chiba
Imberski, R.B. College Park, Maryland
Inagaki, E. Japan, Chiba
Inagaki, E. Netherlands, Leiden
Inoue, A. Japan, Nagasaki
Irvine, S.R. Logan, Utah
Ismail, A.A. U.A.R., Cairo
Istre, G. Notre Dame, Indiana
Ito, S. Japan, Tokyo
Iturra, P. Chile, Santiago
Iyama, S. Japan, Misima
Ibrahim, S. U.A.R., Alexandria
Jackson, J. Lafayette, Indiana
Jackson, P.G. Knoxville, Tennessee
Jacob, M. India, Varanasi
Ječná, J. Czechoslovakia, Prague
Jeffery, D.E. Provo, Utah
Jelnes, J.E. Denmark, Copenhagen
Jenkins, J.B. Swarthmore, Pennsylvania
Jinks, J.L. England, Birmingham
Johansen, J. Norway, Oslo
Johansson, V. Sweden, Uppsala
Johnson, L. Madison, Wisconsin
Johnson, M. Houston, Texas
Johnson, W.W. Albuquerque, New Mexico
Johnston, F. Storrs, Connecticut
Johnston, S. Tucson, Arizona
Jones, D.A. England, Birmingham
Jones, G.H. England, Birmingham
Jones, J.K. England, Reading
Jones-Mortimer, M.C. England, Birmingham
Jonker, F.H. Netherlands, Haren
Jousset, F.X. France, St. Cristol les Alès
Judd, B.H. Austin, Texas
• Jungen, H. Switzerland, Zürich
Kachur, F. Gary, Indiana
Kahlstorf, M.A. Houston, Texas
Kaji, S. Japan, Kobe
Kale, P.G. India, Varanasi
Kälin, M. Switzerland, Zürich
Kambysellis, M.P. Cambridge, Massachusetts
Kanehisa, T. Japan, Kobe
Kaneko, A. Japan, Sapporo
Kang, S.-H. Notre Dame, Indiana
Kang, Y.S. Korea, Seoul
Kaplan, E. Stony Brook, New York
Kaplan, W.D. Duarte, California
Karlik, A. Austria, Vienna

Kasinsky, H. Canada, Vancouver
Kasmar, L. San Diego, California
Kassir, Y. Israel, Jerusalem
Kastritsis, C.D. Dallas, Texas
Kato, C. Japan, Misima
Katz, A.J. Columbus, Ohio
Kaufman, T.C. Austin, Texas
Kaufmann, B.P. Ann Arbor, Michigan
Kawabe, M. Japan, Kobe
Kawaguchi, M. Japan, Nagasaki
Kawaharada, M. Japan, Nagasaki
Kawanishi, M. Japan, Misima
Keane, R. Buffalo, New York
Kearsey, M.J. England, Birmingham
Keith, M.J. Houston, Texas
Keller, E.C. Morgantown, West Virginia
Keller, H. Morgantown, West Virginia
Kernaghan, R.P. Stony Brook, New York
Kersten, H.J.M.G. Netherlands, Utrecht
Kessenich, M. Madison, Wisconsin
Kidwell, J. Providence, Rhode Island
Kidwell, M. Providence, Rhode Island
Kieft, P. Netherlands, Leiden
Kikkawa, H. Japan, Osaka
Kim, K.W. Korea, Kwangju
Kimchi, Z. Israel, Jerusalem
Kimura, M. Japan, Misima
King, R.C. Evanston, Illinois
Kircher, H. Tucson, Arizona
Kirchgessner, T.L. Albuquerque, New Mexico
Kiriazis, W.C. Upton, New York
Kirschbaum, W.F. Argentina, Buenos Aires
Kitagawa, O. Japan, Tokyo
Kitazume, Y. Japan, Kobe
Klayman, M.B. Pasadena, California
Kleager, A. Lincoln, Nebraska
Klein, D.L. Ann Arbor, Michigan
Klekar, A. Austin, Texas
Klepetko, D. Cleveland, Ohio
Klingele, D. Philadelphia, Pennsylvania
Kloby, J. Providence, Rhode Island
Klotz, J.B. Ann Arbor, Michigan
Knight, G.R. Scotland, Edinburgh
Kobelt, D. Switzerland, Zürich
Koehn, R.K. Denmark, Aarhus
Köhler, B. Germany, Tübingen
Kojima, K. Austin, Texas
Komori, M. Japan, Nagasaki
Kondo, K. Japan, Nagoya
Konopka, R.J. Pasadena, California
Kooistra. J. Netherlands, Haren
Koref-Santibañez, S. Chile, Santiago
Korinek, E. Canada, Vancouver
Kosuda, K. Japan, Tokyo
Kothari, R.M. India, Poona
Kovalick, T. Buffalo, New York
Krause, C.E. Storrs, Connecticut
Krause, E. South Orange, New Jersey
Krider, H.M. Madison, Wisconsin
Krimbas, C.B. Greece, Athens
Krivshenko, E. Rochester, New York
Krivshenko, J.D. Rochester, New York
Kroeger, H. Germany, Saarbrücken
Kroman, R.A. Long Beach, California
Kubli, E. Switzerland, Zürich
Kucherlapati, R.S. Urbana, Illinois
Kugler, W. Madison, Wisconsin
Kuhta, A. South Orange, New Jersey
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
Laird, C.D. Austin, Texas
Lakhotia, S.C. India, Calcutta
Lakka, K. England, Chalfont St. Giles
Lakovaara, S. Finland, Helsinki
Lamb, M.J. England, London
Lamborot, M. Chile, Santiago
Landner, L. Sweden, Stockholm
Langley, C.H. Austin, Texas
Langlois, B. France, Gif-sur-Yvette
Lanman, J.T.Jr. DeKalb, Illinois
Lasa, L. Spain, Madrid
Lau, K.C. Eau Claire, Wisconsin
Laugé, G. France, Orsay
Laughnan, J.R. Urbana, Illinois
Lawrence, M.J. England, Birmingham
Lawson, L.R. Dayton, Ohio
Lechien, J. Belgium, Namur
Lechner, J. Philadelphia, Pennsylvania
Lechner, N. Philadelphia, Pennsylvania
Lee, B.W. Korea, Seoul
Lee, C.C. Korea, Seoul
Lee, C.S. Korea, Seoul
Lee, F.C.-J. Madison, Wisconsin
Lee, T.J. Korea, Seoul
Lee, W.R. Baton Rouge, Louisiana
Lefevre, G. Northridge, California
Leffel, L.E. Madison, Wisconsin
Leigh, B. Netherlands, Leiden
• Lemeunier, F. France, Gif-sur-Yvette
Leonard, S. Wales, Glamorgan, Swansea
Leto, G. DeKalb, Illinois
Leuthold, U. Switzerland, Zürich
Leventhal, E.A. Madison, Wisconsin
Levin, B. Providence, Rhode Island
Levine, H. Storrs, Connecticut
Levins, R. Chicago, Illinois
Levitan, E. New York, New York
Levitan, M. New York, New York
Levy, A.W. Australia, Adelaide
Lewis, E.B. Pasadena, California
Lewis, H.W. Washington D.C.
Lewontin, R.C. Chicago, Illinois
Lezzi, M. Switzerland, Zürich
L'Hélias, C. France, Gif-sur-Yvette
Libion, M. Belgium, Namur
Lifschytz, E. San Diego, California
Lim, J.K. Eau Claire, Wisconsin
Lin, F.-J. Austin, Texas
Lindsay, H.A. Austin, Texas

Lindsley, D.L. San Diego, California
Linney, R. England, Birmingham
Lipps, K. Davis, California
Llewellyn, M. Bellingham, Washington
Lloyd, B. Australia, Adelaide
LoCascio, N. Buffalo, New York
Locker, D. France, Gif-sur-Yvette
Lokki, J.M. 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. France, Gif-sur-Yvette
Loukas, M. Greece, Athens
Loyal, R. Germany, Freiburg
Lu, C.C. Houston, Texas
Lucas, A. Australia, Adelaide
Lucchesi, J.C. Chapel Hill, North Carolina
Luce, W.M. Urbana, Illinois
Luck, J. Austin, Texas
Ludwig, M.R. Brasil, Pôrto Alegre
Lundelius, J. Austin, Texas
Lüning, K.G. Sweden, Stockholm
Lutes, C.M. Radford, Virginia
Lütolf, H.-U. Switzerland, Zürich
MacBean, I.T. Australia, Bundoora
MacCaulay, S. Canada, Vancouver
Machado, D.M. Brasil, Pôrto Alegre
Machida, I. Japan, Chiba
Machlus, B. Chapel Hill, North Carolina
Machová, H. Czechoslovakia, Brno
Macht, V. Notre Dame, Indiana
MacIntyre, R. Ithaca, New York
Maddern, R.H. Australia, Adelaide
Madhaven, K. Switzerland, Zürich
Maeda, M. Japan, Misima
Maeda, Y. Japan, Kobe
Magalhaes, L.E. de Brasil, Sao Paulo
Magnusson, J. Sweden, Stockholm
Maher, E.P. Scotland, Aberdeen
Mainx, F. Austria, Vienna
Majoral, J. Spain, Barcelona
Majumdar, S.K. Easton, Pennsylvania
Makela, M.E. Austin, Texas
Manchester, W. Riverside, California
Mange, A.P. Amberst, Massachusetts
Manna, G.K. India, Kalyani
Manna, P.K. India, Calcutta
Manning, I. Houston, Texas
Marble, J.R. Eugene, Oregon
Marinic, S.E. Argentina, Buenos Aires
Mark, H. Providence, Rhode Island
Marques, E. Ithaca, New York
Marques, E.K. Brasil, Pôrto Alegre
Marrakechi, M. France, Gif-sur-Yvette
Martin, A.O. Cleveland, Ohio
Martinez, C.P. Argentina, Buenos Aires
Martínez, J.M. Spain, Barcelona
Martinez, M.N. Brasil, Pôrto Alegre
Maruyama, T. Japan, Misima
Maruyama, T. Japan, Tokyo
Marynick, S.P. Dallas, Texas
Masry, A.M. U.A.R., Alexandria
Masterson, J.E. Ames, Iowa
Masuda, H. Japan, Misima
Mather, W.B. Australia, Brisbane
Matheson, A.C. Australia, Bundoora
Matter, B. Switzerland, Zürich
Matulová, O. Czechoslovakia, Prague
Mayeda, K. Detroit, Michigan
Mayo, M.J. Australia, Adelaide
Mayor, H. Canada, Vancouver
Mazar Barnett, B. Argentina, Buenos Aires
Mazijk, M.E. van Netherlands, Utrecht
McCarron, M. Storrs, Connecticut
McCarthy, P. Detroit, Michigan
McCrady, E. III Greensboro, North Carolina
McCune, T.B. Austin, Texas
McFadyen, M. Tucson, Arizona
McKay, C.M. Pullman, Washington
McKinley, A. Australia, Sydney
McMillan, I. Canada, Toronto
McMurtrey, M. Houston, Texas
McNeil, H.M. Dallas, Texas
McNew, R.W. Lafayette, Indiana
McQuigg, M. Austin, Texas
Mead, C.G. Oak Ridge, Tennessee
Meeles, E. Netherlands, Haren
Meier, P. Switzerland, Zürich
Meltzer, P.S. Pasadena, California
Mendoza, G. New York, New York
Ménsua, J.L. Spain, Barcelona
Mercio, A.L. Brasil, Pôrto Alegre
Merle, J. France, Lyon
Metcalf, J.A. England, Heslington
Meyer, G.F. Germany, Tübingen
Mickey, G.H. Ridgefield, Connecticut
Miles, M. San Marcos, Texas
Millar, D.H. Australia, Sydney
Miller, D.D. Lincoln, Nebraska
Miller, D.H. Australia, Sydney
Miller, M.J. Pasadena, Californi
Miller, S. Seattle, Washington
Minato, K. Japan, Misima
Minnier, D. Morgantown, West Virginia
Mishima, A. Japan, Sakado-Machi
Mitchell, B. Denton, Texas
Mitchell, H.K. Pasadena, California
Mittler, S. DeKalb, Illinois
Mitra, N. India, Calcutta
Miyamoto, T. Japan, Hiroshima
Mizuguchi, Y. Brasil, São Paulo
Mohr, M.A. Houston, Texas
Moisand, R. Buffalo, New York
Mollah, S.I.Md.S.A. Wales, Glamorgan
Mollet, P. Switzerland, Zürich
Molten, P. Chapel Hill, North Carolina
Momma, E. Japan, Sapporo
Monclús, M. Spain, Barcelona
Montelius, I. Sweden, Stockholm
Montgomery, I.N. Urbana, Illinois
Moon, M.A. Denton, Texas

Moore, C.M. Oak Ridge, Tennessee
Moore, M. Duarte, California
Morales, N.B. Brasil, Pôrto Alegre
Morata, G. Spain, Madrid
Moree, R. Pullman, Washington
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
Mossige, J. Norway, Oslo
Moth, J.J. Australia, Sydney
Mourad, A.M. U.A.R., Alexandria
Mourao, C.A. New York, New York
Muckenthaler, F.A. Albany, New York
Mukherjee, A.S. India, Calcutta
Mukherjee, R.N. Austria, Vienna
Mukherjee, U. Austria, Vienna
Mulindwa, D. Uganda, Kampala
Mulrennan, C.A. Weston, Massachusetts
Munoz, E. Netherlands, Leiden
Muñoz, E.R. Argentina, Buenos Aires
Murakami, A. Japan, Misima
Murata, M. Japan, Chiba
Murphy, C. Berkeley, California
Murphy, J.P. Austin, Texas
Myszewski, M.E. Des Moines, Iowa
Nadolney, C. New York, New York
Nafei, H.A. U.A.R., Giza
Nair, P.S. Austin, Texas
Nakagawa, K. Japan, Tokyo
Nakai, S. Japan, Chiba
Nakai, S. Japan, Osaka
Nakamura, A. Japan, Hiroshima
Nakanishi, H.Y. Japan, Chiba
Nakao, Y. Japan, Hiroshima
Nakashima-Tanaka, E. Japan, Osaka
Narayanan, Y. St. Louis, Missouri
Nardin, H. Baton Rouge, Louisiana
Narise, S. Japan, Tokyo
Narise, T. Japan, Sakado-Machi
Nash, D.J. Fort Collins, Colorado
Nasrat, G.E. U.A.R., Giza
Natori, S. New Haven, Connecticut
Nawa, S. Japan, Misima
Neeley, J.C. Portland, Oregon
Nei, M. Providence, Rhode Island
Newton, J.A. Chapel Hill, North Carolina
Nicklas, R.B. Durham, North Carolina
Nielsen, J.T. Denmark, Aarhus
Niet, J.P. van der Netherlands, Leiden
Nigon, V. France, Lyon
Nilson, L.R. Sweden, Uppsala
Nilsson, B. Sweden, Stockholm
Nishimori, T. Japan, Hiroshima
Nishiura, J.T. Seattle, Washington
Nix, C.E. Chicago, Illinois
Nogués, R. Spain, Barcelona
Nolph, M. Rochester, Michigan
Norby, S. Denmark, Copenhagen
Norton, S. Charlottesville, Virginia
Nöthiger, R. Switzerland, Zürich
Novitski, E. Eugene, Oregon
Nozawa, H. Seattle, Washington
Nozawa, K. Japan, Nagoya
Numanoi, H. Japan, Tokyo
O'Brien, S. Ithaca, New York
Oftedal, P. Norway, Oslo
Ogah, F. Ithaca, New York
Ogaki, M. Japan, Osaka
Ogita, Z. Japan, Osaka
Ogonji, G. Washington D.C.
Oguma, Y. Japan, Tokyo
Ohba, S. Japan, Tokyo
Ohki, K. Japan, Nagasaki
Ohta, T. Japan, Misima
Oishi, S. New Haven, Connecticut
Oishi, T. New Haven, Connecticut
Okada, T. Japan, Tokyo
Oksala, T.A. Finland, Turku
Okuno, T. Japan, Osaka
Olive, M. Houston, Texas
Oliver, C.P. Austin, Texas
Oliver, D. Austin, Texas
Olsher, N.B. Pullman, Washington
Olthoff, H.M. Netherlands, Haren
Olvera, O. Mexico, Mexico City
Ondřej, M. Czechoslovakia, Prague
Orevi, N. Israel, Jerusalem
Ortiz, E. Spain, Madrid
Oshima, C. Japan, Misima
Ostrowski, R. Notre Dame, Indiana
Ouweneel, W.J. Netherlands, Utrecht
Paika, I.J. Lincoln, Nebraska
Pak, W.L. Lafayette, Indiana
Pakonen, C.Z. Pullman, Washington
Palabost, L. France, Paris
Park, E.H. Korea, Seoul
Park, M.S. Korea, Kwangju
Parker, D.R. Netherlands, Leiden
Parker, M. Houston, Texas
Parry, D. Seattle, Washington
Parsons, P.A. Australia, Bundoora
Pasteur, G. Dallas, Texas
Pasteur, N. Dallas, Texas
Pastorok, R.A. Notre Dame, Indiana
Paterson, H.E. Australia, Nedlands
Pavlovsky, O. New York, New York
Paz, C. Argentina, Buenos Aires
Peacock, W.J. San Diego, California
Pederson, N. Portland, Oregon
Peedin, N. Durham, North Carolina
Perdrix-Gillot, S. France, Lyon
Pereyra, E. Argentina, Buenos Aires
Perez-Chiesa, I. Chicago, Illinois
Perez-Salas, S. New York, New York
Periquet, G. France, Paris
Pernaveau, A. Columbus, Ohio
Perreault, W.J. Ann Arbor, Michigan
Perry, M. Scotland, Edinburgh
Peterson, K. Northridge, California

Petit, C. France, Paris
Phillips, J. Austin, Texas
Pilares, L. Chile, Santiago
Pipkin, S.B. Washington D.C.
Pittendrigh, C.S. Stanford, California
Plus, N. France, St. Christol les Alès
Poirier, M. Durham, North Carolina
Policansky, D.J. Eugene, Oregon
Polivanov, S. Washington D.C.
Pomato, N.J. Notre Dame, Indiana
Pomeroy, M. Canada, Ottawa
Pomponio, E.A. Ithaca, New York
Portin, P. Finland, Turku
Poskitt, P. Cambridge, Massachusetts
Pospisil, Z.V. Eugene, Oregon
Potter, J.H. College Park, Maryland
Poulson, D.F. New Haven, Connecticut
Powell, J.R. New York, New York
Powers, L.M. Washington D.C.
Prakash, S. Rochester, New York
Pratt, R. Canada, Vancouver
Prevosti, A. Spain, Barcelona
Procunier, D. Ames, Iowa
Proust, J. France, Orsay
Prud'homme, N. France, Gif-sur-Yvette
Prudommeau, C. France, Orsay
Pscheidt, R. Galesburg, Illinois
Pullen, A.E. Netherlands, Leiden
Püntener, W. Switzerland, Zürich
Puro, J. Finland, Turku
Querubin, M.A. Brasil, São Paulo
Quinton, E. Chapel Hill, North Carolina
Radcliffe, C. Long Beach, California
Radford, W.L. Knoxville, Tennessee
Rai Chaudhuri, A. India, Calcutta
Rajaraman, R. Canada, Halifax
Ram, J. Pasadena, California
Ramamurthy, G. Berkeley, California
Ramel, C. Sweden, Stockholm
Ramila, D. Brasil, Pôrto Alegre
Rathie, K.A. Australia, Sydney
Rathnasabapathy, V. India, Madras
Ratty, F.J. San Diego, California
Rawls, J.M. Chapel Hill, North Carolina
Ray-Chaudhuri, S.P. India, Varanasi
Rayle, R.E. Chapel Hill, North Carolina
Raymond, J. Eugene, Oregon
Razzini, A. Italy, Milan
Reguly, M.L. Brasil, Pôrto Alegre
Reichert, D.F. Austin, Texas
Relichová, J. Czechoslovakia, Brno
Relton, J. England, Sheffield
Remondini, D.J. Spokane, Washington
• Rendel, J.M. Australia, Sydney
Resch, K.M. Austin, Texas
Reveley, M.A. Austin, Texas
Rha, C.H. Korea, Kwangju
Ribó, G. Barcelona, Spain
Rice, T. New Haven, Connecticut
Richardson, R.H. Austin, Texas
Richmond, R.C. New York, New York
Riese, R. Long Beach, California
Rifaat, O.H. U.A.R., Giza
Rijnsburger, T. Netherlands, Leiden
Rinehart, R.R. San Diego, California
Risch, P. Switzerland, Zürich
Ristow, H. New Haven, Connecticut
Rivera, M. New York, New York
Rivera, M.L. Spain, Barcelona
Rivera, R. New York, New York
Rizki, R.M. Ann Arbor, Michigan
Rizki, T.M. Ann Arbor, Michigan
Robbins, L. Seattle, Washington
Robert, M. Germany, Saarbrücken
Roberts, D.B. England, Leeds
Roberts, R. Chicago, Illinois
Robertson, A. Scotland, Edinburgh
Robertson, E. Scotland, Edinburgh
Robertson, K. Houston, Texas
Robinson, A. Moscow, Idaho
Rockwell, R.F. Dayton, Ohio
Rockwood, S. Tucson, Arizona
Rodinò, E. Italy, Padova
Rojas, E. Chile, Santiago
Rokop, S. San Diego, California
Romrell, L.J. Logan, Utah
Rosenbluth, R. Canada, Vancouver
Rosenfeld, A. Seattle, Washington
Rosenthal, N. Berkeley, California
Rotter, D. DeKalb, Illinois
Ruch, P. Switzerland, Zürich
Ruderer-Doschek, E. Austria, Vienna
Rumsower, L. Duarte, California
Runge, R. Lake Forest, Illinois
Russell, J. Tucson, Arizona
Sack, C. East Lansing, Michigan
Saeki, T. Japan, Chiba
Saito, C. Japan, Chiba
Sakaguchi, B. Japan, Fukuoka
Sakai, K.I. Japan, Misima
Sakar, D.N. India, Varanasi
Sakoyama, Y. Japan, Osaka
Salas, E. Spain, Madrid
Salceda, V. Mexico, Mexico City
Sander, K. Germany, Freiburg
Sanders, T. Canada, Vancouver
Sandler, L. Seattle, Washington
Sanford, R. Galesburg, Illinois
Sankaranarayanan, K. Netherlands, Leiden
Santamaría, P. Spain, Madrid
Santos, E.P. dos Brasil, São Paulo
Sanyal, C. India, Calcutta
Saraswathy, T. India, Varanasi
Sarmiento, L.A. Pasadena, California
Sasaki, F. Japan, Tokyo
Saul, S. Rochester, New York
Saura, A. Finland, Helsinki
Savolainen, S. Finland, Turku
Savontaus, M.-L. Finland, Turku
Schabtach, E. Eugene, Oregon
Schalet, A. Storrs, Connecticut
Scharloo, W. Netherlands, Leiden

Scheer, J.M. Ithaca, New York
Scheid, W. Germany, Münster
Scheidt, G.C. Pasadena, California
Schewe, M. Canada, Vancouver
Schlager, P. Notre Dame, Indiana
Schouten, S.C.M. Netherlands, Utrecht
Schneider, K. Trenton, New Jersey
Schultz, E.G. Brasil, Pôrto Alegre
Schwalm, F. Notre Dame, Indiana
Schweizer, P. Switzerland, Zürich
Schwinck, I. Storrs, Connecticut
Sciandra, R. Buffalo, New York
Seaman, R. Fort Collins, Colorado
Searcy, K.B. Pasadena, California
Searle, N. Bellingham, Washington
Šedivá, B. Czechoslovakia, Prague
Seecof, R.L. Duarte, California
Sega, G.A. Baton Rouge, Louisiana
Seiger, M.B. Dayton, Ohio
Seki, T. Japan, Osaka
Sene, F.M. Brasil, São Paulo
Sengün, A. Turkey, Istanbul
Settegast, M. Houston, Texas
Ševela, A. Czechoslovakia, Brno
Sewell, D.F. England, Sheffield
Seybold, W.D. Pasadena, California
Shaffer, P.V. Stony Brook, New York
Shamay, E. Israel, Jerusalem
Shannon, M.P. Austin, Texas
Shearn, A. New Haven, Connecticut
Sheldon, B.L. Australia, Sydney
Shen, M.W. Austin, Texas
Sheppard, P.M. England, Liverpool
Sherald, A. Charlottesville, Virginia
Shidler, D. Lafayette, Indiana
Shifley, B.C. Columbus, Ohio
Shima, T. Japan, Sapporo
Shindo, N. Japan, Tokyo
Shine, D.H. Lubbock, Texas
Shiomi, T. Japan, Nagasaki
Shoeb, Y. U.A.R., Alexandria
Shorrocks, B. England, Leeds
Shoupp, W. Morgantown, West Virginia
Sick, K. Denmark, Copenhagen
Siervogel, R.M. Eugene, Oregon
Sillans, D. France, Lyon
Simmons, J.R. Logan, Utah
Simonsen, V. Denmark, Aarhus
Singeisen, C. Switzerland, Zürich
Singh, B.N. India, Varanasi
Skala, L. Ithaca, New York
Skavaril, R.V. Columbus, Ohio
Sladká, D. Czechoslovakia, Brno
Slatis, H.M. East Lansing, Michigan
Slawson, J. Long Beach, California
Slimmers, S.A. Netherlands, Leiden
Slizynska, H. Scotland, Edinburgh
Smit, A.M. Pasadena, California
Smith, D.A. England, Birmingham
Smith, J. Chapel Hill, North Carolina
Smith, J.E. Columbus, Ohio
Smith, M.J. Northridge, California
Smith, M.K. Ann Arbor, Michigan
Smith, P.D. Storrs, Connecticut
Smith, R.C. Lexington, Kentucky
Sobels, F.H. Netherlands, Leiden
Sokoloff, A. San Bernardino, California
Somero, M.G. Canada, Vancouver
Sorsa, M. Finland, Helsinki
Sorsa, V. Finland, Helsinki
Souza, H.M.L. de Brasil, São Paulo
Spassky, B. New York, New York
Sperlich, D. Austria, Vienna
Spieth, H.T. Davis, California
Spieth, P.T. Eugene, Oregon
Spofford, J.B. Chicago, Illinois
Sprackling, L.E. Fort Collins, Colorado
Springer, R. Austria, Vienna
Šrám, R.J. Czechoslovakia, Prague
Stafford, D.W. Chapel Hill, North Carolina
Stalker, H.D. St. Louis, Missouri
Stauffer, H. Berkeley, California
Steegen, J.J. Netherlands, Haren
Steen, I. Norway, Oslo
Steen, L. van der Netherlands, Leiden
Steen, R. Charlottesville, Virginia
Steffensen, D.M. Urbana, Illinois
Steiner, E. Switzerland, Zürich
Steinfeld, R. Stony Brook, New York
Stern, C. Berkeley, California
Stocker, A.J. Dallas, Texas
Střebická, M. Czechoslovakia, Brno
Strickberger, M.W. St. Louis, Missouri
Strid, L. Sweden, Uppsala
Stuart, F. Eugene, Oregon
Stunell, M. Houston, Texas
Sturtevant, A.H. Pasadena, California
Subbarao, S.K. Urbana, Illinois
Sugimoto, K. Japan, Hiroshima
Suomalainen, E. Finland, Helsinki
Surver, W.M. Notre Dame, Indiana
Suter, K. Switzerland, Zürich
Suzuki, D. Canada, Vancouver
Swadley, J. Morgantown, West Virginia
Tabatabaie, H. Canada, Vancouver
Taira, T. Japan, Tokyo
Takada, H. Japan, Sapporo
Takahashi, Y. Japan, Sapporo
Takaya, H. Japan, Kobe
Tammisola, J. Finland, Turku
Tancock, R. Australia, Adelaide
Tantawy, A.O. U.A.R., Alexandria
Targa, H.J. Brasil, São Paulo
Tasaka, E. Canada, Vancouver
Tates, A.D. Netherlands, Leiden
Tattantire, A.C. Uganda, Kampala
Tawara, K. Japan, Fukuoka
Taylor, C. Columbus, Ohio
Taylor, W.C. Seattle, Washington
Tedeschi, M.V. Brasil, São Paulo
Teissier, G. France, Gif-sur-Yvette
Tengblad, E. Sweden, Uppsala

• Thoday, J.M. England, Cambridge
Thomas, R. New York, New York
Thomasson, W.A. Pasadena, California
• Thompson, J.M.Jr. Oklahoma City, Oklahoma
Thompson, S.R. Ithaca, New York
Thörig, G.E.W. Netherlands, Haren
Throckmorton, L.H. Chicago, Illinois
Thurlow, B.J. England, Chalfont St. Giles
Thurman, J. St. Louis, Missouri
Tigerstedt, P. Finland, Helsinki
Tiivola, A. Finland, Helsinki
Tillmann, J. Eau Claire, Wisconsin
Tjoa, F.H.B. Netherlands, Leiden
Tobajas, J. Spain, Madrid
Tobari, I. Japan, Chiba
Tobari, Y.N. Japan, Tokyo
Tobler, J.E. Logan, Utah
Tokunaga, C. Berkeley, California
Toledo, J.S. de T. Brasil, São Paulo
Tomita, T. Japan, Nagoya
Tomonaga, K. Japan, Nagasaki
Tonomura, Y. Japan, Tokyo
Tonzetich, J. Durham, North Carolina
Toribio, P. Duarte, California
Torres, A. Houston, Texas
Torroja, E. Spain, Madrid
Touzet, J. France, Lyon
Tracy, M. Providence, Rhode Island
Tracy, U.W. Pasadena, California
Traut, A. Germany, Münster
Traut, H. Germany, Münster
Triay, F.G. Scotland, Edinburgh
Trigueros, M. Mexico, Mexico City
Trout, W.E. III Duarte, California
Tsacas, L. France, Gif-sur-Yvette
Tsakas, S. Greece, Athens
Tsuno, K. Japan, Tokyo
Tuinstra, E.J. Netherlands, Utrecht
Turner, S.H. Austin, Texas
U, R. Durham, North Carolina
Uchibori, M. Japan, Hiroshima
Ulrich, E. Switzerland, Zürich
Ulrich, H. Switzerland, Zürich
Ulrich, V. Morgantown, West Virginia
Ulrichs, P.C. Berkeley, California
Urbischek, E.F. Wales, Swansea
Ursprung, H. Switzerland, Zürich
Vaidya, V.G. India, Poona
Valencia, R.M. Argentina, Buenos Aires
Valentin, J. Sweden, Stockholm
Van Deventer, M. Netherlands, Utrecht
Van Dyke, D. DeKalb, Illinois
Van Herrewege, C. France, Lyon
Van Herrewege, J. France, Lyon
Vann, E.G. Detroit, Michigan
Van Valen, L. Chicago, Illinois
Varak, E. Ithaca, New York
Vepsäläinen, K.M. Finland, Helsinki
Vicente, L. Spain, Madrid
Viinikka, Y. Finland, Turku
Vítek, J. Czechoslovakia, Brno
Vlist, J. van der Netherlands, Leiden
Voelker, D. Austin, Texas
Voelker, R.A. Austin, Texas
von Borstel, R.C. Oak Ridge, Tennessee
Von Halle, E.S. Oak Ridge, Tennessee
Voss, R. Israel, Jerusalem
Vydelingum, N. England, London
Waddington, C.H. Scotland, Edinburgh
Wakahama, K.-I. Japan, Matsue
Wakil, M.A. U.A.R., Alexandria
Walker, S. England, Cambridge
Walker, S. England, Liverpool
Wallace, B. Ithaca, New York
Ward, B. Tucson, Arizona
Ward, C.L. Durham, North Carolina
• Ward, R.D. England, Cambridge
Wass, J.A. DeKalb, Illinois
Watanabe, I. Japan, Chiba
Watanabe, S. Austin, Texas
Watanabe, T.K. Japan, Misima
Watson, W.A.F. Scotland, Aberdeen
Watt, B.J. Australia, Bundoora
Wattiaux, J.M. Belgium, Namur
Wearden, S. Morgantown, West Virginia
Weideli, H. Switzerland, Zürich
Weinmann, R.S. Ames, Iowa
Weisbrot, D.R. Binghampton, New York
Welshons, W.J. Ames, Iowa
Westphal, N.J. Lincoln, Nebraska
Wheeler, M.R. Austin, Texas
White, P. Storrs, Connecticut
Whitney, J.B. III Chapel Hill, N. Carolina
Whittinghill, M. Chapel Hill, N. Carolina
Widmer, B. Switzerland, Zürich
Wieschaus, E. New Haven, Connecticut
Wilkerson, R.D. Oak Ridge, Tennessee
Williams, G. Berkeley, California
Williams, J. III Baton Rouge, Louisiana
Williams, P. Australia, Adelaide
Williams, S. Ann Arbor, Michigan
Williamson, D.L. Philadelphia, Pennsylvania
Williamson, J.H. Canada, Calgary
Williamson, R. Canada, Vancouver
Willis, W. Canada, Vancouver
Willott, D. England, Chalfont St. Giles
Wilson, F.D. Austin, Texas
Wilson, L.D. Austin, Texas
Wilson, M. St. Louis, Missouri
Wilson, M.S. Austin, Texas
Wing, M. Austin, Texas
Winge, H. Brasil, Pôrto Alegre
Winicur, S. Pasadena, California
Winslow, R. Houston, Texas
Wiseman, P. Knoxville, Tennessee
Wolf, T. Detroit, Michigan
Wolff, M.L. Washington D.C.
Wong, P. Duarte, California
Wood, R.E. Logan, Utah
Woodruff, R.C. Logan, Utah
Worton, R. New Haven, Connecticut
Wright, C. Buffalo, New York

Wright, T.R.F. Charlottesville, Virginia
Wu, D. Austin, Texas
Wui, I.S. Korea, Kwangju
Würgler, F.E. Switzerland. Zürich
Xavier, J. Brasil, Pôrto Alegre
Yamada, M.A. Japan, Misima
Yamaguchi, O. Japan, Tokyo
Yamazaki, H.I. Japan, Tokyo
Yamazaki, T. Chicago, Illinois
Yanders, A.F. Columbia, Missouri
Yang, H.L. Austin, Texas
Yasbin, R. Ithaca, New York
Yasuda, N. Japan, Chiba
Yasuzumi, F. Ann Arbor, Michigan
Yoo, B.H. Australia, Sydney
Yoshikawa, I. Japan, Nagasaki
Young, S.S.Y. Columbus, Ohio
Young, W.J. Burlington, Vermont
Youssef, M.K. U.A.R., Alexandria
Ytterborn, K.H. Sweden, Stockholm
Yu, R. DeKalb, Illinois
Yuan, L.C. Pasadena, California
Yundt, J.C. Baton Rouge, Louisiana
Zack, N. Cleveland, Ohio
Zalokar, M. France, Gif-sur-Yvette
Zamburlini, P. Italy, Padova
Zanete, V.A. Brasil, Pôrto Alegre
Zarate, E. Chile, Santiago
Zimmering, S. Providence, Rhode Island
Zimmermann, G. Germany, Tübingen
Zita, M. Eau Claire, Wisconsin
Zouros, E. Chicago, Illinois
Zouros, E.G. Greece, Athens
Zowarka, B. San Marcos, Texas
Zudová, Z. Czechoslovakia, Prague
Zuill, E. England, Oxford

ALPHABETICAL DIRECTORY APPENDIX

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Aberle, A., Germany, Berlin
Adelsberger, H., Germany, Berlin
Anders, A., Germany, Giessen
Anderson, B.S., St. Paul, Minnesota
Baker, E.P., Australia, Sydney
Bay, D., Washington, D.C.
Belitz, H.J., Germany, Berlin
Casey, L., Amherst, Massachusetts
Childress, D., St. Paul, Minnesota
Comley-Frye, S.H., Bloomington, Indiana
Comstock, R.E., St. Paul, Minnesota
Ericksson, M., Sweden, Umeå
Fahrig, R., Germany, Freiburg
Goldstein, E.S., St. Paul, Minnesota
Hammar, I., Sweden, Umeå
Hamzahussain, B., India, Mysore
Hartl, D.L., St. Paul, Minnesota
Henze, M., Germany, Giessen
Hexter, W.M., Amherst, Massachusetts
Holmgren, P., Sweden, Umeå
Ives, P.T., Amherst, Massachusetts
Jacobs, M.E., Goshen, Indiana
Kezer, J., Eugene, Oregon
Kliesch, U., Germany, Berlin
Krikortz, M., Sweden, Umeå
Krishnamurthy, N.B., India, Mysore
Lambertsson, A., Sweden, Umeå
Lüers, H., Germany, Berlin
Manny, E., St. Paul, Minnesota
McFarlane, J.L., Riverside, California
Merriam, J.R., Los Angeles, California
Montell, I., Sweden, Umeå
Nirmala Sajjan, S., India, Mysore
Nöthel, H., Germany, Berlin
Olofsson, E., Sweden, Umeå
Palmer, W., Washington, D.C.
Parker, D.R., Riverside, California
Persson, K., Sweden, Umeå
Plough, H.H., Amherst, Massachusetts
Prout, T., Riverside, California
Puckett, L., St. Paul, Minnesota
Rajasakarasetty, M.R., India, Mysore
Rajeshwari, O., India, Mysore
Rasmuson, M., Sweden, Umeå
Rasmusson, B., Sweden, Umeå
Russell, P., Amherst, Massachusetts
Ryan, J., Riverside, California
Schneider, I., Washington, D.C.
Snyder, L.A., St. Paul, Minnesota
Södergren, A., Sweden, Umeå
Sreerama Reddy, G., India, Mysore
Stewart, B., Los Angeles, California
Stubblefield, P., Riverside, California
Svahlin, H., Sweden, Umeå
Tiffany, B., Amherst, Massachusetts
Vogel, E., Germany, Freiburg
Weber, Mrs., Germany, Berlin
Wolf, B., Germany, Giessen
Yost, H.T., Amherst, Massachusetts
Zimmerman, W.F., Amherst, Massachusetts