DROSOPHILA INFORMATION SERVICE

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OTHER DROSOPHILA SPECIES Stock Lists

TECHNICAL NOTES

ANNOUNCEMENT

Announcing the forthcoming publication of "The Genetic Variations of Drosophila melanogaster" by D. L. Lindsley and E. H. Grell. The volume is being published by the Carnegie Institution and current estimates of the publication date are the second half of March. Orders may be placed directly with the Office of Publications, Carnegie Institution of Washington, 1530 P. Street, N. W., Washington, D. C., 20005. The price has not been determined; orders placed now will be invoiced at the time the orders are filled.

Editor's Note: In this issue of DIS, the stock list from Oak Ridge National Laboratory (43:23) has used the nomenclature adopted for the above book.

Comment from the Editor

This issue is being processed by the Drosophila Group at Oregon, while the Editor is on sabbatical in Australia. Many thanks to Mrs. Frances Stuart, Mrs. Elizabeth Ehrlich and Mrs. Martha Kaplan.

All items should be typewritten, double-spaced, and modeled on the form used in the present issue. Distinguish between the number 1 and the letter l when there is any ambiguity by underlining the letter l. Be sure to proofread the typed copy before sending it to us.

Enclose, on separate sheets (except that those outside the United States may combine information of different types on one sheet if this seems desirable from the standpoint of economy in mailing) identified by institution, the following items:

1. Stock list, melanogaster.
2. New mutants, melanogaster.
3. Linkage data, melanogaster.
4. Stock lists, other species.
5. New mutants, other species.
6. Linkage data, other species.
7. Research notes. Observe carefully DIS format. Illustrations, charts and half-tones may be included, but it should be kept in mind that these will be photographed directly off the contributor's copy and will be reduced 10% during reproduction. Please order reprints when contributions are sent in. The cost of half-tone illustrations are:

Size 3X4 inches,	$ 5.00
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   As explained on page 8 of DIS 38, each worker will be given gratis the equivalent of a half page of DIS for his contributions, either singly or as a co-author. Any space in excess of that amount will be charged at the rate of $20.00 per page or fraction thereof. Accompanying each bill for page costs will be a statement avowing lack of funds for payment which the contributor may sign in lieu of payment. This is meant particularly for contributors outside the U. S. where research funds may not be quite as available as in the U. S. or where currency exchange problems make payments difficult It is hoped that all Drosophila workers in the position to do so will look upon these charges as a welcome means of meeting the continually increasing cost of publishing DIS.
8. Technical notes. See remarks under 7.
9. Teaching notes. See remarks under 7.
10. Personal and laboratory news.
11. Material requested or available.
12. Announcements.
13. Bibliography
14. Quotability of notes. Until a more satisfactory solution to the problem of quoting DIS notes is found, we will list those notes which you are willing to have quoted, without prior permission, from all previous issues. The information in these notes should be essentially correct, or unchanged, to the best of your knowledge. If a subsequent note by you or someone else modifies the content of the note (as by changing a locus, a symbol, or a mutant designation) then that note should follow the first in parentheses. If more than one note appears on a single page, the first is lettered a, the second b, etc.

PASADENA, CALIFORNIA: CALIFORNIA INSTITUTE OF TECHNOLOGY

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

Wild Stocks

1 Canton-S
* Florida 800
2 Lausanna-S
3 Oregon-R-C
4 Swedish-c
5 Urbana-S

Chromosome 1

* ac 170
6 ac^3 w^a
7 amx/FM3, y^{31d} sc^8 dm B l
8 amx lz^g/y f:=
* amx^{55} . . . . . (see lz^K)
9 Ax
* bb . . . . . 24, 59, etc.
* bb^{G3} 130
* bb^1 . . . . . 710, 753
* bb^N 164
* bb^{poi} . . . . . (see bb^{G3})
10 B
11 B Bx^r car/y f:=
* B^3 39
* B^iB^i 42
* BB 40
* BB^{36b} 41
12 Bg B/In(1)AM
13 bi ct^6 g^2
14 bo v
15 br
16 br w^e ec rb t^4/FM1, y^{31d} sc^8 w^a lz^s B
* br^3 651
17 Bx_2
18 Bx^2
19 Bx^3
20 Bx^J
* Bx^r 11
* Bx^{r49k} 141
21 car
* cho 97
* cho^2 182
22 cm
23 cm ct^6
* Co 179
24 cs^{53}/y w bb
* ct^6 . . . . . 13, 23, etc.
* ct^K 171
25 ct^n oc/FM1, y^{31d} sc^8 w^a lz^s B
* cu-X 759
* cv . . . . . 98, 99, etc.
26 cx
27 cx^{tg} oc/FM1, y^{31d} sc^8 w^a lz^s B
28 dm/y f:=
29 dor/y f:=
30 dor^1/FM6, y^{31d} sc^8 dm B (nub/+); see also 736
31 dow/FM6, y^{31d} sc^8 dm B
* dx 35, 122
* dx^{st} 651, 652
32 dy
* e(bx) . . . . (= en-bx) 760
* $e(bx)^2$ . . . . (= en^2-bx) 661
* $e(S)^X$ . . . . (= en^X-S) 653
33 ec
34 ec ct^6 s car/FM6, y^{31d} sc^8 dm B
35 ec dx
* eq 98, 709
36 Ext/FM6, y^{31d} sc^8 dm B
37 f
38 f B/y f:=
39 f B^3/y f:=
40 f BB/y f:=
41 f BB^{36b}/y f:=
42 f B^iB^i/y f:=
43 f fu/y f:=
* f^3 164
* f^5 154
44 f^{36a}
* f^{B15} 748
* f^{B27} 749
45 fa
* fa^n 755
* flp . . . . . (see flw)
46 flw
47 fo
48 fx/y f:=
* fu 43
* fw 143, 167
* fw^{34e} 189
49 g^2
50 g^2 pl/FM3, y^{31d} sc^8 dm B l
51 g^2 ty/y f:=
* g^4 . . . . . 654, 705, etc.
52 gg^2/FM6, y^{31d} sc^8 dm B
53 gg^3
54 gt w^a
* Hw . . . . . 110, 654, etc.
55 Hw^{49c}/FM1, y^{31d} sc^8 w^a lz^s B
56 if^3
57 kz
* l(1)7 . . . . . (see dor^1)
58 l(1)J1 sc^{J1}/l(1)J1 sc^{J1} Dp(1;f)24
59 lh B car bb/y f:=

60 lz/FM3, y^{31d} sc^8 dm B l
61 lz^3/y f:=
62 lz^{34}/y f:=
63 lz^{36}/y f:=
64 lz^{37}
65 lz^{48f}/y f:=
* lz^{50e} 739
66 lz^{BS} lz^{46} lz^g ras^4 v/y f:=
* lz^D 654
* -lz^g . . . . . 8, 175, etc.
67 lz^K
* lz^s . . . . . 16, 25, etc.
* lz^{y4} 115, 739
68 m
* m^2 . . . . . 654, 705, etc.
69 m^D/FM3, y^{31d} sc^8 dm B l
70 M(1)n/FM6, y^{31d} sc^8 dm B
71 M(1)o f/FM6, y^{31d} sc^8 dm B
72 $M(1)o^{Sp}$/FM6, y^{31d} sc^8 dm B
* M(1)Sp . . . . . .(see $M(1)o^{Sp}$)
73 mal/y f:=
* mal^{bz} 178
74 na/y f:=
75 ny f/FM1, y^{31d} sc^8 w^a lz^s B (ri)
76 oc ptg^3/ClB
* od . . . . . (see os^o)
77 os^o
78 os^s
79 pa sn^3/FM6, y^{31d} sc^8 dm B
80 peb v
* pl 50
* pn . . . . . 105, 172, etc.
81 pn^2
* pn^3 101
* ptg 662
82 ptg^2
* ptg^3 76
* ptg^4 138
83 r^9/y f:=
* r^{39k} f B/In(1)AM
85 ras dy
86 ras^2
87 ras^3 m
* ras^4 66
* ras^v 814
88 rb
89 rb cx
90 rg
91 rst^2/FM1, y^{31d} sc^8 w^a lz^s B
92 rux/FM6, y^{31d} sc^8 dm B
93 rux^2
94 s
95 sbr
96 sc
97 sc cho t
98 sc cv v eq
99 sc cv v f
100 sc ec cv ct^6 v g^2 f/FM3, y^{31d} sc^8 dm B l
101 sc pn^3 g^{2rv} Bx^2 . . . . . (g^2 reverted)
102 sc z ec ct^6
103 sc z w^{17G2} ec ct^6
104 sc^2
105 sc^2 pn/y f:=
106 sc^{3B}
107 sc^{3-1} w/y f:=
* sc^4 765
108 sc^5
109 sc^6 w^a
* sc^7 . . . . . .695, 767, etc.
* sc^8 . . . . . 757, 772, etc.
* sc^9 776
* sc^{10} . . . . . (see ac^3)
110 sc^{10-1}/y Hw
* sc^{19} 808
* sc^{D2} 177
* sc^{J1} 58
* sc^{J4} 742
* sc^{S1} . . . . . .173, 740, etc.
* sc^{S2} 807
* sc^{260-14} 777
* sc^{260-15} 815
* sc^{260-22} 778
111 scp t
112 sd
113 shf^2
* sl (in ClB, ClB^{36d})
* sl^2 696
* sn 688
* sn^2 158
114 sn^3
115 sn^3 lz^{y4} v/y f:=
116 sn^4
117 sn^{34e}
118 sn^{36a}/y f:=
* sp-w. . . . . (see w^{sp})
119 spl
120 sta/FM3, y^{31d} sc^8 dm B l
121 sta/y f:=
* su(Cbx) 660
122 su(dx) dx
* su(f) . . . . . .(= su^w-f). 142
123 $su(s)^2$ v (bw)
124 $su(s)^2$ w^a cv t
125 $su(s)^3$ cv v f/FMA3, y^2 (bw)
126 $su(s)^S$ v/FMA3, y^2 (bw)
* $su(w^a)$ . . . . (= su-w^a) . 682, 683, etc.
* su^{S2}-v-pr. . . . . (see $su(s)^S$)
127 svr
128 svr w^a
129 svr^{poi}
130 $svr^{poi-dish}$ bb^{G3}
131 sw
132 sx vb^2 os^s/FM6, y^{31d} sc^8 dm B
* sy . . . . . .(see os^s)

133 t
134 t^2 v f
135 t^3
* t^4 16
136 t^5 v r^{12}
* tuh-1 . . . . .(= tu-h) 658
137 tw/FM1, y^{31d} sc^8 w^a lz^s B
* ty 51
* tyl . . . . . .(= ty-1) 752, 753
138 un Bx^2/In(1)AM, ptg^4
139 un^4
140 v
141 v f Bx^{r49k} car/y f:=
142 v f su(f)
143 v^2 fw
144 v^{36f}
* v^{0f} 754
145 vb
* vb^2 132
146 vs
147 w
148 w m f
149 w sn^3 m
* w^{11E4} 194
* w^{17G2} 103
150 w^a
151 w^{a2}
152 w^{a3}
153 w^{a4}
154 w^{bf} f^5
155 w^{bf2}
* w^{bf3} 679
156 w^{Bwx}
157 w^{ch} wy_2
158 w^{co} sn^2
159 w^{col}
160 w^e
161 w^{e2}
162 w^{ec3}
163 w^h
164 w^i f^3 bb^N
165 w^{sat}
166 w^{sp}
167 w^t fw
168 w^u
* wy^2 187
169 y
170 y ac_v
171 y ct^K
172 y pn
173 y pn w cm ct^6 sn^3 oc ras^2 v dy g^2 f os^o car sw/In(1)sc^{S1}, In(1)dl-49, y v B
174 y sc
175 y sc_5 lz^g v f/y f:=
176 y sc^5
177 y sc^{D2}
178 y v f mal^{bz}
179 y w Co/y f:=
180 y w spl
181 y^2
182 y^2 cho^2
183 y^2 cv v f
184 y^2 sc w^a ec
185 y^2 w^a
186 y^2 w^a w
187 y^2 wy^2 g^2 (g^2 partly reverted)
188 y^{2S}
189 y^{2S} fw^{34e}
190 y^{3d}/y f:=
* y^{3P} 782
* y^4 784
* y^{31d} . . . . . 7, 16, etc.
191 y^{34c}
* y^{59b} 684
192 y^{td}
193 y^{v2}
194 z w^{11E4}

Chromosome 2

195 a px or
196 a px sp
197 ab_2
198 ab^2/T(Y;2)E
199 ab^2 ix^2 bw sp^2/In(2L+2R)Cy, Cy dp^{lvI} Bl L^4 sp^2
* abb 397
200 abr/In(2L+2R)Cy, Cy hk^2
201 abr/SM5, al^2 Cy lt^V sp^2
202 ad
203 al
204 al b c sp^2
205 al dp b bw l(2)ax/SM5, al^2 Cy lt^V sp^2
206 al dp b pr ap^{blt} bw/SM5, al^2 Cy lt^V sp^2
207 al dp b pr Bl c px sp/SM1, al^2 Cy sp^2
208 al dp b pr c px sp
209 al dp b pr Hx
210 al S ast ho/SM1, al^2 Cy sp^2
* al^2 . . . . . .205, 206, etc.
* alpha-1 . . . . . (see tyr-1)
211 Alu
212 an/SM5, al^2 Cy lt^V sp^2
213 an^2/SM1, al^2 Cy sp^2
214 ang
215 ant(ro)
216 ap^4/SM5, al^2 Cy lt^V sp^2
217 ap^{blt}
218 arch ch1/SM5, al^2 Cy lt^V sp^2
219 ast ho cl
* ast^3 785
220 ast^4 dp cl
* ast^X 295
221 At
222 b
223 b tyr-1
224 b cn beta
225 b el rd^s pr cn

226 b Go/In(2LR)Gla
227 b Go/SM5, al Cy lt^v sp^2
228 b gp
229 b j
230 b l(2)Bld pr_2c px sp/SM5, al^2 Cy lt^v sp^2
231 b lt wx^{wxt} bw
232 b nub pr
233 b pr tk/T(Y;2)G
234 b sf
235 b vg
* ba 246
236 Bl/In(2L+2R)Cy, Cy bw^{45a} sp^2 or^{45a}
237 Bl/T(2;3)dp
238 Bl L^2/SM5, al^2 Cy lt^v sp^2
239 Bl stw^{48} ap^{blt} tuf sp/SM5, al^2 Cy lt^v sp^2
240 Bla/SM5, al^2 Cy lt^v sp^2
241 blo
* blt. . . . . (see ap^{blt})
242 bri
* bs_2. 375
243 bs^3
* bs^3. 323
244 bur fs(2)El/SM5, al^2 Cy lt^v sp^2
245 bw
246 bw ba
247 bw tu
248 bw^{2b}
249 bw^4
* bw^{45a}. 236
250 bw^D
* bw^{V1} . . . . . 323, 349, etc.
* bw^{V32g} 348, 711
* bw^{V34k} 337
251 c
252 c wt px
253 cg c/SM5, al^2 Cy lt^v sp^2
254 cg c/In(2LR)U
255 ch
256 chl
257 chl en/SM5, al^2 Cy lt^v sp^2
258 chl l(2)bw bw^{2b} mr^2/SM5, al^2 Cy lt^v sp^2
259 chy
260 ck/SM5, al^2 Cy lt^v sp^2
261 cl
262 cl^2/T(Y;2)E
263 cn
* cn^2. . . (in all stocks containing In(2R)Cy)
264 cn bw
265 cn en/SM5, al^2 Cy lt^v sp^2
266 cn l(2)crc/SM5, al^2 Cy lt^v sp^2
267 cn^3/T(Y;2)C
268 cn^{35k}
* cq . . . . . (see rk^4)
269 cru/In(2L+2R)Cy, Cy (w^e)
270 d/SM5, al^2 Cy lt^v sp^2
271 d b/SM5, al^2 Cy lt^v sp^2
272 da/SM1, al^2 Cy sp^2
273 dil^2 hv bw sp/SM5, al^2 Cy lt^v sp^2
274 dke c
275 dp
276 dp_2cn bw
* dp^2 . . . . . (see dp^{lv2})
* dp_D 842
277 dp^{lv} b/SM5, al^2 Cy lt^v sp^2
* dp^{lv2} . . . . . 287, 288, etc.
* dp^{lvI} 199
* dp^{Nov} . . . . . (see dp^{ovN})
278 dp^o
279 dp^{o2}
280 dp^{olvR}/SM5, al^2 Cy lt^v sp^2
281 dp^{ovN}
* dp^{Rf}. . . . . (see dp^{olvR})
* dp^{Th}. . . . . (see dp^{lvI})
* dp^{tx}. . . . . (see dp^{lv})
* dp^v. 671
282 dp^{v2}
* dp^{vl}. . . . . (see dp^{vM})
283 dp^{vM}/SM5, al^2 Cy lt^v sp^2
284 ds dp
285 ds^{rv} ft dp^{v2} l(2)M b pr/SM5, al^2 Cy lt^v sp^2
286 ds S_4G b_L pr_2/In(2L+2R)Cy, al^2 Cy lt^3 sp^2
287 ds^W/In(2L)$Cy^{L}t^{R}$, Su(S) dp^{lv2} pr
* ds^{33k} . . . . . 323, 349, etc.
288 ds^{38k}/In(2L)Cy, Cy dp^{lv2} b pr
289 dsr
290 dw-24F cl/SM5, al^2 Cy lt^v sp^2
291 dw-24F l(2)cg, cg/SM5, al^2 Cy lt^v sp^2
* E(S). . . (= En-S). . . 330, 390, etc.
292 ed Su(dx)^2
293 el
* en 257, 265, 720
* esc 786
294 ex
295 ex ds S^X ast^X/SM1, al^2 Cy sp^2
* fes . . . . . (see fs(2)B)
296 fj l(2)Su(H)/SM5, al^2 Cy lt^v sp^2
297 fj wt/SM5, al^2 Cy lt^v sp^2
298 fr/In(2L+2R)Cy, Cy dp^{lv2}
299 fr^2 wt/SM5, al^2 Cy lt^v sp^2
300 Frd/In(2L+2R)Cy, Cy sp^2
* fs 2.1 . . . . . (see fs(2)E1)
301 fs(2)B Alu lt/SM5, al^2 Cy lt^v sp^2
* fs(2)El 244
302 ft
* G 286
303 G^{rv}/SM5, al^2 Cy lt^v sp^2
* Go. 226, 227
* gp 228
* gt-4 410
* Hia 432, 433
304 hk
305 hk pr
* hk^2 200
306 ho

307 hv/SM5, al^2 Cy lt^v sp^2
308 Hx; see also 209
309 hy/SM5, al^2 Cy lt^v sp^2
310 hy a px sp/SM1, al^2 Cy sp^2
* ix 369
* ix^2 199
311 j
312 J/In(2L)NS
313 J^{34e}
314 kn
315 L
316 L^2
317 L^4
318 L^5
319 L^G
320 L^r
321 L^{si}
* l(2)30l 362
322 l(2)39 a px slt sp/SM5, al^2 Cy lt^v sp^2
323 l(2)a bs^3, In(2L)t/bw^{V1}, ds^{33k}
* l(2)ax 205
324 l(2)ay b c sp^2/SM5, al^2 Cy lt^v sp^2
* l(2)Bld 230
* l(2)bw 258
* l(2)C 393
* l(2)cg 291
* l(2)crc. 266
325 l(2)gl a px or/SM5, al^2 Cy lt^v sp^2
326 l(2)H L^2/SM5, al^2 Cy lt^v sp^2
* l(2)M 285
327 l(2)mat/SM5, al^2 Cy lt^v sp^2
328 l(2)me/SM1, al^2 Cy sp^2
* $l(2)mr^2$. 710
* l(2)R 405
* l(2)Su(H). 296, 420
* ll. 358
329 ll^2
330 lm/In(2L+2R)Cy, Cy S^2 dp^{lv2} E(S)
331 lt/T(Y;2)A
332 lt std/SM2, al^2 Cy lt^v sp^2
333 lt^3 stw^3
* lt^3. . . . . 286, 830, 853
* lt^v. . . . . 201, 205, etc.
334 ltd
335 lw
* lys. 673
336 M(2)172/SM5, al^2 Cy lt^v sp^2
* M(2)B. . . . . (see $M(2)z^B$)
337 $M(2)e^s$/In(2L+2R)Cy, Cy, In(2R)bw^{V34}
338 $M(2)e^s$/SM5, al^2 Cy lt^v sp^2
339 $M(2)H^{S5}$/SM5, al^2 Cy lt^v sp^2
340 $M(2)l^2$/SM1, al^2 Cy sp^2
341 $M(2)m^{S6}$/SM5, al^2 Cy lt^v sp^2
342 $M(2)S2^3$/SM2, al^2 Cy lt^v sp^2
343 $M(2)S2^9$/SM5, al^2 Cy lt^v sp^2
* M(2)S3 . . . . . (see $M(2)S2^3$)
* M(2)S5 . . . . . (see $M(2)H^{S5}$)
* M(2)S6 . . . . . (see $M(2)m^{S6}$)
344 M(2)S7/SM5, al^2 Cy lt^v sp^2
* M(2)S9 . . . . . (see $M(2)S2^9$)
* M(2)S11 . . . . . (see $M(2)e^s$)
345 M(2)z/SM5, al^2 Cy lt^v sp^2
346 M(2)z Sk b/In(2L)Cy, Cy dp^{lv2} b pr
347 $M(2)z^B$/SM5, al^2 Cy lt^v sp^2
* Mal 676
348 mi/bw^{V32g}
349 mr bs^2/bw^{V1}, ds^{33k}
350 mr^2/In(2R)Cy, cn^2 Bld
351 msf/SM5, al^2 Cy lt^v sp^2
* N-2G . . . . . (= N-2) 407
352 net
353 net al ex ds S ast shv ho rub/SM1, al^2 Cy sp^2
354 net ed Su(dx)^2
* nub 232
355 nub^2
356 nw^2/In(2L)Cy, In(2R)NS
* or^{45}. 195, 325
* or^{45a}. 236
357 pd
358 pd ll
359 pd ll^2 sp
360 Pfd/SM5, al^2 Cy lt^v sp^2
361 pi/SM5, al^2 Cy lt^v sp^2
362 pi l(2)30l/SM5, al^2 Cy lt^v sp^2
* Pin 409
363 pk cn
364 pk tuf (sp^2/+)
* Pm. . . . . (see bw^{V1})
* Pm^2. . . . . (see bw^{V32g})
365 po vg
366 po^2
367 pr
368 pr cn/T(Y;2)C
369 pr cn ix/SM5, al^2 Cy lt^v sp^2
370 pr^{bw}
371 pu Gr
* Pu^{Gr} 846
372 puf
373 pw-c/SM5, al^2 Cy lt^v sp^2
374 px
375 px bs (old Berlin stock of Goldschmidt)
376 px bw sp/T(Y;2)J
377 px bw mr sp/bw^{V1}, ds^{33k}
378 px slt sp
379 pym/In(2L+2R)Cy, Cy
380 pys
381 Q
* rc 673
382 rd/SM5, al^2 Cy lt^v sp^2
* rd^s. 225
383 rdo
384 rdo^2 pr
* Rev. 793
* Rev^B 726
385 rh
386 rk^4

387 rl
* rn 847
* Roi 434
388 rub
389 Ruf/bw^{V1}, ds^{33k}
* Rvd. . . . . .(see Rev^{B})
390 S/In(2L+2R)Cy, Cy E(S) (homozygous for K-pn)
391 S Sp ab^{2} ltd/SM5, al^{2} Cy lt^{v} sp^{2}
* S^{2} 330, 744
392 S^{R}/bw^{V1}, ds^{33k}
* S^{X} 295
393 sca l(2)C/SM5, al^{2} Cy lt^{v} sp^{2}
394 SD-5/SM1, al^{2} Cy sp^{2}
395 SD-72/SM5, al^{2} Cy lt^{v} sp^{2}
* sf^{2}. 234
396 sf^{2}
397 shr bw^{2b} abb sp/SM5, al^{2} Cy lt^{v} sp^{2}
398 shv
399 shv ho
* Sk 346
* slt 322, 378
400 sm px/SM5, al^{2} Cy lt^{v} sp^{2}
401 sm px pd/SM5, al^{2} Cy lt^{v} sp^{2}
402 so
403 so^{2} b cn
* sp. . . . . .196, 207, etc.
404 sp^{2} bs^{2}
405 Sp/In(2L)t, l(2)R
406 Sp/SM5, al^{2} Cy lt^{v} sp^{2}
407 Sp Bl N-2G/SM5, al^{2} Cy lt^{v} sp^{2}
408 Sp J/SM5, al^{2} Cy lt^{v} sp^{2}
409 Sp J L^{2} Pin/SM5, al^{2} Cy lt^{v} sp^{2}
410 spd gt-4/SM5, al^{2} Cy lt^{v} sp^{2}
411 sple
412 spt
413 std/SM5, al^{2} Cy lt^{v} sp^{2}
414 stw
415 stw^{2}
416 stw^{3}/T(Y;2)B
417 stw^{5}
418 stw^{48} ap^{blt} tuf sp^{2}
* Su(dx) . . . . (= Su-dx) 652
* Su(dx)^{2}. . . . (= Su^{2}-dx). . .354, 651
* Su(er) . . . . (= Su-er) 675
419 Su(H)/In(2L+2R)Cy, Cy pr
420 Su(H) whd l(2)Su(H)/SM5, al^{2} Cy lt^{v} sp^{2}
* Su(S). 287
* tet. 655
421 Tft/SM1, al^{2} Cy sp^{2}
* Tg 788
* tk 233
422 tkd/SM5, al^{2} Cy lt^{v} sp^{2}
423 tkv
424 tri vg^{No2}/SM5, al^{2} Cy lt^{v} sp^{2}
* tu 247
* tu-36a . . . (= tu^{36a}) 592
425 tuf ltd
426 tyr-l (p^{p}); see also 223
427 Uf
428 vg
429 vg^{D}/SM5, al^{2} Cy lt^{v} sp^{2}
430 vg^{ni}
* vg^{No2} 424
431 vg^{np}
432 vg^{nw} Hia/SM5, al^{2} Cy lt^{v} sp^{2}
433 vg^{nw} Hia/T(2;3)S^{M}, In(2L+2R)Cy, Cy
434 vg^{U}/In(2L)t, Roi, In(2R)Cy, bw sp^{2} or
435 vst/SM5, al^{2} Cy lt^{v} sp^{2}
436 whd
437 wt
* wx^{wxt} . . . . . (= wxt) 231

Chromosome 3

438 a(3)26
* a-3 . . . . . .(see a(3)26)
439 aa h
440 aa tu-36e
441 abd
* Antp^{B} 796
442 app
443 as^{hg}
444 as^{hg} e^{s}
* Ata 834
445 bar-3
* Bd. 556
446 Bd^{G}/In(3R)C, l(3)a
447 bf/In(3R)C, e l(3)e
* bod 553
* bp. . . . . (see bul^{bp})
448 bul
449 bul^{bp}/TM1, Mé ri sbd^{1}
450 bv
* bx . . . . . .582, 596, etc.
451 bx^{3} Cbx Ubx bxd pbx/T(2;3) ap^{Xa}
452 bx^{34e}
* bxd 451, 839
* bxd^{107}. 867
* by 565, 566
* c(3)G . . . . (= c3G) 588
453 ca
454 ca bv
455 ca K-pn
456 ca^{2}
* cand. 488
* ca^{v} 488
457 Cbx
458 cd
459 cmp ca/In(3R)C, e
460 cp
461 cp in ri p^{p}
462 cu
463 cu kar
464 cur
465 cv-c
466 cv-c sbd^{2}
467 cv-d

553 ri bod e^s/In(3L)P, Mé, In(3R)C, Sb e l(3)e
554 ri p^p/T(Y;2;3)F, st
555 ro
556 ro Bd ca/In(3R)C, l(3)a
557 ro ra ca/T(2;3)Mé
558 rs^2
559 rsd
* rt^2. 575
560 ru
561 ru h th st p^p H e^s ro/In(3L+3R)P, M(3)x e^x
562 ru h th st cu sr e^s ca
563 ru h th st cu sr e^s Pr ca/T(2;3)Mé
564 ru h th st p^p cu sr e^s
565 ru lxd by
566 ru^g jv se by
567 ry
568 ry^2
569 Sb/In(3LR)Ubx^{101}, Ubx^{101}
570 Sb H/In(3R)C, cd
571 Sb Ubx/T(2;3)ap^{Xa}
572 Sb^{Spi}/In(3LR)Cx
* Sb^V 850, 851
* sbd 591
* sbd^{104}. 868
* sbd^{105}. 730
* sbd^1 . . . . . 449, 516, etc.
573 se
574 se h
575 se rt^2 th/In(3L)P, Mé
576 se ss k e^s ro
* sed . . . . . (see Hn^{r3})
* sep 546, 785, 850
577 Ser/In(3R)C, e l(3)e
578 snb
579 sr
580 sr gl
581 ss
582 ss bx $Su(ss)^2$
583 ss bxd k e^s/T(2;3)ap^{Xa}
584 ss^a
585 ss^{aB}
586 ss^{a40a}
587 st
588 st c(3)G ca/TM1, Mé ri sbd^1 (sp^2)
589 st in ri p^p
590 st Ki p^p
591 st sbd e^s ro ca
592 st sr e^s ro ca (tu-36a)
593 st sr H^2 ca/In(3R)P^W, st l(3)W ca
594 st^{sp}
* su(pd). . . . . (= su-pd) 662
595 $su(pr)^B$/In(3R)C, e (pr)
596 $su(Hw)^2$ bx bxd/TM1, Mé ri sbd^1 (sp^2)
* $Su(ss)^2$ . . . . (= Su^2-ss). 582
597 su(t) (t)
* su(tu) . . . . (= su-tu) 675
598 su(ve) ru ve h th
599 th
600 th st cp
601 th st pb p^p/In(3LR)Cx
602 th st pb p^p cu kar $su(Hw)^2$ jvl ss bx sr gl/TM1, Mé ri sbd^1
* tra 659
603 Tri/In(3LR)DcxF
604 tt wo
605 Tu (=Tubby)
* tu-36e . . . . . (= tu^{36e}) . . 440
* tuh-3 658
606 tx
607 Ubx e^4/In(3L+3R)P, Dfd ca
608 Ubx^{61d}/H^{57c}
* Ubx^{101}. 569
* Ubx^{130}. . . . . 476, 659, etc.
609 ve
610 ve h th
611 ve R/In(3L)P, gm
* vo-3 . . . . . (see $e(dp)^v$)
612 W
613 W Sb/In(3LR)Cx
614 We/In(3L)P, Mé, In(3R)C, e l(3)e
615 wk/In(3L+3R)P, Dfd ca
616 wo
* Xa. . . (= T(2;3)ap^{Xa}). . . .451, 534, etc.

Chromosome 4

617 ar/ey^D
618 bt
619 bt ey^R sv^n
620 bt^D/ci^D
621 Ce^2/spa^{Cat}
622 ci ey^R
623 ci ey^R sv^n
624 ci gvl bt
625 ci gvl ey^R sv^n
626 ci sv^n
627 ci^{36l}
628 ci^{57g}
629 ci^D/ey^D
630 ci^W
631 ey
632 ey^2
633 ey^4
* ey^D 617, 629, 649
* ey^R . . . . . 622, 623, etc.
634 gvl
635 gvl ey^R
636 $l(4)2^f$/ci^D (Hochman)
637 $l(4)4^c$/ci^D "
638 $l(4)6^b$/ci^D "
639 $l(4)14^b$/ci^D "
640 $l(4)15^2$/ci^D "
641 l(4)21/ci^D "
642 l(4)22/ci^D "
643 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 676
644 spa
645 spa^{Cat}/ci^D
646 spa^p
647 spa^{p65}
648 sv^{35a}
649 sv^{de}/ey^D
650 sv^n

Multichromosomal Stocks

651 br^3 dx^{st}; ed $Su(dx)^2$ (1;2)
652 dx^{st}; Su(dx) (1;2)
653 $e(S)^x$/FMA3, y^2; al S ast ho/SM1, al^2 Cy sp^2 (1;2)
654 lz^D/In(1)dl-49, m^2 g^4; bw^{V1}/In(2L+2R)Cy, Cy (1;2)
655 os^s; tet (1;2)
656 v; bw (1;2)
657 v; In(2R)bw^{VDel}/SM1, al^2 Cy sp^2
658 tuh-1; tuh-3 (1;3)
659 w^a v/FMA3, y^2; tra/In(3LR)Ubx^{130}, Ubx^{130} e^s (1;3)
660 y su(Cbx) v; Cbx (1;3)
661 y^2 $e(bx)^2$ w^{bf}/FMA3, y^2; sbd^2 ss bx^{34e}/TM1, Mé ri sbd^1 (1;3)
662 ptg; px pd; su(pd) (1;2;3)
663 y f:=; bw; e; ci ey^R (1;2;3;4)
664 y f:=; bw; e; spa^p (1;2;3;4)
665 al dp b Bl c px sp/In(2L+2R)Cy, Cy; D/In(3L+3R)P (2;3)
666 b Su(er)+ bw; st er (2;3)
667 bw; st (2;3)
668 bw^4; st (2;3)
669 bw^{V1}, dp b/In(2L+2R)Cy, Cy sp^2; Sb/In(3LR)DcxF (ru h ca?) (2;3)
670 bw^{V1}, ds^{33k}/In(2L+2R)Cy, Cy; H/In(3R)Mo, sr (2;3)
671 cn; ry^2 (2;3)
672 dp^v; $e(dp)^v$ (2;3)
673 lys rc; ss (2;3)
674 px pd; Pdr H, Dp(2;3)P/Pdr (2;3)
675 Su(er) tu bw; st er su(tu) (2;3)
676 pr; Mal (2;4)

Attached-X

677 br ec/y^{3d}
678 f B/$su(s)^S$
* FMA3, y^2 . . . . (= FMA3) . . 125, 126, etc.
679 w^{6f3}/ sn36a
680 y/g^2 ty
681 y pn/FM6, y^{31d} sc^8 dm B
* y pn v 684
* y v f 692
* y v f car 753
* y w bb 24
* y w f 756
* y^2 sc w^a ec 687
682 y^2 $su(w^a)$ w^a bb/y sc^{4L} sc^{8R}

Attached-XY

683 v f B, XY/y^2 $su(w^a)$ w^a bb
684 y^{59b} $su(w^a)$ w^a, XY^L·Y^S/y pn v (extra Y present)
685 Y^S/g^2 B·Y^L and y f:= (dp^{olv}) (Stern)
686 Y^S X·Y^L, In(1)EN, In(1)dl-49, Y^S y·Y^L/y X·Y; bw; e; ci ey^R

Triploid

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

Extra Y

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

Closed-X

689 In(X^{c2})w^{vc}/In(1)dl-49, y w^8 lz^s female; In(1)dl-49, y w lz^s/sc^8·Y male
690 X^{c2}, cv v f/ClB, v

Closed-Y

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

Deficiencies

Deficiencies-X

693 Df(1)259-4c — Df(1)259-4c/FM4, y^{31d} sc^8 dm B
694 Df(1)260-1 — Df(1)260-1/FM4, y^{31d} sc^8 dm B
695 $Df(1)B^{263-20}$ — $Df(1)B^{263-20}$/In(1)sc^7, In(1)AM, sc^7 car
696 Df(1)bb — Df(1)bb, y sl^2 bb-/FM4, y^{31d} sc^8 dm B
697 Df(1)bb — Df(1)bb, y v car bb-/In(1)AM
* $Df(1)bb^1$ 710
698 $Df(1)ct^{268-42}$ — $Df(1)ct^{268-42}$, y/FM4, y^{31d} sc^8 dm B
699 $Df(1)g^1$ — $Df(1)g^1$, f B/In(1)AM
700 Df(1)mal — Df(1)mal/In(1)dl-49, lz^s
701 $Df(1)N^8$ — $Df(1)N^8$/FM1, y^{31d} sc^8 w^a lz^s B
702 $Df(1)N^{264-39}$ — $Df(1)N^{264-39}$, w^{ch}/FM4, y^{31d} sc^8 dm B
703 $Df(1)N^{264-105}$ — $Df(1)N^{264-105}$/FM1, y^{31d} sc^8 w^a lz^s B
* $Df(1)rst^2$ 91
* $Df(1)sc^{4L}$ sc^{8R} 766
* $Df(1)sc^8$ 742
704 Df(1)svr — Df(1)svr, Dp(1;f)101, spl/y f:=
705 $Df(1)w^{258-11}$ — $Df(1)w^{258-11}$, y/In(1)dl-49, y Hw m^2 g^4
706 $Df(1)w^{258-42}$ — $Df(1)w^{258-42}$, y/FM1, y^{31d} sc^8 w^a lz^s B

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

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

Deficiencies-Y

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

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

Deficiencies-2

711 Df(2)M33a Df(2)M33a/bw^{V32g}

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

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

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

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

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

716 Df(2L)al Df(2L)al/In(2L+2R)Cy, Cy E(S)

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

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

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

720 Df(2R)42 Df(2R)42, en/SM1, al^{2} Cy sp^{2}

721 Df(2R)bw^{5} Df(2R)bw^{5}, sp^{2}/T(2;3)ap^{Xa}

722 Df(2R)bw^{VDe2L} Cy^{R} Df(2R)bw^{VDe2L}, In(2R)Cy^{R}/ Gla

* Df(2R)P 723

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

724 Df(2R)Px^{2} Df(2R)Px^{2}, bw sp/SM1, al^{2} Cy sp^{2}

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

726 Df(2R)vg^{C} Df(2R)vg^{C}/In(2LR)Rev^{B}

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

* Df(2R)vg^{D} . . . . . (=vg^{D}) . . . 429

Deficiencies-3

* Df(3L)Ly . . . (=Ly) 518, 519

728 Df(3R)M-S31 Df(3R)M-S31/T(2;3)Mé

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

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

Deficiencies-4

731 Df(4)M Df(4)M/ey^{D}

Duplications

* Dp(1;f)24 . . . (= Del(1)24) . . 58, 779

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

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

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

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

736 Dp(1;f)R Dp(1;f)R/y dor^{1}/y dor^{1}

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

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

738 Dp(1;1)112 Dp(1;1)112, y f (homozygous)

739 Dp(1;1)lz Dp(1;1)lz, lz^{50e} lz^{y4}/FM6, y^{31d} sc^{8} dm B

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

741 Dp(1;3)126 Dp(1;3)126; v f/In(3L+3R)P, Dfd ca

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

* Dp(1;3)w^{vco} 708

743 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 674, 723

744 Qn(2;2)S Qn(2;2)S, $(ast)_{5}$, al ho/In (2L+2R)Cy, Cy S^{2} E(S)

Inversions

Inversions-X

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

* In(1)AM . . . . . 12, 84, etc.

746 In(1)B^{M1} In(1)B^{M1}, v B^{M1} (tan-like); see also 757, 758, etc.

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

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

749 In(1)B^{M2} In(1)B^{M2}, f^{B27} B^{M2}/Clb

* In(1)bb 696, 697

750 In(1)ClB In(1)Cl, sc t^{2} v sl B (= ClB). . . . 76, 690, etc.

751 In(1)ClB^{36d} In(1)Cl, sc t^{2} v sl B^{36d} (= ClB^{36d}). 817

752 In(1)dl-49 In(1)dl-49, tyl

753 In(1)dl-49 In(1)dl-49, tyl bb^{1}/y v f car

754 In(1)dl-49 In(1)dl-49, v^{of} f

755 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}. 704, 768

756 In(1)dl-49 In(1)dl-49, y Su(Hw) Hw m^{2} g^{4}/y w f; (nub/+)

757 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} sc^{8R}, y sc^{S1} sc^{8} pn w ec rb cm ct^{6} sn^{3} ras^{2} g^{2} f sy od car 1/1(1)Jl^{+}.Y (= "Maxy")

758 Ins(1)dl-49, B^{M1} In(1)dl-49, In(1)B^{M1}, sc v B^{M1} (homozygous)

759 Ins(1)dl-49, B^{M1} In(1)dl-49, In(1)B^{M1}, y sc v cu-X B^{M1}

760 In(1)e(bx) In(1)e(bx), e(bx)/y f:=

* In(1)EN 686

*	In(1)FM1	In(1)FM1, In(1)dl-49, y^{31d} sc^{8} w^{a} lz^{s} B (= FM1). . . . . 16, 25, etc.
*	In(1)FM3	In(1)FM3, y^{31d} sc^{8} dm B l (= FM3). . . 7, 50, etc.
*	In(1)FM4	In(1)FM4, y^{31d} sc^{8} dm B (= FM4). . . . . 687, 696, etc.
761	In(1)FM6	In(1)FM6, y^{31d} sc^{8} dm B/y f:=; see also 30, 31, etc.
*	In(1)FMA3	In(1)FMA3, y^{2} (=FMA3) . . . . . 125, 126, etc.
762	In(1)N^{264-84}	In(1)N^{264-84}, y/FM6, y^{31d} sc^{8} dm B
763	In(1)rst^{3}	In(1)rst^{3}, rst^{3} (homozygous)
764	In(1)rst^{3}	In(1)rst^{3}, y rst^{3} car bb
*	In(1)S	. . . . . 780, 783
765	In(1)sc^{4}	In(1)sc^{4}, y sc^{4}
766	In(1)sc^{4L} sc^{8R}	In(1)sc^{4L} sc^{8R}, y; see also 682
767	In(1)sc^{7}	In(1)sc^{7}, sc^{7}
768	In(1)sc^{7}	In(1)sc^{7}, sc^{7} w^{a}
769	Ins(1)sc^{7}, AM	In(1)sc^{7}, In(1)AM, sc^{7}/In(1) dl-49, y Hw m^{2} g^{4}
770	Ins(1)sc^{7}, AM	In(1)sc^{7}, In(1)AM, sc^{7} car/ FM4, y^{31d} sc^{8} dm(without B)
771	Ins(1)sc^{7}, B^{M1}	In(1)sc^{7}, In(1)B^{M1}, sc^{7} w^{43b} B^{M1}/y f:=
772	In(1)sc^{8}	In(1)sc^{8}, sc^{8}
773	In(1)sc^{8}	In(1)sc^{8}, sc^{8} cv v f/y f:=
774	In(1)sc^{8}	In(1)sc^{8}, y^{31d} sc^{8} w^{a}
775	In(1)sc^{8}, dl-49	In(1)sc^{8}, In(1)dl-49, y^{31d} sc^{8} (homozygous)
*	In(1)sc^{8R}	. . . . . 682, 757, etc.
776	In(1)sc^{9}	In(1)sc^{9}, sc^{9} Bx f t w^{a} (homozygous)
777	In(1)sc^{260-14}	In(1)sc^{260-14}, sc^{260-14}
778	In(1)sc^{260-22}	In(1)sc^{260-22}, sc^{260-22}
779	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 173
*	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 sy od car l 757
780	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}	 783
781	In(1)w^{m4}	In(1)w^{m4} (bb?)
782	In(1)y^{3P}	In(1)y^{3P}, y^{3P} B
783	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
784	In(1)y^{4}	In(1)y^{4}, y^{4}

2L Inversions

785	In(2L)Cy	In(2L)Cy, al^{2} ast^{3} b pr (does not carry Cy mutant)
*	In(2L)Cy	In(2L)Cy, Cy dp^{lv2} b pr. 288, 346
*	In(2L)Cy$L_{t}R$	In(2L)Cy$L_{t}R$, Su(S) dp^{lv2} pr. 287
*	In(2L)NS	 312
*	In(2L)t	 323
786	In(2L)t	In(2L)t, esc c sp/SM5, al^{2} Cy lt^{v} sp^{2}
787	In(2L)t	In(2L)t, lt l L^{4} sp^{2}/ bw^{V1}, ds^{33k}
*	In(2L)t	In(2L)t, l(2)R . . . 405
788	In(2L)Tg	In(2L)Tg, Tg/SM5, al^{2} Cy lt^{v} sp^{2}

2L + 2R Inversions

789	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}. .286, 830, 853
*		In(2L+2R)Cy, Cy . . . . . 269, 379, etc.
*		In(2L+2R)Cy, Cy bw^{45a} sp^{2} or^{45a} 236
*		In(2L+2R)Cy, Cy dp^{lv2} 298
*		In(2L+2R)Cy, Cy dp^{lv1} Bl L^{4} sp^{2}. 199
*		In(2L+2R)Cy, Cy dp^{lv1} pr 808
*		In(2L+2R)Cy, Cy E(S). . . . . 390, 716, etc.
*		In(2L+2R)Cy, Cy hk^{2} . 200
*		In(2L+2R)Cy, Cy L^{4} sp^{2} 828, 835
*		In(2L+2R)Cy, Cy pr . . . . . 419, 852, etc.
*		In(2L+2R)Cy, Cy sp^{2} 300, 669
*		In(2L+2R)Cy, Cy S^{2} E(S) 744
*		In(2L+2R)Cy, Cy S^{2} dp^{lv2} E(S). 330
*	Ins(2L+2R)Cy, bw^{V34k}	In(2L+2R)Cy, Cy In(2R) bw^{V34k} 337
*	Ins(2L)Cy, (2R)NS	In(2L)Cy, Cy dp^{lv2} pr, In (2R)NS, l px l(2)NS sp 356
790	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 . 710
*	Ins(2L)t, (2R)Cy	In(2L)t, Roi In(2R)Cy, bw sp^{2} or (= Roi) . . 434

2LR Inversions

791	In(2LR)102	In(2LR)102, ds^{W} sp^{2}/SM1, al^{2} Cy sp^{2}
*	In(2LR)bw^{V1}	In(2LR)bw^{V1}, ds^{33k}. . . . . 323, 349, etc.

*	In(2LR)bw^{V32g}	. . . . . 348, 711
*	In(2LR)dp	. . . . . (see T(2;3)dp)
*	In(2LR)Gla	. . (= Gla) 226
*	In(2LR)Pm	. . . .(see In(2LR)bw^{V1})
*	In(2LR)Pm^{2}	. . . (see In(2LR)bw^{V32g})
*	In(2LR)Rev	. . (=Rev) 793
*	In(2LR)Rev^{B}	726
*	In(2LR)Rvd	. . . .(see In(2LR)Rev^{B})
*	In(2LR)SM1	In(2LR)SM1, al^{2} Cy sp^{2}. . . (= SM1). 207, 210, etc.
*	In(2LR)SM5	In(2LR)SM5, al^{2} Cy lt^{v} sp^{2} . (= SM5). 201, 205, etc.
*	In(2LR)U	. . . . . (= U) . . . 254

2R Inversions

*	In(2R)vw^{V34k}	 337
792	In(2R)bw^{VDe1}	In(2R)bw^{VDe1}, b/lt l cn mi sp
793	In(2R)bw^{VDe2}	In(2R)bw^{VDe2}/In(2LR)Rev l
*	In(2R)Cy	In(2R)Cy, cn^{2} Bld . . 350
*	In(2R)Mo^{K}	 834
*	In(2R)NS	 356

3L Inversions

*	In(3L)D	. . (= D) . 468, 513, etc.
*	In(3L)D^{3}	. . (= D^{3}). . . . 469, 518
*	In(3L)P	In(3L)P, gm . . . 549, 611
*	In(3L)P	In(3L)P, Mé . 522, 523, etc.
*	In(3L)P	In(3L)P, Mé ca. . 857, 859
794	In(3L)P	In(3L)P, mot-36e/R

3L + 3R Inversions

*	In(3L+3R)LVM	. (= LVM) . 497, 514, 519
*	In(3L+3R)P	In(3L+3R)P, l(3)PL l(3)PR . . (= Payne) . . . . . .469, 482, etc.
*		In(3L+3R)P, l(3)PL l(3)PR, Dfd ca . . 478, 479, etc.
*		In(3L+3R)P, M(3)x e^{s}. 561
*	Ins(3L)P, (3R)C	In(3L)P, Mé, In(3R)C, e l(3)e 614
*		In(3L)P, Mé, In(3R)C, Sb e l(3)e 553
*	Ins(3L)P, (3R)P18	In(3L)P, In(3R)P18, Mé Ubx e^{4}. 515

3LR Inversions

*	In(3LR)Cx	. .(= Cx) . 471, 489, etc.
*	In(3LR)Cx	In(3LR)Cx, D. . . 513, 858
*	In(3LR)DcxF	. .(=In(3LR)CxF). 603, 669
*	In(3LR)DcxF	In(3LR)DcxF, ru h ca. .810
795	In(3LR)sep	In(3LR)sep, sep ri p^{p}
*	In(3LR)P35	. . (= In(3LR)Pasadena-35). 851
*	In(3LR)TM1	In(3LR)TM1, Mé ri sbd^{1} . . (= TM1). . 449, 516, etc.
*	In(3LR)TM3	In(3LR)TM3, y^{+} ac^{+} ri p^{p} sep bx^{34e} e^{s} (=TM3). 546, 850
*	In(3LR)Ubx^{101}	 569
*	In(3LR)Ubx^{130}	In(3LR)Ubx^{130}, Ubx^{130} e^{s}. . .(= Ubx^{130}) . . . . . 476, 659, etc.

3R Inversions

796	In(3R)Antp^{B}	In(3R)Antp^{B}, Antp^{B}/TM1, Mé ri sbd^{1}
*	In(3R)C	In(3R)C, cd . . . 570
*		In(3R)C, e . . . . . 459, 474, etc.
*		In(3R)C, e l(3)e . . . . . 447, 527, etc.
*		In(3R)C, l(3)a . . . . . 446, 475, etc.
*		In(3R)C, Sb e l(3)e 480, 505
*	In(3R)ca^{v}	 488
*	In(3R)Cyd	In(3R)Cyd, Cyd . . . . (= Cyd) 481
797	In(3R)Dl^{B}	In(3R)Dl^{B}, st Dl^{B}/In(3R) P^{W}, st l(3)W ca
798	In(3R)Hu	In(3R)Hu, Hu Sb^{Spi}/In(3L+3R)P
799	In(3R)Mo	In(3R)Mo, sr/T(2;3)ap^{Xa}, ca; see also 670
*	In(3R)P	. . . . . 473, 502
*	In(3R)P18	. . . (= In(3R)Pasadena 18) 515
800	In(3R)P^{Fla}	In(3R)P^{Fla} (homozygous)
*	In(3R)P^{W}	In(3R)P^{W}, st l(3)W ca 593, 797

Translocations-1; Y

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

Translocations-1;2

803	T(1;2)Bld	T(1;2)Bld, Bld/ClB (carries In(2R)Cy)
804	T(1;2)f$^{257-15}$	T(1;2)f$^{257-15}$/In(1)AM
805	T(1;2)lt	T(1;2)lt/In(2L+2R)Cy, Cy (carries eq and possibly su(s)^{3})
806	T(1;2)N$^{264-10}$	T(1;2)N$^{264-10}$/FM6, y^{31d} sc^{8} dm B
807	T(1;2)sc^{S2}	T(1;2)sc^{S2}/In(2L+2R)Cy, Cy
808	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^{lv1} pr

Translocations-1;3

809	T(1;3)263-4	T(1;3)263-4, y sc B^{l}/ In(1)AM
810	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)
811 T(1;3)N^{264-6} T(1;3)N^{264-6}, y/y w dm (= N^{6})
812 T(1;3)04 T(1;3)04/ClB
813 T(1;3)05 T(1;3)05, D/y f:=
814 T(1;3)ras^{v} T(1;3)ras^{v}/y f:=
* T(1;3)sc^{J4} 822
815 T(1;3)sc^{260-15} T(1;3)sc^{260-15}/FM6, y^{31d} sc^{8} dm B
816 T(1;3)v T(1;3)v, v/FM6, y^{31d} sc^{8} dm B
817 T(1;3)w^{vco} T(1;3)w^{vco}, v f/ClB^{36d}

Translocations-1;4

818 T(1;4)B^{S} T(1;4)B^{S}/y f:=
* T(1;4)N^{8a} . . . . . (see T(1;4)N^{264-12})
819 T(1;4)N^{264-12} T(1;4)N^{264-12}/FM6, y^{31d} sc^{8} dm B
820 T(1;4)sc^{8} T(1;4)sc^{8},B w^{a}/y f:=
821 T(1;4)w^{m5} T(1;4)w^{m5}/ey^{D}
822 T(1;4)w^{m5} (1;3)sc^{J4} T(1;4)w^{m5}; T(1;3)sc^{J4} (ClB)
823 T(1;4)$w^{m258-18}$ T(1;4)$w^{m258-18}$, y/ci^{D}
824 T(1;4)$w^{m258-21}$ T(1;4)$w^{m258-21}$, y/ci^{D}
825 T(1;4)$w^{m258-21}$ T(1;4)$w^{m258-21}$, y w^{a}/FM4, y^{31d} sc^{8} dm B
* T(1;4)w^{VD3} . . . . (see T(1;4)$w^{m258-21}$)

Translocations-Y;2

* T(Y;2)A 331
826 T(Y;2)B T(Y;2)B/b; see also 416
* T(Y;2)C 267, 368
* T(Y;2)E 198, 262
* T(Y;2)G 233
* T(Y;2)J 376
827 T(Y;2)rl T(Y;2)rl, lt cn/b lt bw

Translocations-Y;2;3

* T(Y;2;3)F 554

Translocations-2;3

828 T(2;3)101 T(2;3)101, al^{2} sp^{2}/In(2L+2R) Cy, Cy L^{4} sp^{2}
829 T(2;3)101 T(2;3)101; ru h e^{4} ro ca/In (3L+3R)P, Dfd ca
830 T(2;3)108 T(2;3)108, al c sp^{2}/In(2L+2R) Cy, al^{2} Cy lt^{3} L^{4} s^{2}
831 T(2;3)109 T(2;3)109, p^{p}/In(3L+3R)P, Dfd ca
832 T(2;3)A T(2;3)A, Bl; ru h D TA ss e^{s} /In(3L+3R)P
* T(2;3)ap^{Xa} . . . (= Xa). 451, 534, etc.
* T(2;3)ap^{Xa} T(2;3)ap^{Xa}, ca . . 547, 799
833 T(2;3)ap^{Xa} T(2;3)ap^{Xa}/1(3)XaR
834 T(2;3)Ata T(2;3)Ata, Ata/In(2R)Mo^{K}
835 T(2;3)B T(2;3)B, al sp^{2}/In(2L+2R)Cy, Cy L^{4} sp^{2}
836 T(2;3)B T(2;3)B; ru h D TB ss e^{s}/In (3L+3R)P
837 T(2;3)bw^{V4} T(2;3)bw^{V4}/SM1, al^{2} Cy sp^{2}
838 T(2;3)bw^{V5} T(2;3)bw^{V5}_{2}/SM5, al^{2} Cy lt^{v} sp^{2}
839 T(2;3) bw^{VDe3} T(2;3)bw^{VDe3}; Ubx bxd/ In(3LR)Cx
840 T(2;3) bw^{VDe4} T(2;3)bw^{VDe4}/SM5, al^{2} Cy lt^{v} sp^{2}
841 T(2;3)C T(2;3)C; ru h D TC ss e^{s}/In(3L+3R)P
* T(2;3)dp 237
842 T(2;3)dp^{D} T(2;3)dp^{D}, dp^{D}/SM1, al^{2} Cy sp^{2}
843 T(2;3)E T(2;3)E/SM5, al^{2} Cy lt^{v} sp^{2}
844 T(2;3)Hn T(2;3)Hn, Df(3L)Hn, Hn/ In(3LR)Ubx^{130}, Ubx^{130} e^{s}
* T(2;3)Mé . . . . 524, 525, etc.
* T(2;3)P T(2;3)P, P . . 674, 723
* T(2;3)p^{Gr} . . . (see T(2;3)Pu^{Gr})
845 T(2;3)Pu^{4} T(2;3)Pu^{4}, Pu^{4}/C(3)x
846 T(2;3)Pu^{Gr} T(2;3)Pu^{Gr}, Pu^{Gr}/SM1, al^{2} Cy sp^{2}
847 T(2;3)rn T(2;3)rn/SM1, al^{2} Cy sp^{2}
848 T(2;3)Dp-S T(2;3)Dp-S, ho/In(2L+2R) Cy, Cy E(S) (hom. viable)
849 T(2;3)S^{L} T(2;3)S^{L}/In(2L+2R)Cy, Cy E(S)
* T(2;3)S^{M} 433
850 T(2;3)Sb^{V} T(2;3)Sb^{V}, Sb^{V}, In(3R) Mo/TM3, y^{+} ac^{+} ri pP sep bx^{34e} e^{s}
851 T(2;3)Sb^{V} T(2;3)Sb^{V}, Sb^{V}, In(3R)Mo, In(3LR)P35/SM1, al^{2} Cy sp^{2}; In(3LR)Ubx^{130}, Ubx^{130} e^{s}
* T(2;3)Xa . . . (see T(2;3)ap^{Xa})

Translocations-2; 4

852 T(2;4)a T(2;4)a/In(2L+2R)Cy, Cy pr; ey^{2}
853 T(2;4)ast^{v} T(2;4)ast^{v}/In(2L+2R)Cy, al^{2} Cy lt^{3} L^{4} sp^{2}
854 T(2;4)b T(2;4)b/In(2L+2R)Cy, Cy pr; ey^{2}
855 T(2;4)d T(2;4)d, al dp px sp/In (2L+2R)Cy, Cy pr; ey^{2}
856 T(2;4)d T(2;4)d/In(2L+2R)Cy, Cy pr

Translocations-3;4

857 T(3;4)A2 T(3;4)A2/In(3L)P, Mé ca
858 T(3;4)A12 T(3;4)A12/In(3LR)Cx, D
859 T(3;4)A13 T(3;4)A13, ve ca/In(3L) P, Mé ca
860 T(3;4)A28 T(3;4)A28, ve ca (homozygous)
861 T(3;4)c T(3;4)c/In(3L+3R)P, Dfd ca
862 T(3;4)e T(3;4)e/In(3LR)Ubx^{130}, Ubx^{130} e^{s}

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

Transpositions

866	Tp(3)bxd^{100}	Tp(3)bxd^{100}, ri/T(2;3)Mé
867	Tp(3)bxd^{107}	Tp(3)bxd^{107}, bx bxd^{107} sr e^s/bx^{34e} Mc
868	Tp(3)sbd^{104}	Tp(3)sbd^{104}/In(3LR)Ubx^{130}, Ubx^{130} e^s
869	Tp(3)Vno	Tp(3)Vno/H^2

BUFFALO, NEW YORK: STATE UNIVERSITY COLLEGE
Department of Biology

Wild Stocks

1 wild
2 Canton-S (India)
3 Canton-S (Israel)

Chromosome 1

4 B
5 M^5 (Ins(1)$sc^{51}sc^8$B w^a(1))
6 rb
7 v
8 w
9 w (Greece)
10 w (India)
11 w (Israel)
12 w^a
13 y
14 y (Greece)
15 y (India)
16 y (Israel)
17 y
18 y w m

Chromosome 2

19 b
20 b/Cy
21 b/vg
22 bw
23 dp
24 ho
25 S/Cy
26 vg

Chromosome 3

27 In(3L)
28 se
29 se/e^3
30 ss^a

Chromosome 4

31 ey^2
32 pol

Multichromosomal

33 Cy/Pm^{ds33K}; H/Sb-C(3)
34 v/bw
35 vg/se

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

Wild Stock

Oregon-R

Chromosome 1

B
m^D B
sc^{S1} B In w^a sc^8
w
w^a
w^e
w B
w m
w m f

Chromosome 2

b vg
L

Chromosome 3

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

Multichromosomal

sc^{S1} B In w^a sc^8; SM1, al^2 Cy sp^2/ dp b Pm ds^{33K}; C Sb/Ubx^{130} e^s; spa^{Pol}(1;2;3;4)

OAK RIDGE, TENNESSEE: OAK RIDGE NATIONAL LABORATORY

Wild Stocks

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

X Chromosome Stocks

b-1 B/C(1)DX, y f
b-2 car bb
b-3 cv v f/C(1)DX, y f
b-4 dor/ClB
b-5 f
b-6 f BB/C(1)DX, y f
b-7 fa^{g}
b-8 fu^{59}/C(1)DX, y f
b-9 l(1)J1 sc^{J1}/Dp(1;f)24
b-10 m f car/C(1)DX, y w f
b-11 mal
b-12 pn
b-13 ptg^{3} v m g^{2} sd f/C(1)DX, y f
b-14 sc cv v f B/C(1)DX, y f
b-15 sc ec cv ct^{6} v g/In(1)dl-49, y Hw m^{2} g^{4}
b-16 sc ec cv ptg^{3} v/C(1)RM, y v f car
b-17 sc z ec ct
b-18 spl
b-19 v
b-20 v f su(f)
b-21 v m g^{2} sd f
b-22 w
b-23 w^{a}
b-24 w^{a} fa fa^{no} rb/C(1)DX, y f
b-25 w^{a} fa spl
b-26 w^{a} fa^{g} fa^{no} rb/C(1)DX, y w f
b-27 w^{a} fa^{g} spl sn^{3}
b-28 w^{a} fa^{no} spl rb/C)1)DX, y f
b-29 w^{a} pdf
b-30 w^{a} spl rb
b-31 w^{e} bb^{1}/C(1)DX, y f/$B^{S}Y$
b-32 w^{sp}
b-33 y
b-34 y ac sc pn
b-35 y ac sc pn/C(1)DX, y f
b-36 y ac sc pn w rb cm ct^{6} sn^{3} ras^{2} v dy g^{2} f car/C(1)RM, In(1) sc^{S1} + dl-49, sc^{S1} v f car
b-37 y B/C(1)DX, y f
b-38 y bb^{1-3a}/C(1)RM, y w/y^{+} Y
b-39 y cv f
b-40 y cv v f
b-41 y cv v f car
b-42 y ec cv ct^{6}/FM6
b-43 y ec cv ct^{6} v/FM6
b-44 y fa^{n} sn^{3}
b-45 y Hw/C(1)RM, In(1)sc^{S1L} sc^{8R} + S, sc^{S1} sc^{8} w^{a} B
b-46 y l(1)451/FM6, y^{31d} sc^{8} dm B
b-47 y w
b-48 y w bb^{ds}
b-49 y w fa^{no}
b-50 y w spl sn^{3}
b-51 y w^{a}
b-52 y w^{a} fa^{no} spl/C(1)RM, y w fa^{no}
b-53 y w^{a} m f car
b-54 y w^{a} spl rb
b-55 y^{2}/C(1)DX, y f
b-56 y^{2} cv v f
b-57 y^{2} cv v wy car/FM6
b-58 y^{2} sc f
b-59 y^{2} sc f su(f)
b-60 y^{2} sc f su(f)/C(1)DX, y f
b-61 y^{2} sc w^{a} ec
b-62 y^{2} v f car/C(1)DX, y f
b-63 y^{2} v mal^{bz}
b-64 y^{2} w^{cf}
b-65 y^{2} wy car/C(1)DX, y f

Chromosome 2

c-1 a px or
c-2 Adh^{n1}
c-3 al b c sp
c-4 al dp b pr c px sp
c-5 al dp b pr Bl c px sp/CyO
c-6 al dp spd^{fg}
c-7 b Adh^{n1} Tft/CyO
c-8 b cn c bw
c-9 b j
c-10 b pr c Elp px
c-11 b pr tu-bw^{e} px
c-12 b pu
c-13 Bl L^{2}/SM5, al^{2} Cy lt^{v} sp^{2}
c-14 $bw^{}$
c-15 bw^{D}
c-16 cl spd^{fg}
c-17 cn bw
c-18 cn en/SM5
c-19 cui
c-20 d
c-21 da/In(2L)Cy + In(2R)Cy
c-22 fj wt/SM5
c-23 Fo/SM1, al^{2} Cy sp^{2}
c-24 lt stw^{3}
c-25 lys
c-26 lys $rc^{}$
c-27 lys rc^{2}
c-28 lys^{2} rc^{2}/CyO
c-29 M(2)S7/SM5
c-30 M(2)$S2^{10}$/In(2L)Cy + In(2R)Cy + Dp(2;2)41^{2}, Cy pr
c-31 net al ex ds S ast shv ho rub/SM1

c-32 nub^2
c-33 nub^2 b Sco pr cn/CyO
c-34 nub^2 Bl/SM5
c-35 nub^2 Bl L/SM5
c-36 nw^D Pu^2 Pin^{Yt} /SM1
c-37 Pin
c-38 Pin^2/bw^{V57e} SM1
c-39 Pin^{Yt} /SM1
c-40 Pu^2/SM1
c-41 px sp Pin^2/SM1
c-42 rc pr
c-43 S Sp Tft nw^D Pin^{Yt}/CyO
c-44 Sco/In(2L)Cy + In(2R)Cy, Cy Roi
c-45 sp^2 bs^2
c-46 Sp J (L^2) Pin/SM5
c-47 Sp lys d/SM1
c-48 Sp lys d J/In(2L)Cy + In(2R)Cy
c-49 Sp lys rc J/SM1
c-50 Sp rc^2/In(2L)Cy + In(2R)Cy
c-51 spd^{fg}
c-52 spd^{fg} Sp d/SM1
c-53 stw
c-54 stw^3 c
c-55 Tft/SM1
c-56 Tft L/SM5
c-57 vg
c-58 vg^U/In(2L)t + In(2R)Cy, Roi bw^{45a} sp^2 or^{45a}

Chromosome 3

d-1 Bd^G/In(3R)C, l(3)e
d-2 bx^{34e}
d-3 ca
d-4 ca K-pn
d-5 ca^{nd}/TM3, Sb Ser
d-6 cu kar
d-7 cv-c sbd^2
d-8 D^3 H/In(3L)P, Me
d-9 e
d-10 e^s
d-11 Gl Sb/LVM
d-12 gro
d-13 h st ry^3 ss^a/In(3LR)Ubx^{130}, Ubx^{130} e^s
d-14 H^2/Tp(3)Vno, Vno
d-15 jvl
d-16 Me^{65d} h th/TM3, Sb Ser
d-17 Me^{65d} jv se by/TM3, Sb Ser
d-18 p^p bx sr e^s
d-19 p^p Ki
d-20 Pr/In(3R)C, e
d-21 Pr^K Dr/In(3L)P + In(3R)P
d-22 Pr^L/Tp(3)Vno, Vno
d-23 Pr^L Bd^G Dr/TM1. Me
d-24 Pr^L gro/TM1, Me
d-25 R Ly/In(3L)P, gm
d-26 red
d-27 red Ubx ca K-pn/TM1, Me
d-28 ru
d-29 ru h st p^p ss e^s
d-30 ru h th st cu sr e^s ca
d-31 ru h th st cu sr e^s Pr ca/TM1, Me ri
d-32 ru^g jv se by
d-33 ry^2
d-34 se
d-35 se ss k e^s ro
d-36 sr gl
d-37 ss^a
d-38 st
d-39 st c(3)G ca/In(3LR)Ubx^{130}, Ubx^{130} e^s
d-40 st in ri p^p
d-41 st ry^2 sr e^s
d-42 st sbd e^s ro ca
d-43 $su(Hw)^2$ sbd/In(3LR)Ubx^{130}, Ubx^{130} e^s
d-44 TM6, Ubx^{17005}/T(2;3)ap^{Xa}
d-45 tra^D Sb e/In(3LR)Ubx^{130}, Ubx^{130} e^s and T(1;3)OR60, y
d-46 ve h th
d-47 ve h th c(3)G Sb Ubx/st c(3)G ca
d-48 ve h th cu bx e^s ro ca

Chromosome 4

e-1 Ce^2/spa^{Cat}
e-2 ci ey^R
e-3 ci gvl ey^R sv^n
e-4 ci gvl spa^{Cat}/ci^D
e-5 ci^D/ey^D
e-6 sv^n
e-7 spa^{pol}

Multichromosomal Stocks

f-1 v/Y^{bb}
f-2 In(1)w^{m4}; E(var)7/In(2L)Cy + In(2R)Cy
f-3 v; In(2R)bw^{VDe1} /SM1
f-4 y/C(1)DX, y f; dp lys rc pr
f-5 y f; nub^2
f-6 y^2; T(2;3)101, al sp^2/Cy L^4 sp^2
f-7 y; In(2L)Cy + In(2R)Cy/In(2LR)bw^{V1}, bw^{V1}; Sb/In(3L)D, D
f-8 FM6, y^{31d} sc^8 dm B/In(1)AM, y^2; pr Bl/SM1; Tp(3)Vno, Vno/In(3LR)Ubx^{130}, Ubx^{130} e^s; pol
f-9 +/C(1)DX, y f; bw; st; pol
f-10 y; bw; st; pol
f-11 y^2; TM3, Sb Ser p^p/Ki p^p ry sr e^s
f-12 y^2; ve h th ro ca/TM1, Me ri
f-13 v; In(2R)bw^{VDe1}/SM1; M(4)/ey^D
f-14 In(1)sc^{8L} sc^{S1R} + dl-49, y^{31d} sc^- v f (B)/y w^a N^{Nic}/y^+Y w^+; ca K-pn
f-15 y^2; ci gvl ey^R sv^n
f-16 y^2; ci^D/ey^D
f-17 y^2; pol
f-18 y^2 w^{cf}; ci gvl spa^{Cat}/ci^D pol
f-19 b pr Bl/SM1; Tp(3)Vno, Vno/In(3LR)Ubx^{130}, Ubx^{130} e^s
f-20 bw; st
f-21 SM1/In(2LR)bw^{V1}, bw^{V1}; Sb/In(3LR)Ubx^{130}, Ubx^{130} e^s
f-22 bw; sim-4/ci^D

Inverted Chromosomes

g-1 In(1)65, y f/$B^{S}Y$
g-2 In(1)481, y bb^{l-481}/FM6/$y^{+}Y$
g-3 In(1)AB, y f
g-4 In(1)AM/T(1;3)65, y
g-5 In(1)B^{M1}, cm B^{M1} /C(1)DX, y f
g-6 In(1)dl-49, y v f car bb^{amg}/C(1)DX, y_{L8}f
g-7 In(1)dl-49, y w lz^{s} bb/In(1)sc^{L8}, sc^{L8} w^{a} m car
g-8 In(1)dl-49, y^{2} sc w^{a}/$B^{S}Y$
g-9 In(1)dl-49 + B^{M1}, sc v B^{M1}
g-10 In(1)dl-49 + B^{M1}, y^{2} sc w^{a} v B^{M1}
g-11 In(1)EN, y bb/$y^{+}Y$
g-12 In(1)l-59, y l(1)v59/C(1)RM, y w/$y^{+}Y$
g-13 In(1)l-v132, y l(1)v132/C(1)RM, y w/$y^{+}Y$
g-14 In(1)l-v231, y l(1)v231/C(1)RM, y w/$y^{+}Y$
g-15 In(1)rst^{3}, rst^{3}
g-16 In(1)rst^{3}, y rst^{3} car (bb?)/C(1)RM, In (1)sc^{S1L} sc^{8R} + S, y^{31d} sc^{S1} sc^{8} w^{a} B
g-17 In(1)rst^{3}, rst^{3} ras v f/C(1)DX, y f; bw
g-18 In(1)sc^{4}, y sc^{4}
g-19 In(1)sc^{4}, y sc^{4} cv v B/C(1)DX, y f/$B^{S}Y$
g-20 In(1)sc^{4L} sc^{8R}, y sc^{4} sc^{8} cv v B/C(1)DX, y f/$B^{S}Y$
g-21 In(1)sc^{4L} sc^{8R} + S, y sc^{4} sc^{8} w^{a} B/C(1) RM, y^{2} su(w^{a}) w^{a} bb/$y^{+}Y$
g-22 In(1)sc^{7}, sc^{7} w^{a} ec/C(1)DX, y f
g-23 In(1)sc^{7}, sc^{7} w fa^{no}/C(1)DX, y f
g-24 In(1)sc^{8}, sc^{8} v f car
g-25 In(1)sc^{8}, y^{31d} sc^{8} w^{a}
g-26 In(1)sc^{8} + S, y^{-} ac^{-} sc^{8} w^{a}/$y^{+}Y$
g-27 In(1)sc^{9}, sc^{9} w^{a} t Bx
g-28 In(1)sc^{S1} + dl-49, y^{62k} sc^{S1} v f car/C (1)DX, y f
g-29 In(1)sc^{S1L} sc^{4R}, sc^{S1} sc^{4} cv v B/C(1)RM, y f/$B^{S}Y$
g-30 In(1)sc^{S1L} sc^{8R} + dl-49, sc^{S1} sc^{8} v B car/C(1)DX, y f
g-31 In(1)w^{m4L} $N^{264-84R}$, y w^{m4} N^{264-84} sn/FM3/ $B^{S}Y$ y^{+}
g-32 In(1)y^{3P}, y^{3P} B
g-33 In(1)y^{4}, y^{4}
g-34 In(1LR)sc^{V1}, v•sc^{V1} y^{+}/In(1)sc^{8} + dl-49, y^{31d} sc^{8} v f B
g-35 In(1LR)l-v139, w^{m139} l(1)v139/C(1)DX, y w f/Y/Y
g-36 In(2LR)lt^{m3}, lt^{m3}/SM5
g-37 In(2LR)lt^{m12}/SM5
g-38 Basc
g-39 Biny/l(1)$J1^{259}$ w m f/$y^{+}Y$
g-40 FM4/C(1)DX, y f
g-41 FM6/C(1)DX, y f/$y^{+}Y$/Y

Translocated Chromosomes

h-1 T(1;2)Bld/T(1;2)64, y and Fs(2)Do
h-2 T(1;4)A7, y w/y^{2} su(w^{a}) w^{a} bb
h-3 T(1;4)A13(18C5)
h-4 T(1;4)A17(8A2)/C(1)DX, y f
h-5 T(1;4)A19
h-6 T(1;4)A20/C(1)DX, y f
h-7 T(1;4)B^{S}(16A1), B^{S}/C(1)DX, y f
h-8 T(1;4)B^{S}(16A1), y^{2} cv v B^{S} car/C(1)DX, y f
h-9 T(1;4)e15
h-10 T(1;4)h4
h-11 T(1;4)h6
h-12 T(1;4)11(15A), y l^{11} car/$y^{+}Y$
h-13 T(1;4)w^{m5}(3C3), w^{m5}
h-14 T(1;4)4C3/C(1)DX, y f
h-15 T(1;4)(13B8-9)/C(1)DX, y f
h-16 T(2;3)A, Bl/In(2L)Cy + In(2R)bw^{VDe1}
h-17 T(2;3)lt^{m7}(98C), lt^{m7}/SM5
h-18 T(3;4)89E, ss bx bxd/ey^{D}
h-19 T(Y;2)E/b el rd^{s} pr cn
h-20 T(1;4)w^{m5}, 9A, w^{m5}/FM4, y^{31d} w dm B
h-21 T(1;4)w^{m5}, B^{S}, w^{m5} v B^{S}/FM4, y^{31d} w dm f

Closed X Chromosomes (examined cytologically October, 1967)

i-1 R(1)1, y/C(1)DX, y f/$y^{+}Y$
i-2 R(1)2, w^{spont} v f/C(1)RM, In(1)sc^{S1L} sc^{8R} + S, sc^{S1} sc^{8} w^{a}/Y
i-3 R(1)2, y B/C(1)DX, y f

X-Y Combinations

j-1 X•Y^{L}, y cv v f car•Y^{L}/C(1)RM, y/Y"
j-2 X•Y^{S} (A3), y w•Y^{S}/C(1)RM, y v f/R(Y)L
j-3 X•Y^{S} (P-8b), In(1)sc^{8L}, EN^{R}, y^{+} f y•Y^{S}/ C(1)RM, y v f/R(Y)L
j-4 Y^{S}X•(FR1), Y^{S} y cv v f•/C(1)DX, y f/Y
j-5 Y^{S}X•(P-7), In(1)EN, Y^{S} y f•/C(1)RM, y v f/Y

Compound X Chromosomes

k-1 C(1)RA (ND-27), sc v f--In(1)sc^{8}, f y sc^{8}•/X•Y^{L}(C-2), y cv v f car bb^{-}•Y^{L}/Y"
k-2 C(1)RA (Muller), In(1) dl-49, y w f--In (1)sc^{8}? f sc^{8}•/X•Y^{S}, y w•Y^{S}/Y^{L}•bb^{+} ac^{+} y^{+}
k-3 C(1)RA•Y^{L}, +--In(1)sc^{8L}, EN^{R}, y•Y^{L} y^{+} /Y^{S}X•Y^{L}, In(1)EN, Y^{S}B y•Y^{L}/y^{+} ac^{+}•Y^{L}
k-4 C(1)RM, pn/Y^{S}X•Y^{L}, y B/Y
k-5 C(1)RM, v f Bx mal/Y^{S}X•Y^{L}, In(1)EN, v f B

Compound Autosomes

l-1 C(2L)RM; C(2R)RM
l-2 C(2L)RM#3; C(2R)RM#3
l-3 C(2L)RM#4, dp; C(2R)RM#4, px
l-4 C(2L)RM, b; C(2R)RM, cn
l-5 C(2L)RM, nub^{2} b^{66h} pr; C(2R)RM, cn
l-6 C(3L)RM; C(3R)RM (Rasmussen no. 26)
l-7 C(3L)RM#1, h^{2} rs^{2}; C(3R)RM#1, +
l-8 C(3L)RM#4, ri; C(3R)RM #4, sr
l-9 C(3L)RM, se h^{2} rs^{2}; C(3R)RM, sbd gl e^{s} (Rasmussen no. 44)

l-10 +/+/B^SY/(Y?); C(2L)RM#3; C(2R)RM#3
l-11 w/w/B^SY; C(2L)RM#3; C(2R)RM#3
l-12 In(1)AM, y^2/FM6; C(2L)RM, dp; C(2R)RM, px
l-13 In(1)AM, y^2/FM6/Y; C(2L)RM#3; C(2R)RM#3
l-14 In(1)AM, y^2/FM6/Y; C(2L)RM (l-1); C(2R)RM (l-1)
l-15 In(1)AM, y^2/FM6/B^SY/(Y?); C(2L)RM #3; C(2R)RM #3
l-16 C(4)RM, ci ey^R/gvl sv^n

Attached XY Chromosomes

m-1 $XY^L{\cdot}Y^S$ (10809 Parker), y^2 su(w^a)w^a $Y^L{\cdot}Y^S$/C(1)RM, y v bb/0
m-2 $XY^S{\cdot}Y^L$ (110-8 Parker), y^2 su(w^a) w^a $Y^S{\cdot}Y^L$ y^+/C(1)RM, y v bb/0
m-3 $Y^SX{\cdot}Y^L$ ($FR\text{-}1^L$, $U\text{-}8d^R$), Y^S y w^a cv v f$\cdot Y^L$/C(1)RM, y^2 su(w^a) w^a bb/0
m-4 $Y^SX{\cdot}Y^L$, In(1)EN, Y^S B f v y$\cdot Y^L y^+$/C(1)RM, y v bb/0
m-5 $Y^SX{\cdot}Y^L$, In(1)EN + dl-49, Y^S car f v y$\cdot Y^L$/C(1)RM, y^2 su(w^a) w^a bb/0
m-6 $Y^SX{\cdot}Y^L$, In(1)EN + dl-49, Y^S f v pn y$\cdot Y^L$

Y Derivatives

n-1 B^Sw^+Y/y w^a
n-2 B^SYy^+/y v; bw
n-3 B^SYy^+/C(1)RM, y/y
n-4 B^SYy^+/In(1)w^{m4L} $N^{264\text{-}84R}$, y sn/FM3
n-5 B^SYy^{31d}/C(1)RM, y w f/Y^S y cv v f
n-6 bw^+Y/y v; bw
n-7 bw^+Yy^+/C(1)DX, y f/y v f
n-8 y^+Y/C(1)RM, y v/y
n-9 y^+Yw^+(11a)/y w^a
n-10 $y^+Yw^+B^S$(Y11)/y w^a
n-11 $y^+Y^L{\cdot}$(FR-2)/C(1)RA, y--In(1)sc^8/$Y^SX{\cdot}Y^L$, In(1)EN, y B
n-12 Ybb$^-$/In(1)w^{m4}
n-13 $Ymal^+$ no. 2/y^2 v mal
n-14 Yw^+ (Y900)/y w^a
n-15 $Y^L{\cdot}ac^+$ y^+(sc^8EN c.o. Y B-a)X$\cdot Y^S$, y w/C(1)RM, y v f
n-16 $Y^L{\cdot}bb^+$ ac^+ y^+ (sc^8EN c.o. Y T-0)/X$\cdot Y^S$, y w$\cdot Y^S$/C(1)DX, y f

Extra Y Chromosome Stocks

o-1 In(1)w^{m4L} $N^{264\text{-}84R}$, y sn/FM3/Y females x dm sn males
o-2 In(1)w^{m4L} $N^{264\text{-}84R}$, y sn/FM3 females x FM3/y^+Y/B^SY males

Deficiencies and Duplications

p-1 Df(3R)ry/In(3LR)Ubx^{130}, Ubx^{130} e^s
p-2 Dp(1;f)3/C(1)RM, y/$XY^L{\cdot}Y^S$, l(1)$J1^{259}$ y w $Y^L{\cdot}Y^S$
p-3 Dp(1;f)18/C(1)RM, y v f/$XY^L{\cdot}Y^S$, l(1)$J1^{259}$ y w $Y^L{\cdot}Y^S$
p-4 Dp(1;f)24
p-5 Dp(1;f)52/C(1)A/$XY^L{\cdot}Y^S$, l(1)$J1^{259}$ y w $Y^L{\cdot}Y^S$
p-6 Dp(1;f)112/C(1)RM, y v f/$XY^L{\cdot}Y^S$, l(1)$J1^{259}$ y w $Y^L{\cdot}Y^S$
p-7 Dp(1;f)122/C(1)RM, y v f/$XY^L{\cdot}Y^S$, l(1)$J1^{259}$ y w $Y^L{\cdot}Y^S$
p-8 Dp(1;f)164/C(1)RM, y v f/$XY^L{\cdot}Y^S$, l(1)$J1^{259}$ y w $Y^L{\cdot}Y^S$
p-9 Dp(1;f)1492/sc^{53k}
p-10 Dp(1;1)B^S(RAG), B^S--In(1)$sc^8{\cdot}$/In(1)sc^7 + AM, sc^7
p-11 Dp(1;1)B^S(TMG), In(1)$sc^4{\cdot}B^S$, y sc^4 m f$\cdot B^S$/In(1)sc^7 + AM, sc^7
p-12 Dp(1;1)B^S(TMG), In(1)sc^{8L}, R(1)$2^R{\cdot}B^S$, f$\cdot B^S$/X^D/$B^SY^L{\cdot}Y^S$
p-13 Dp(1;2)sc^{19i}
p-14 Dp(1;3)w^{49a7} (Spotter)
p-15 Dp(1;3)51/C(1)RM, y v f/$XY^L{\cdot}Y^S$, l(1)$J1^{259}$ y w $Y^L{\cdot}Y^S$
p-16 Dp(1;3)142/C(1)RM, y/$XY^L{\cdot}Y^S$, l(1)$J1^{259}$ y w $Y^L{\cdot}Y^S$
p-17 Dp(1;3)sc^{J4}/C(1)RM, y v f/O?/$XY^L{\cdot}Y^S$, l(1)$J1^{259}$ y w $Y^L{\cdot}Y^S$
p-18 Dp(1;4)w^{51c20}/C(1)DX, y w f/In(1)w^{m4L}, rst^{3R}, rst^3 car
p-19 Dp(1;4)174/C(1)RM, y v f/$XY^L{\cdot}Y^S$, l(1)$J1^{259}$ y w $Y^L{\cdot}Y^S$
p-20 Dp(2;f)1/sp Pin^2/Px^4
p-21 Dp(3;4)ry^+/cu kar/cu kar

Triploid Stocks

q-1 C(1)RM, y w fa^{no}/FM6 females x FM6/B^SYy^+ males
q-2 C(1)RM, y^2 sc w^a ec/FM6 females x FM6/B^SYy^+ males
q-3 C(1)RM, In(1)dl-49, v^{0f} f/FM6 females x FM6 males

ARLINGTON, TEXAS: UNIVERSITY OF TEXAS

Virus-caused CO_2 sensitivity stocks

several strains

Delayed-recovery from CO_2 anesthesia strains

TDR-orange,
TDR-la
TDR-3
TDR-BC_3

AMHERST, MASSACHUSETTS: AMHERST COLLEGE

Wild Stocks

1. Oregon-R: 515, Inbreeding; generation 515 on 67k1
2. Oregon-R: 100, mass culture, from $ #1 at generation 100
3. Oregon-R: 200, mass culture, from $ #1 at generation 200
4. Oregon-R: 300, mass culture, from $ #1 at generation 300
5. Oregon-R: 400, mass culture, from $ #1 at generation 400
6. Oregon-R: 500, mass culture, from $ #1 at generation 500
7. Samarkand 204: inbred for 204 generations; mass culture since 53h4
8. Samarkand 204-202: from $ #7, inbreeding, generation 202 on 67k1
9. Samarkand 204-100: mass culture, from $ #8 at generation 100
10. Samarkand 204-200: mass culture, from $ #8 at generation 200

Chromosome 1

11. B
12. Basc (Muller-5): sc^{S1} B InS w^a sc^8
13. cm
14. sm ct^6
15. cm ct^6 sn^2
16. cm ct^6 sn^3
17. cm ct^6 sn^3 oc/ y f:=
18. cm ct^6 sn^3 sn/ y f:=
19. cm ct^6 sn^4
20. cm ct^6 sn^4 oc ptg/ y f:=
21. cm ct^6 sn^5/ y f:=
22. cm ct^6 sn^{34e}
23. cm ct^6 sn^{36a}/y f:=
24. cm ct^6 sn^{36a} sn^2 oc ptg/ y f:=
25. ct^6 oc/ y f:=
26. ct^n oc/ y f:=
27. ct^n sn^3 sn^2/ y f:=
28. ec rb^{64f14} cv/ y f:=
29. Ext/FM6
30. f od car
31. g
32. g_2 sd
33. g_2 pl
34. g^3
35. g^3 sd
36. g^{50e}
37. g^{53d}
38. g^{53d} sd
39. g^w
40. lz^3 m/ y f:=
41. lz^3 v/ y f:=
42. m g^{53d}
43. oc/ y f:=
44. oc ptg/y f:=
45. pn^2
46. r^{39k} f B/ In(1)AM
47. ras dy
48. ras^2 m/ y f:=
49. rb ec
50. rux^2 vs^{62a}
51. sc z ec ct^6
52. sc^{S1} At f In49 w^a sc^8/ oc ptg
53. sd^{58d}
54. sn/ y f:=
55. sn_2 oc/ y f:=
56. sn^2
57. sn^2 oc ptg^3/ y f:=
58. sn^3
59. sn^3 g^{53d}
60. sn^3 oc/ y f:=
61. sn^4
62. sn^4 oc ptg^3/ y f:=
63. sn^{36a}/ y f:=
64. un od
65. v
66. v fw
67. v g^{53d} sd
68. v wy^2 g^2
69. vs
70. w
71. w/+=
72. w sn^5/ y f:=
73. w^a
74. w^e
75. w^e sn/ y f:=
76. wy^2
77. wy^2 g_2
78. wy^2 g^2
79. wy^2 g^3
80. wy^2 g^4
81. wy^2 g^{53d}
82. wy^2 g^6 sd
83. y ct^6 ras^2 f
84. y g^{53d} sd/+=
85. y sp-w
86. y sn^{50k} ras^2/ y f:=
87. y w^a m f car
88. y^2 sn^3 ras^4 m/B.Basc
89. y^2 w^a cv sn^{55a} v/+=

Chromosome 2

90. a px or
91. a px or If
92. a px sp
93. al cl b c sp^2
94. b
95. b If
96. b nub pr
97. b Bl vg bw/Cy, bw^{45a} or^{45a} sp^2
98. b Bl vg bw/Roi, bw^{45a} or^{45a} sp^2
99. Bl L^2/Cy, sp^2
100. bw
101. cl
102. cn bw
103. Cy, dp^{Th} Bl L^4 sp^2/Pm, ds^{33k}
104. hp
105. L^4
106. ltd
107. net^{38j} b^{38j} cn^{38j} bw^{38j}
108. or^{45a} sp^2
109. or^{49h}
110. sca^{65131}
111. sca l(2)C/SM5, Cy lt^6 sp^2
112. vg^{51h25}
113. vg^{-B}/Cy L^4 sp^2
114. vg^{nw}/Roi, bw^{45a} or^{45a} sp^2
115. vg^{U}/Roi, bw^{45a} or^{45a} sp^2

Chromosome 3

116. bar-3
117. cu
118. D/Gl
119. D^3 Sb/Payne
120. Dl^7/Ubx^{130} e^s
121. gl^3
122. h
123. h st
124. h th st p^p cu sr e^s (hes)
125. p^p
126. rsd
127. ru h th st cu sr e^s ca (rucuca)
128. ru h st cu sr e^s ca (ruc-th)
129. ru h st cu ss ca
130. ru h st p^p ss Su^3-ss e^s
131. ru st ss ca (rusca)
132. se^{50k}
133. se ss
134. se ss Su^3-ss
135. sr Dl^{6115} e^s/DCxF
136. ss
137. ss^{ax}
138. ss bx Su^2-ss
139. TM1, Mé ri sbd^1/D^3
140. TM3, Sb e Ser/D^3

Chromosome 4

141. $ci\ ey^R$
142. ci^D/ey^D
143. ey^2
144. spa^{pol}

Multiple Chromosomes

145. cm sn^3; Ubx^{130} e^s/T (2;3)Xa
146. ras^{4m}/ y f:=;Bl/Cy, bw^{45a} or^{45a} sp^2
147. +/y f:=; bw; e; spa^{pol}
148. bw; e
149. bw; h
150. bw; e; spa^{pol}
151. cn bw; se^{50k} e^{60h}
152. Cy, bw^{45a} or^{45a} sp^2; TM3, Sb e Ser/T(2;3) gl^{63d29}, bw
153. Cy L^4, sp^2; se^{50k}/T(2;3), Bl vg^{51h25}, se^{50k}
154. Cy, SM5;TM1, Mé ri sbd^1/T(2;3)gl^{63d29}
155. Cy, sp^2/Pm, dp b;DCxF/Sb sr
156. net or^{45a} sp^2; ru bv
157. vg^{51h25}; se^{50k}
158. vg^{51h25} bw; se^{50k} e^{60h}
159. vg^{51h25}; se^{50k}; spa^{pol}
160. Ubx^{130} e^s/T(2;3) Xa

Deficiencies

161. Df $(1)g^1$, f B/In (1)AM
* Df $(2)vg^{-B}$ in stock 113

Inversions

* In(1)AM in stocks 46, 161
* Ins(1) Basc (Muller-5) in stocks 12, 88
* In(1) FM6 in stocks 29, 168, 169, 170, 171, 172, 173, 174, 175, 176, 177, 178
162. In(1) sc^{260-22}, sc^{260-22}
163. In(1) sc^{J1}; Del 24
* Ins sc^{S1} 49 sc^8 in stock 52
* In(2) bw^{V1} (Pm) in stocks 103, 155
* Ins(2) Cy in stocks 97, 99, 103, 111, 113, 146, 152, 153, 154, 155
164. In(2) S^{960}/Cy, S^{e}E-S
* Ins (2L) t, (2R) Cy in stocks 98, 114, 115
* Ins (3) DCxF in stocks 135, 155
* In (3) Mo, Sb sr in stock 155
* Ins (3) Payne (LVM) in stock 119
* Ins (3) TM1, Mé ri sbd^1 in stocks 139, 154
* Ins (3) TM3, Sb e Ser in stock 140
* Ins (3) Ubx^{130} in stocks 120, 145, 160

Translocations

165. T(2;3), bw; h
166. T(2;3) gl^{63d29}, bw; e^{60h}
167. T(2;3) gl^{63d29}, se^{50k}
* T(2;3), Bl vg^{51h25}; se^{50k} in stock 153
* T(2;3) gl^{63d29} in stock 154
* T(2;3) gl^{63d29}, bw in 152
* T(2;3) Xa in stocks 145, 160

Triploids

168. cm ct^6 sn^3/FM6, y^{31d} sc^8 dm B & FM6
169. cm ct^6 sn^4/FM6, y^{31d} sc^8 dm B & FM6
170. ct^n sn^3/FM6, y^{31d} sc^8 dm B & FM6
171. cv rb^{64c10} ec/FM6, y^{31d} sc^8 dm B & FM6
172. g^{53d} sd/FM6, y^{31d} sc^8 dm B & FM6
173. rb cx/FM6, y^{31d} sc^8 dm B & FM6
174. sn^3 oc/FM6, y^{31d} sc^8 dm B & FM6

175. v m/FM6, y^{31d} sc^{8} dm B & FM6
176. vs cm/FM6, y^{31d} sc^{8} dm B & FM6
177. wy^{2} g^{53d} sd/FM6, y^{31d} sc^{8} dm B & FM6
178. wy^{2} g^{H}/ FM6, y^{31d} sc^{8} dm B & FM6

BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA
Department of Zoology

Wild Stocks

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

Chromosome 1

100 B
101 B a sc
102 bb^{1} w^{e}/yf:= $C_{(1)}D_{X}/B^{s}\cdot Y$
103 br
104 br ec/y
105 Bx^{3}
106 cm ct^{6} sn^{3}
108 ec
110 Hw^{49c}/FM1, $y^{31d}sc^{8}$ w^{a} lz^{s} B
111 In(1) dl49, y H w m^{2} g^{4}/yf
113 kz $g^{2}B/y$
114 Df(1) N^{8}/dl 49, y Hw m^{2} g^{4}
115 oc ptg^{3}/ClB
116 sc ec cv v g^{2}f/FM3, y^{31d} sc^{8} dm B l
117 sd
119 sx vb^{2} sy/FM4, y^{31d} sc^{8} dm B (extra Y floating)
122 w
123 w^{a}
124 w^{bl}
125 w^{bl}ec/FM4
126 w^{cf}
127 w^{cf}/FM4
129 w^{ch}wy
130 w^{ch}wy/FM4
131 $w^{co}sn^{2}$
132 w^{co}ec/FM4
133 w^{e}
140 y
141 y ac v/yv
142 y ac sn^{3} v
143 y ac sn^{3} B
144 y ac sn^{3} sx vb^{2} sy/y^{2} sc^{51}B v w^{a} sc^{8} (dl 49)
145 y ac v
150 y ac Dp w^{a}, $(w^{a})2/y^{2}$ sc w^{561} ec
151 yf /y ac Dp $(w^{a})_{2}$
155 y sc
156 y sc m f^{5}
157 y sc/y ac^{+} $sc^{+}\cdot Y$
159 y sn^{3}
160 y w
161 y w spl sn^{3}/yf
162 In(1)y^{2}In(1)w
164 y^{2} B v w^{a} (y^{2} sc^{S1} B In49 v w^{a} sc^{8})
165 y^{2} cv v f
166 y^{2} cv v f car
180 X^{c2} f car/yf
181 yw/w^{vc}/w (ring)
182 X^{c2}In(1)w^{vc}, w^{vc}/y w lz/$sc^{8}\cdot Y$
183 X^{c2}In(1)w^{vc}, w^{vc}f/In(1)w^{m4}, yw^{m4}/Y/Y
184 y^{59b} su-w^{a} w^{a}, $XY^{L}\cdot Y^{S}$/yw bb
186 y w bb/$X\cdot Y^{S}$, yw $Y^{S}/Y^{L}\cdot bb^{+}$ ac^{+} y^{+} sc^{8}
187 In(1)EN, $Y^{o}\cdot B$ f v w y $\cdot$ $Y^{L}\cdot y^{+}/y^{2}$ su-w^{a} w^{a} bb
188 yw$\cdot Y^{S}/Y^{L}$/yf
189 $y^{2}\cdot Y\cdot B^{S}$/y/yf

Chromosome 2

200 a px sp
201 al
202 al dp b pr Hx
203 al dp b pr Bl c px sp/SM5, al^{2} Cy lt^{v} sp^{2}
205 al dp b pr c px sp/Cy pr
206 al dp b pr cn vg c a px bw mr sp/S^{2} Cy lt^{3} pr^{+} Bl cn L^{4} sp^{2}
207 Antp.^{B}/NS
208 b
209 Bl/Cy bw^{45a} sp^{2}
210 Bl L^{2}/SM5, al^{2} Cy lt^{v} sp^{2}
212 bw
214 c
215 cg c/U
216 cl
218 cn bw
220 In(2L)t esc c sp/SM5, al^{2} Cy lt^{v} sp^{2}
225 l(2) gl cn bw/Cy al^{2} lt^{3} L^{4} sp^{2}
226 L^{4}
228 pr cn ix/SM5
229 pr en
230 Sco/Ins (2L+R)Cy, dp^{txI} Cy pr Bl cn^{2} L^{4}
232 vg
233 vg^{no}

Chromosome 3

301 cp in ri p^{p}
302 cu
303 cv-c sbd^{2}
308 Gl Sb/LNM
310 gro/D
311 h
312 Ly/D^{3}
313 mwh
314 mwh e
315 p^{p}
316 ru h st p^{p} ss e^{s}
317 ru h th st cu sr e^{s} ca
318 red
319 se

320 se h
321 se dn Sb/LVM
322 se Ly dn/LVM
323 ss
324 ss^{a}
325 ss^{a-B}
327 tet^{Bd}bilat
328 th st cp
329 th st Pc Scx p^{p} ss/ TM1, Mé ri
340 In(3LR)TM; Mé/In(3LR) Ubx e^{s}
350 Pc/T(2,3)Mé

Chromosome 4

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

Multichromosomal

500 w; vg
501 X^{c2}, t; en/y f
502 y; Cy/Pm
503 yf/Y; fes $Dp(1)y^{+}$ Tft/Cy x sc^{19}/Y; fes $Dp(1)y^{+}$ Tft/Cy
504 y ac; esc^{D}
510 y; mwh
511 y; D/tra red
512 ec; sv^{n}
513 y w f/y^{31d} sc^{8} f^{3n} v; Df(4), Dp y^{+} ac^{+}/ey^{D}
514 b;p^{p}
515 Cy/Pm;D/Sb
516 vg; se
517 se h; ci ey^{R}
518 sn^{3}; cn bw; ri
519 y; Gla/cn bw; ri_{R}
520 yf; bw; e; ci ey^{R}
521 y; al; h
522 b; Msc/+

Triploid

552 y^{2} sc w^{a} ec/FM4
553 oc sn^{3}/FM6, y^{31d} sc^{8} dm B
554 cm ct^{6} sn^{4}/FM6
555 g^{H} wy^{2}/FM6

Translocations

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

Drosophila species

700 D. simulans wild
800 D. virilis Pasadena wild
801 D. virilis scarlet
900 D. montium Abidiaw
950 Zaprionus vittiger

HOUSTON, TEXAS: THE UNIVERSITY OF TEXAS
M.D. Anderson Hospital and Tumor Institute

Tumor Stocks

1 tu^{h}
2 tu^{h}Cy
3 w^{e} sn Bl^{t}/dl-49 y Hw
4 bw tu
5 st sr e^{s} ro ca; tu^{36a}
6 tu^{g}
7 aa tu^{36e}
8 gl/Pm; se e^{11} tu^{49h}
9 tu^{wps}
10 tu^{50d}
11 vg mt^{A} bw
12 mt^{A}
13 vg mt^{A} al sp
14 vg mt^{A} sp
15 vg bw tu
16 vg bw tu/al sp
17 vg tu
18 tu^{47}
19 tu^{47}Cy
20 tu^{50i}
21 tu^{is-21}
22 tu^{50j}
23 tu^{51m}
24 tu^{bs}
25 tu^{54e}
26 tu vg; y^{2};ca
27 tu e
28 tu w
29 Su-er tu bw; st er Su-tu
30 b(Su-er)$^{+}$bw; st er
31 tu PW
32 tu^{h} Cy
33 F15/tu^{wps}
34 F114/tu^{wps}
35 F1191/tu^{wps}
36 F129 tu^{wps}Cy
37 F129 tu^{h}
38 tu^{48j} mt^{A}

CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGO
Department of Zoology

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

Wild type

1 Chicago wild-type

Chromosome 1

2 Bx^3 ma-l
3 Bx^{46}
4 fu^{57a}/FM-1
6 lix
7 pn^{51n8}
8 pn g^2
9 pn rb
10 sc^2 pn
11 sc^{10-1}/y Hw
12 sc z^{59d15}
13 sc z^1
14 sc z^m
16 y ac br pn w^e spl
17 y ac z ec ct
18 y z^a

Chromosome 2

19 bw^D
20 bw^{75}
21 bw^{81}
22 bw^{5-}/Cy cn^2 L^4 sp^2
23 In(2R)bw^{V34}, Cy/al dp b Bl c px sp
24 In(2LR)lt^{m3}
25 T(2;3)lt^{m29}
26 pr ltd

Chromosome 3

27 ru h th st cu sr e^s ca

Chromosome 4

28 spa^{Cat}/ci^D

Inversion-X

29 In(1)AM/M(1)o f
30 In(1)EN, y/y f:=
30b In(1)J1, sc^{J1}/Del(1)24
31 In(1)rst^3, y w^{60i29} rst^3 car bb
32 In(1)sc^4, y sc^4 f v cv w^a_4/y w
33 In(1)sc^4 dl-49 sc^8_4, y sc^4 dl-49 w^a sc^8/y f:=
34 Ins(1)sc^4 S, y sc^4 B InS/sc^8·Y/y f:=
35 In(1)sc^7, sc^7
36 In(1)sc^8, sc^8
37 In(1)sc^{260-14}
38 Ins(1)sc^{S1} sc^8, y sc^{S1} car m w^a sc^8/ In(1)dl-49, y w lz^s
39 In(1)sc^{V1} v/Ins(1)sc^8 dl-49, y^{31d} v f B sc^8
40 In(1)w^{m4}, w^{m4}
41 In(1)y^4, y^4

Deficiencies - Duplications - X Chromosomes

42 w^{-59k13} spl/Dp(1;3)w^{Vco}
43 Df(1)w^{258-45}, y w^- spl dm; Dp(1;3)w^{Vco}/y w f
44 Df(1)w^{258-45}, y w^-; Dp(1;3)w^{Vco}/y w f
45 Dp(1;2R)w^{51b7}; y w rst^3/y w f
46 Dp(1)$w^{vc6094b}$/y^S w y·Y^L y^+/y w/sc^8·Y
47 Dp(1;3)$w^{m264-58a}$/+; y w f Y^L·Y^S/y w/sc^8·Y
48 Dp(1;3)$w^{m264-58a}$, Su-V/Su-V; Y^S w y·Y^L y^+/y w
49 Dp(1;3)$w^{m264-58a}$, Su-V^+/Su-V^+; Y^S w y·Y^L y^+/y w

Reversed Acrocentrogenic

50 B^S·sc^8/Ins(1)sc^7 AM

Ring-X

51 X^{c1}, y/sc^8·Y/y v f car
51b X^{c2}, cv v f/C l B
52 X^{c1}, y v/y w f:=
52b X^{c2}, f/y f:=
52c X^{c2}, y B/y f:=

Reversed Acrocentric

53 RA, y ac sc pn -- In(1)sc^8/In(1)sc^8 (C.O.J-3) y^- ac^- sc^8 w^a/sc^8·Y, y
53b RA, def(1)60g, dl-49, y Hw B/Dp(1;f)60g, y^{31d}/X·Y, y v
53c RA, def(1)60g, y B/Y, su^+-f/y

X with Y fragments Attached

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

Attached X-Y; no free Y

57 y^2 su-w^a w^a Y^L·Y^S/y/0
58 y w f Y^L·Y^S/y w/0
59 y w spl Y^L·Y^S/y w/0

Altered Complete Y's

60 B^S Y/y^2 su-w^a w^a/y
60b B^S Y/y^2 su-w^a w^a
60c B^S Y^L·bb^+ Y^S y^+/y v; bw
61 sc^8·Y, y^{54e} ac^{54e}/y v; bw
62 Y w^+ Co/y w^a_{54k}
63 sc^8·Y: bw^{54k}/y v; bw

Y^S Fragments

65 Y^S/g^2 B·Y^L/y f:=
66 Y^S: y^+ bb^+-5/B·Y^L/y w
68 Y^S: y^+ bb^+-7/g^2 B·Y^L/y w
69 Y^S: y^+ bb^+-8/y g^2 B·Y^L/y v bb; bw

70 $Y^S \cdot Y^S$/sc $sc^8 \cdot Y^L$/y f:=
72 $sc^{V1} \cdot Y^S$/y v f bb$\cdot Y^L$/y f:=

Y^L Fragments

73 sc$\cdot Y^L$/y ac w^a ct^6 f$\cdot Y^S$/y f:=
74 $sc^{S1} \cdot Y^L$#2/y ct^6 f$\cdot Y^S$/y w
75 Y^{cL}: bb^+-12/y w f$\cdot Y^S$/y w
76 Y^L-13/y ct^6 f$\cdot Y^S$/y v bb; bw
77 Y^{cL}-15/y f$\cdot Y^S$/y w

Multichromosomal

79 SM-1, al Cy sp^2/In(2LR)102 ds^w sp^2; Ins(3LP,3RC)Sb e^s/Ubx e^s
80 sp^2; st C_3G ca/TM-1, Mé ri
81 Su-V; Y^S w y$\cdot Y^L$ y^+/Y/y w
82 y/$sc^8 \cdot$Y; ru h th st p^p cu sr e^s
83 w; Cy/Pm; CxD/Sb, In(3R)Mo

NEW YORK, NEW YORK: COLUMBIA UNIVERSITY
Department of Biological Sciences

The stocks bearing sex transformation genes listed in DIS 42 are no longer being carried at Columbia. All except #1 and #4, which have been lost, may be obtained from D. J. Komma, Department of Microbiology, University of Illinois, Urbana.

LE MARS, IOWA: WESTMAR COLLEGE
Department of Biology

Wild Stocks

a-1 Oregon-R

Chromosome 1 (X)

b-1 B
b-2 f
b-3 w
b-4 y
b-5 y sc cv v f car/y f:=
b-6 y^2 w^a v f car/y f:=
b-7 y cv v f

Altered Y Chromosomes

c-1 YB^S (B^S $Y^L \cdot bb^+ Y^S$)/y v ♂ YB^S (B^S $Y^L \cdot bb^+$ Y^S)/y f:=
c-2 Y^{bw+} (Y^L $bw^+ \cdot bb^+ Y^S$)/y v; $bw_♂$ y v; $bw_♀$

Chromosome 2

d-1 bw^4
d-2 bw^{59}
d-3 bw^{81}
d-4 bw^{Am}
d-5 bw^{M58}
d-6 bw^{Mi59}
d-7 cn bw
d-8 cn su-Pm/SM1, al^2 Cy sp^2
d-9 px
d-10 px bw sp
d-11 sp
d-12 vg
d-13 vg^u/Ins(2L + 2R) Roi, bw^{45a} sp^2 or^{45a}

Chromosome 3

e-1 e
e-2 ry^2
e-3 se
e-4 st

Chromosome 4

f-1 ci ey^R
f-2 spa^{pol}

Multichromosomal

g-1 y v; bw
g-2 w; e
g-3 y; e
g-4 bw^{2b}; st
g-5 bw^4; st
g-6 bw^{81}; st
g-7 bw^{M58}; st
g-8 bw^{Mi59}; st
g-9 px; st
g-10 px bw sp^2; st
g-11 px bw^{59} sp^2; st
g-12 px bw^{75} sp^2; st
g-13 px bw^{81} sp^2; st
g-14 sp^2; st
g-15 vg; e
g-16 vg; se

Inversions

j-1 In(1) dl-49, y w
j-2 In(1) EN3, EN3
j-3 In(1) sc^8, In(1) dl-49, sc^8 v f; y f:=

PITTSBURGH, PENNSYLVANIA: UNIVERSITY OF PITTSBURGH
Department of Biology

Wild Type

1 Canton-S
2 Oregon-R
11 Magaliesberg
13 Umgazi River
14 Chicago
15 Roma
16 Pavia
17 Bisignano
18 Sciolze

X-Chromosome

100 B_5
103 f^5
104 Hw^{49c}/FM1
106 Hw^{49c} sn^3/ ClB
108 w
112 Hw^{49c} f^{5}, y f:=
115 y Hw/Ins(1) sc^{S1L}, S, sc^{8R}, sc^{S1+8}, w^a
116 y w sn^3
122 Hw^{49c} m^3 /FM6
126 Su^x - dx dx
127 Su^2-s v (bw)
128 Su^{S2}- v - pr v/FM3 (bw)
129 v f su^W - f

Chromosome 2

222 vg
225 ed dp^{ov} cl
226 ed dp^{1v} cl/In Cy L, Cy, Bl cm^2 L^4 sp^2
227 ed dp^{ol5} cl/In Cy L, Cy, Bl cm^2 L^4 sp^2
228 Pm/SM1

Chromosome 3

302 h
304 se e^{11}
311 ry_B
312 Su^B - pr/In(3R)C, e (pr)
313 Su^2 - Hw bx bxd/TM1
315 h^1 gs th
316 su - t (t)
320 e^{11}

Multichromosomal

501 bw; st
502 bw^{75}; st
505 vg; e^{11}
506 v; bw
507 Cy_{05}/Pm; Ubx/Sb
512 br^3 dx^{st}; ed Su^2 - dx
513 Su - er tu bw; st er su - tu

Co-Isogenic Stocks

900 y $Iso(2;3)_1$
901 sn^3 $Iso(2;3)_2$
902 y Iso $(2;3)_2$
904 y ac $Iso(2;3)_2$
909 yf_{49c}:= /FM6 $Iso(2;3)_2$
911 Hw_{49c}/FM6 $Iso(2;3)_2$
912 Hw^{49c}/yf:= $Iso(2;3)_2$

URBANA, ILLINOIS: UNIVERSITY OF ILLINOIS
Department of Microbiology

Only unusual stocks listed.

1. Zw^a M^{Zw}
2. D InsCXF ru h/tra p; Zw^A M^{Zw}/ $sc^8 \cdot Y \cdot B^S$
3. D InsCXF ru h/tra p; y $Zw^A/sc^8 \cdot Y \cdot B^S$
4. D InsCXF ru h/tra p; y w sn^3 $Zw^B/sc^8 \cdot Y \cdot B^S$
5. D InsCXF ru h/tra p; Zw^B & y Zw^A.=
6. D InsCXF/dsx; Zw^A & y Zw^A.=
7. D InsCXF/dsx; w m f $Zw^B/sc^8 \cdot Y \cdot B^S$
8. SM5/pr cn ix; Zw^A
9. SM5/pr cn ix; car Zw^B

BOSTON, MASS.: NORTHEASTERN UNIVERSITY
Department of Biology

DDT Tolerant

e
vg

Salt Tolerant

e
vg

Populations maintained with a mutant segregating at about .5 for nearly 160 generations

e x FW
e x SP
e x GB
al x FW
vg x FW

EAST LANSING, MICHIGAN: MICHIGAN STATE UNIVERSITY
Department of Zoology

Wild Stocks

Oregon-R
Samarkand
Swedish-b

Chromosome 1

B
f
g
lz^1/ y f:=
m^D
m^D/FM3, y^{31d} sc^8 dm B l
pn
r^9/y f:=
rb^{S1}
sc
sc^{S1} B InS w^a sc^8 (Basc)
v
w
w m
w m f
w^a
w^e
y
y m f/y w f:=

Chromosome 2

al dp b pr c px sp
b
b pr
bw
cn bw
Cy/ds^{33k}, Pm
dp
pk cn
vg

Chromosome 3

e
e se
h th st cu sr e^s ca
se
st

Multichromosomal Stocks

1, 2
v; bw

1, 2, 3
w; bw; st

1, 2, 3, 4
y; bw; e; ci Cy^R

2, 3
bw; e

bw; st

MINNEAPOLIS, MINNESOTA: AUGSBURG COLLEGE

Chromosome 3
(Texas stocks showing delayed recovery from carbon dioxide (TDR stocks).

1. Dly Stock TDR-1
2. Dly Stock TDR-3
3. Dly st ca Stock TDR-orange, yellow

Carbon Dioxide Sensitive Stocks

4. PO
5. TDR-Oregon
6. TDR-Pittsburg

Other Stocks

7. TDR-B
8. Paris

ST. LOUIS, MO.: WASHINGTON UNIVERSITY
Department of Biology

y
D_3 $SbCa_2$/ Payne
se ss ro
$Oregon_8$RC (wild)
b-42 N^8/y Hw In^{49} m^2 g^4
w
bw;st
cn bw sp
Pm dp b/Cy sp^2; Sb/D Cx F
y ct^6 car f/yf
w m f

LAKE FOREST, ILLINOIS: LAKE FOREST COLLEGE
Department of Biology

Wild Stocks

Urbana - 1119.10
Urbana - S

Chromosome 1

B
br
cm ct^6
dy
ec
ec ct^6 v g^3
f odsy car
fa
fo
g^2
rb
ras^2
rg
s
sc cho
sc cv v f
sc cv v g f
sc ec ct^6 v g f
sc ec cv ct^6 v g^2 f/Fm3 By^3/d sc^6 dm
spl
svr
sw
w
w m f
y
y pn
y pn w dm ct^6 sv^3 oc ras v dy g^2 f od car
sw/sc dl-49y V B
y w f

Chromosome 2

al
b
blo
bw
bw ba
c
dp
ex
ft
ho
J^{34e}
L^4
net
px
stw

Chromosome 3

e^{11}
eyg
se
se rt^2 th/Me
st
ss^a
sr
th
ve

Chromosome 4

ey^2
ci gvl ey sv^n
sv^n

Multichromosomal

y; bw;e;ci ey

Inversions

Bl L/Cy
Cy/Pm; D/Sb
SM1 Cy/102; Ubx/Sb
Ly Sb/bal
Muller-5 (basc)
w mf/ClB

Attached-X

y f/f B car (see duplications)
y f/odsy car
y w f/ras^2

Translocations

T(2;Y)A

Deficiencies

$M_a{}^{33}$/bal

Duplications

f B car (long duplication:
f B^+odsy$^+$B odsy car)

NEW HAVEN, CONNECTICUT: YALE UNIVERSITY
Department of Biology

Revision to DIS 41: 29-30.

Delete Nos. 58, 105, 143 and 157.

STOCKHOLM, SWEDEN: UNIVERSITY OF STOCKHOLM
Institute of Genetics

Wild stocks

1 Canton S
2 Karsnäs
3 Oregon
4 Skaftö
5 Karsnäs 60

Isogenic

4 lines, all kept as 1 virg.♀ and 1 brother for c. 100 generations.

Chromosome 1

101 B x y f:=
102 ct^6
103 cv
104 dow/FM6, $y^{31d}sc^8$ dm B
105 Df w^{258-45}, y^2 w/FM4, $y^{31d}sc^8$ dm B
106 Dp (X:X) B^S (RMG), y w^a (1?)•B^S/sc^{S1} In dl-49 v ("doubler")
107 Dp (X:X) B^S (TMG), y sc^4 mf B^S/sc^7 In AM, ptg^4
108 ec ct v f
109 f
110 l w^{47b} H/FM6, $y^{31d}sc^8$ dm B
111 m
112 pn
113 R(1) X^{C2}, y B x y f:=
114 RMC(1), y ct^n oc ptg l car sn^{X2} dl-49 In $ct^l y/Y^{Lc}$ x X•Y^S, oc ptg/ Y^{Lc} ("Y^{Lc} snocty")
115 sc^8
116 sc cv
117 sc cv v f
118 sc cv v car
119 sc^{S1} B InS w^a sc^8 ("Muller 5")
120 sc^{S1} InS w^a sc^8; y^{53i} sc•Y
121 sn^3
122 v
123 vs^{66k}
124 w
125 w ct^6 f
126 w cv sn^3
127 w^a
128 w^a B x y f:=
129 w^{ec2}
130 w^h
131 y
132 y^4
133 y^{16}; sc^8•Y
134 y^{329}
135 y ac sc pn sn^3; sc^8•Y
136 y ac sc pn w rb cm ct sn^3 ras^4 v m g f car/FM6, $y^{31d}sc^8$ dm B
137 y ec ct^6 v f
138 y ec ct^6 v f; sc^8•Y
139 y^2 eq; Df(Y) Y^{bb-}
140 y f car
141 y Hw m g_8f Eb/FM6, y^{31d} sc^8 dm B
142 y sc^4 sc^8
143 FM1, y sc^8 Bf v x y f:=
144 y sc^{S1} B InS w^a sc^8
145 y sc^{S1} B InS w^a sc^8/sc^8•Y x y f:=/ sc^8•Y
146 y sc^{S1} InS w^a sc^8
147 y sn oc x y f:=
148 y v f:= x Y^S•Y^L In dl-49, y v f car
149 y v g f
150 y w ct^6 m f
151 y w^a; w^+•Y
152 y w^{bf} spl sn^3 x y f:=
153 y w sn^3
154 y w sn^3; sc^8•Y
155 y w sn^3/y w sn^3/sc^8•Y & y w sn^3/ sc^8•Y

Triploid

156 sn^3 oc/FM6, y^{31d} sc^8 dm B

Chromosome 2

201 a px sp
202 al b c sp
203 al dp b pr $c_4$px sp/al^2 Cy lt^3 L^4 sp^2
204 al^2 Cy lt^3 L^4 sp^2/Pm
205 b sn vg
206 b pr vg
207 bw^D
208 cn bw
209 Cy/Pm
210 dp b
211 oph
212 Pt/Cy
213 px bw mr sp/ds^{33k} Pm
214 RMC(2L); RMC(2R)
215 RMC(2L), b; RMC(2R), cn
216 S^2 Cy pr Bl cn^2 L^4 bw sp/Ins NS, px sp
217 S Sp Bl bw^D/al^2 Cy lt^3 L^4 sp^2
218 vg
219 vg^D/SM5, al^2 Cy lt^v sp^2

Chromosome 3

301 ca K-pn
302 D^3/Ins P
303 D/Sb
304 e^{11}
305 Gl Sb/LVM
306 ru h st p^p ss e^s
307 ru se h st p^p ss e^s
308 se
309 ss

310 ss^a
311 st
312 st ss e^{11}
313 W

Chromosome 4

401 C(4) AF2, ci ey^R/gvl sv^n
402 ci ey^R
403 ci^D spa^{pol}/spa^{Cat}

Multichromosomal

501 bw; st
502 cn bw; e^{11}
503 sc z is; Cy/T(2;3)Xa/Ubx
504 sp; th
505 T(1;2) B^{bd}/Cy x M(2) e/Cy
506 T(2;3) bw^{VDe4}/Cy
507 v; bw
508 vg; ss
509 y ec ct^6 v f; sp
510 y^{S1} sc^8 InS y^{3P}; al^2 Cy lt^3 sp^2/dp b Pm^1; ru h Ins C x F, D^3 ca/ Sb In(3R) ("Marked Inversions")
511 y v; bw x y v/bw^+•Y; bw
512 y w sn^3/y w sn^3/sc^8•Y; Cy L/+ & y w sn^3/sc^8•Y; Cy L/+

NAGASAKI, JAPAN: NAGASAKI UNIVERSITY
School of Medicine

Wild Stocks

1 Canton-S
2 Erie, Pa.(isogenic)
3 Oregon-RS

Chromosome 1

4 sc^{S1} B InS w^a sc^8
5 sc^8•Y/In(1) dl-49, y B & y f:=
6 y w m f

Chromosome 2

7 cn bw
8 Df(2) bw^5 sp^2/Xa
9 Ins SM1, al^2 Cy sp^2/dp b Pm ds^{33k}

Chromosome 3

10 Sb/Ubx^{130} e^s

Chromosome 4

11 pol

Multichromosomal

12 sc^{S1} (B) In-S w^a sc^8; Ins SM1, al^2 Cy sp^2/ dp b Pm ds^{33k}; Sb/Ubx^{130} e^s; pol
13 y f:= & Ins FM4, y^{31d} sc^8 dm B; Ins SM1, al^2 Cy sp^2/Sp; Sb/Ubx^{130} e^s; pol
14 y sc^{S1} In49 sc^8; bw; st p^p

X-Chromosome Lethals

50 strains

READING, ENGLAND: THE UNIVERSITY OF READING
Department of Zoology

Chromosome 1

w^a
w m f
M 5
y w spl
y v f/sc v f car
y v f/cm ny un
y v f/ec cv m f Bx
various r alleles /M5
Df 1 N^8/dl 49, y Hw m^2 g^4

Chromosome 2

bw
Cy/Gla
vg
sep In 2 L R; ri p^p

Chromosome 3

st
e
Sb/+
Dl/Ubx^{130}, Ubx^{130} e^s

MÜNCHEN, GERMANY: ZOOLOGISCHES INSTITUT DER UNIVERSITÄT MUNCHEN

Wild Stocks

Berlin

Chromosome 1

br
cs^{53}/y w bb
ct^{6}
cv
g^{2}
if^{3}
lz^{50e30}
rb
rb^{66a}
rux^{2}/Y
sc z w^{11G2} ec ct^{6}
sc z w^{2m}
sc z w^{2l}
scp t
sn^{3} lz^{46} ras^{4} v/y w f:=
sn^{65a}/y f:=
spl
v
vs
vs^{66a}
w
w^{a}
w^{a} ec sn^{3}
w^{co} sn^{2}
w^{co} spl cv f
w spl cv f
y
y pn
y sc w^{Bwx}
y sc z w^{a} spl ec/y f:=
y sc z $w^{r,def.}$ spl f/y f:=
y v f
y w
y w spl
y w spl cv f
$y\ z^{a}$
y^{2} sc w^{a} w^{ch} fa/y f:=
y^{2} w^{bf} spl sn^{3}
z
z w^{11E4}

Chromosome 2

al b c sp
b dp
ft
lgl cn bw/Cy
vg bw

Chromosome 3

e
ru h th st cu sr e^{s} ca ("rucuca")
ru^{g}
ru^{g} jv se by
ry
se h
th cu sr e^{s} ro ca ("curoca")
ve h th
W

Multichromosomal Stocks

y v; bw (1;2)
ptg; px pd; su-pd (1;2;3)
FM6, y^{31d} sc^{8} dm B/In(1)AM, y^{2}; pr Bl/SM1, al^{2} Cy sp^{2}; Tp(3)Vno, Vno/In(3LR) Ubx^{130}, Ubx^{130} e^{s}; pol (1;2;3;4)
w; se h (1;3)
w^{co} sn^{2}; ru^{g} (1;3)
w^{co} sn^{2}; se h (1;3)
y; se h (1;3)
z; ru^{g} (1;3)
z; se h (1;3)
w; ey (1;4)
b; se (2;3)
c; e (2;3)
cn; st (2;3)
Cy/Pm; CxD/Sb (2;3)
SM1, al^{2} Cy sp^{2}/In(2LR)102 ds^{w} sp^{2}; In(3L?,3RC)Sb e^{s}/Ubx^{130} e^{s} (2;3)
b; ey (2;4)
ri; ci ey^{R} (3;4)

X Chromosomes with a Y Arm Attached

$X\circ Y^{S}$(A-3), y $w\circ Y^{S}$/y v f/Y^{Lc}
$X\circ Y^{L}$(A-2), y $w\circ Y^{L}$/y v bb/Y"
$X\circ Y^{L}$(U-8e),y $w\circ Y^{L}$/y v bb/Y"
$X\circ Y^{L}$,y^{2} sc^{+} su-w^{a} $w^{a}\circ Y^{L}$ sc^{+}/y f:=/Y

Attached - XY

X $Y^{S}\circ Y^{L}$(115-9 Parker), y^{2} su-w^{a} w^{a} $Y^{S}\circ Y^{L}$ y^{+}/y v bb/O
X $Y^{L}\circ Y^{S}$(108-9 Parker), y^{2} su-w^{a} w^{a} $Y^{L}\circ Y^{S}$/y v bb/O
X $Y^{L}\circ Y^{S}$(2-10 T 13 Parker), y^{2} su-w^{a} $Y^{L}\circ Y^{S}$/y/Y
X·Y, v f $B\circ Y$/y^{2} su-w^{a} w^{a}/O
Y^{S} $X\circ Y^{L}$,Y^{S} y w^{a}rb $f\circ Y^{L}$/y w/O

Closed-X

$In(1)X^{c2}$, w^{vc} f/y w lz
X^{c2} y f & y w f:=

Altered Y's

sc^{8} Y/y w/y (y^{+} ac^{+} $Y^{L}\cdot bb^{+}$ Y^{S})

Deficiencies

$Df(1)N^{8}$/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
$Df(1)N^{264-105}$/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
$Df(1)w^{258-11}$, y/dl-49, y Hw m^{2} g^{4}
$Df(1)w^{258-42}$, y/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
$Df(1)w^{258-48}$, y sc^{5} spl; Dp(1;3) w^{vco}/y f:=

Duplications

$Dp(1;3)w^{m264-58a}$/Y^{S} w $y\cdot Y^{L}$ y^{+}/Y/y w; +Su-V

Inversions

In(1)ClB
In(1)ClB/w^a
In(1)N$^{264-84}$, y/FM6, y^{31d} sc^8 dm B
In(1)rst^3
In(1)rst^3, w m y f/M-5
In(1)rst^3, w$^{48-3}$ v f/y f:=
In(1)rst^3, y car bb
In(1)rst^3, y w^{60i29} car bb
In(1)sc^4, y
In(1)sc^8
In(1)sc^8, w^a
In(1)sc^{L8}, car m w^a/y w In49 lz^s
In(1)w^{m4}
In(1)w^{m4}, ct^6 f
In(1)w^{m4}, f
In(1)w^{m4};Cy/En-var-7
In(1)w^{m4}; Y's extra
In(2)Cy
In(3L)61A; 71A
In(3L)P, gm/R Ly
In(3L)P, Mé/D^3 H

Translocations

T(1;3)2B7; 84A5 y/sc^{S1} dl-49, sc v f car/B^s Y
T(1;3)N$^{264-6}$, y/y w dm
T(1;4)w$^{258-18}$(3C4), y
T(1;4)w$^{258-18}$(3C4), y/dl-49, y Hw m^2 g^4
T(1;4)w$^{258-18}$(3C4), y/y w dm
T(1;4)w$^{258-21}$/y w spl
T(1;4)w^{m5}/w; ey
T(1;4)w^{m5}/w; ey ci^R
T(3;4)A12/CxD
T(3;4)f/Mé

TUBINGEN, GERMANY: MAX PLANCK INSTITUT FÜR BIOLOGIE

Chromosome 1

w^a
lz
In(1) sc^8, sc^8
In(1) sc^8, w^a sc^8

Chromosome 2

so

Chromosome 3

se

Chromosome 4

ey^2

BIRMINGHAM, ENGLAND: UNIVERSITY OF BIRMINGHAM
Department of Genetics

Wild Stocks

1 Edinburgh
2 Wellington
3 Florida

Inbred for 70-700 Generations

4 Oregon
5 Samarkand
6 6 C/L

Chromosome 1

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

Chromosome 2

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

Chromosome 3

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

Chromosome 4

21 ey^2

Multichromosomal

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

Inversions

26 ClB/+
27 Muller-5
28 Mé Sb e/He
29 Cy L/Pm; H/Sb
30 y sc^{S1} B dl-49 w^a sc^8
31 s g^3/FM6 y^{31d} sc^8 dm B
32 Df(3)ry^k/Ubx^{130}
33 M 34 Dfd ry^1/Mé Ins ri sbd^1
34 Mn ma-l/y^{31d} sc^8 dm B;TM3 Sb Ser y$^+$/cu$_2$ kar l^{26}
35 Ins(2L+2R)Cy cn^2/In(2LR)Pm al^4 ds^{33k} lt bw^{v1}; In(3LR)DcxF D/Sb
36 y; Ins(2L+2R)Cy cn^2/In(2LR)Pm al^4 ds^{33k} lt bw^{v1}; In(3L)D, D/Sb

Attached-X

37 y x w

ADELAIDE, AUSTRALIA: FLINDERS UNIVERSITY
School of Biological Sciences

Chromosome 1

101 B
102 y apr
103 w
104 sc cv v f
105 y/X^{c2} v f
106 sc^{S1} In- S apr sc^{8} (asc)
107 y/vs^{64j}
108 "sx" + ($X{\cdot}Y^{S}/Y^{Lc}$) (sterilizer stock)
109 C1B/y^{2} apr ec cv ct v f
110 N^{8}/dl^{49} y m^{2} g^{4}
111 y/asc/sc^{8} $Y{\cdot}B^{s}$
112 y/yv/sc^{8} $Y{\cdot}B^{s}$
113 y/X^{c2}v f/sc^{8} $Y{\cdot}B^{s}$
114 cv ct v
115 w ct f

Chromosome 2

201 b cn c bw
202 S/Cy cn^{2} L^{4} Sp^{2}
203 dp

Chromosome 3

301 ru-cu-ca (ru h th st cu su c^{s} ca)
302 Ly/D^{3}
303 e^{4}

Chromosome 4

401 ci
402 ey^{2}

Multichromosomal

501 bw; st
502 v; e^{4}ro
503 vg; se
504 y; Cy/Pm; H/Sb
505 $X{\cdot}Y^{S}/Y^{Lc}$; bw; st (Sterilizer Stock)
506 $X{\cdot}Y^{S}/Y^{Lc}$; vg; se (Sterilizer Stock)
507 y apr; bw; st
508 yv/sc^{8} $Y{\cdot}B^{S}$; bw
509 e^{4}; dp
510 M^{5}; Cy/Pm; H/Sb

Wild Stocks

601 + S50 iso 60
602 Canton S.

SEOUL, KOREA: EWHA WOMANS UNIVERSITY
Department of Science Education

Wild Stocks

1 Canton-S
2 Choonchun (Korea)
3 Koonsan (Korea)
4 Kwangju (Korea)
5 Kyunggi-1 (Korea)
6 Namhai (Korea)
7 Oregon-R
8 Pusan (Korea)
9 Quelparts (Korea)
10 Sinchon (Korea)
11 Yusoo (Korea)

Chromosome 1

12 w
13 y

Chromosome 2

14 cn bw
15 vg

Chromosome 3

16 cu
17 e
18 Sb^{w}
19 se

Chromosome 4

20 ey

Multichromosomal

21 cn bw; e; ey
22 cn bw; ey
23 cn bw; se
24 cn bw; ey; se

SD

25 SD-72
26 R-1
27 SD^{NH} -2
28 R(SD^{NH} -1)

LONDON, W.C.1, ENGLAND: UNIVERSITY OF LONDON
Birkbeck College, Department of Zoology

Wild Stocks

Oregon-R
Oregon +

Chromosome 1

M-5
w
w m f
v

Chromosome 2

a px
a px pd bw
bw
Cy/B1 L^2
Cy/el b pr lt ltd cn a px pd bw
dp
el b
ltd cn
vg

Chromosome 3

se ss
ss
ss^a

Multichromosomal stock

bw; e

Inversions

$In(1)sc^{S1}L_{sc}{}^{4}R_{sc}{}^{S1}sc^{4}$cv v B/C(1)DX, y f/$B^{S}Y$
$In(1)sc^{4}L_{sc}{}^{8}R_{y}$ $sc^{4}sc^{8}$cv v B/C(1)DX, y f/$B^{S}Y$

Attached-X

$y/w^{bl}wy$ f

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

Inbred Temperature Lines

1 line raised at 25°C for 175 generations (Abeele - Belgium).
3 lines raised at 18°C for 125 generations (Gabarros - Spain), duplicated at 25°C since F_{110}, now at F_{135}.
2 lines raised at 25°C for 287 generations.
4 lines raised at 18°C for 145-160 generations, duplicated at 25°C since F_{140}, now at F_{170}.

These two last items coming originally from Dept. of Botany and Plant Pathology, Pennsylvania State University (see DIS 34)

Wild Stocks

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

Chromosome 1

B
$gt\text{-}w^a$
m
v
w
w^a

Chromosome 2

bw
CyL^4/Pm
ho
sp^2bl^2
stw^2
vg

Chromosome 3

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

Multichromosomal

e;vg
ri;stw^2
w;ri

BRIGHTON, SUSSEX, ENGLAND: UNIVERSITY OF SUSSEX
School of Biology

Wild Stocks

Ore K
Ore +
Ore R
Ore S
N.B.
Edinburgh

Chromosome 1

ma-l x y f:=
M. 5
N8/dl49 $Min^2 y H^w g^4$
chieti vermilion
wild vermilion
vermilion erupt
vermilion
w sn m
w m f
yf:=Ca X pn x ma-l Ca Z pn
y pn X^X x FM6 ♂
y^y/w^{bl} wyf
gt w^a
B

Chromosome 2

ab
b gp
dp ey^2

tu^k B_3
tu^k Ey OrK
tu 55g
tu pr
tu 48^a

Chromosome 3

ri
Rucuca
tt wo
Sb/+
ess^a
tet^{Bd} bilat
ry^2/ry^2

Chromosome 4

Ant x Ey k
ci ey^R
Ci D/spa^{cat}

Multichromosomal

SM5/Bli^2; st/st
tu bw; $+^{su-tu}$
tu bw su er; st su tu-er
tu^k bw; TM3/sb
tu^k TM3/sb
vg/m
sc^{67f} ab
l(2)gl cn bw/SM5 al^2 Cy lt v sp^2
gt bb^{11}/Cl B

CHIBA-SHI, JAPAN: NATIONAL INSTITUTE OF RADIOLOGICAL SCIENCES

Wild Stocks

Oregon-R
Canton-S
Swedish-C

Chromosome 1

sc^{S1}B Ins w^a sc^8(Muller-5)
sc^8Y/B & y f:=
sc^8Y B^S/sc^{S1}B In49 sc^8 & y f:=
v f B/y^2 su-w^a w^a bb
y & y/sc^8Y

Chromosome 2

Cy/Pm
dp^T Sp cn bw sp/$S^2(ls^2)$Cy, InL cn bw sp

Multichromosomal

Cy/Pm; Sb/Ubx
sc^8Y/In49 B & y f:=; bw
vg; e
$X^{c2}v$ & y; bw; e; ey
y sc^8 In49 sc^{S1}; bw; st
y sc^8 In49 sc^{S1}; dp bw; st p^p
y w m f; sp

SYDNEY, NEW SOUTH WALES, AUSTRALIA: UNIVERSITY OF SYDNEY
Department of Agricultural Botany

Wild Stock

1 Oregon-R-C

Chromosome 1

2 N4
3 w
4 B
5 BB
6 w f
7 y w^e
8 f B
9 ct v f
10 sc cv v f

Chromosome 2

11 dp
12 b
13 b vg
14 b j
15 vg
16 vg pr
17 al dp d b c px sp/Cy sp

Chromosome 3

18 e

Multichromosomal

19 v; bw
20 bw; st
21 dp; e
22 y w; dp
23 al; e^{11}
24 al; e^s
25 R; ci ey^2

Special Stocks

26 Pr/In(3R) e
27 Cy j
28 Cy/Pm
29 y/B
30 y/lz^{57j}
31 Muller-5 (sc^{S1} B In S apr sc^8)
32 ClB---Multiple (y w^a ec cv ct v f)
33 w f/ClB
34 w f/ClBB
35 oc ptg^3/ClB

VARANASI, INDIA: BANARAS HINDU UNIVERSITY
Department of Zoology

Wild Stocks

a Canton-S
b Oregon-R
c Kerala

Chromosome 1

ClB
X^{c-2}
w^i
w^a
w^{bl}
w^e
w^{co}
w^h
82-y sc^{S1} B In^{49} ct^{ns} sc^8

Chromosome 2

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

Chromosome 3

se cu
se h
ss^a
Ly/D_3

Chromosome 4

ey^2
ci^w

Multichromosomal

fs 13-y+ ac+ sc^8 Y/y B; B_w^D; st^+ ♂ & y f:=; bw^D ♀
J 102 - Y^S X In EN In 49 y Y^L; st (no free Y)

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

Wild Stocks

a1 Wild type from México
a2 Florida
a3 Oregon-R
a4 Canton-S

Chromosome 1

b1 B
b2 w m f
b4 w
b5 y
b6 y w^a
b8 v
b9 w m f/ClB
b15 y v
b16 y w sn^3
b17 y^2 v
b18 y w
b19 y^2 w^a
b20 gt w^a

Scute Alleles

c2 y^{S1} sc^8B f In-49 v

Combinations of scute

d1 M-5; sc^{S1} B In-S w^a sc^8
d2 sc^{S1} In-S w^a sc^8

Altered Y's

f1 y/sc^8 Y
f2 y w sn^3/sc^8 Y
f3 y w^{m4}/sc^8 Y
f4 y^2 w^a/sc^8 Y
f8 y cv v f car/sc^8 Y
f10 y^{S1} sc^8 B f In 49 v/sc^8 Y

Chromosome 2

g1 bw
g3 cn
g4 dp
g5 L^2

Chromosome 3

h1 se ss
h2 st
h3 e
h4 se
h5 ry^2
h6 ca^{nd}/TM3 Sb Ser
h7 st C3G ca/TM1 Mé ri sbd^1 (sp^2)
h8 st C3G ca/In(3LR) Ubx^{130}, Ubx^{130} e^s
h9 Tft/SM1, al^2 Cy sp^2
h10 SD-5/SM1, al^2 Cy sp^2
h11 Dl^7/In(3LR) Ubx^{130}, Ubx^{130} e^s
h12 c3G
h15 ru st C3G sr e^s

Chromosome 4

i1 ey^2
i2 br ey^R sv^n

Multiple Chromosomes

j1 y^2 w^a/sc^8 Y; ru st C3G sr e^s
j2 y^{S1} sc^8 B f In-49 v/sc^8 Y; TM3 Sb Ser/ca^{nd}
j3 bw; st
j4 y;bw;e; ci ey^R
j5 y/sc^8Y; dp
j6 y^2 wa/sc^8Y; e
j7 Cy/Pm; D/Sb
j8 y cv v f car/sc^8Y; dp
j9 y w sn^3/sc^8 Y; ru st C3G sr e^s
j13 SM1 al^2 Cy sp^2/Pm; In(3LR) Ubx^{130}, $Ubx^{130}e^s$/sb
j14 y/sc^8Y; dp
j15 y^2 w^a/sc^8Y; e
j10 y^{S1} sc^8 B f In-49 v/sc^8Y; dp

Closed X

k2 X^{c2}, y v

Attached (compound) Chromosomes

m1 Y^S X•Y^L In(1)EN, Y^S B y•Y^L/y^2 su-w^a w^a bb/0
m2 yf:=/Y & y sc cv v f car
m3 sc cv v f B/y f:=

LIVERPOOL, ENGLAND: UNIVERSITY OF LIVERPOOL
Department of Genetics

Chromosome 1

w sn m
B
y w
w^a
w m f
C1B/w m f

Chromosome 2

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

Chromosome 3

e
se cp e

Multichromosomal

y;bw;st
ŷy;bw;st
M-5;Ly/Mé

BLINDERN, NORWAY: UNIVERSITY OF OSLO
Institute of General Genetics

Wild Stocks

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

Chromosome 1

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

Altered Y's

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

Chromosome 2

35 al^2 Cy, InL, lt^3/b pr Bl lt^3 InCyR L^4 sp^2
36 b j pr cn
37 bw
38 cn bw
39 dp^{51126}
40 fj px sp
41 j^{49k}
42 j^{50e5}
43 L^2/Cy
44 M(2) z/Cy L
45 M(2)50j7/Cy, al^2 lt^3 L^4 sp^2
46 Pm/Cy, al^2 lt^3 L^4 sp^2
47 pr en
48 S^{37b}/Cy
49 vg

Chromosome 3

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

Chromosome 4

62 ar/ey^D
63 ci ey^R
64 ey^2

Multichromosomal

65 sc^{S1}B InS $w^a sc^8$; cn bw; e^{11}
66 bw; st
67 cn bw; e^{11}
68 cn bw; ru h st p^p ss e^s
69 Cy/Pm; H/C Sb
70 y; bw; e; ci ey^R

Deficiencies and Translocations

71 Df(1) N^8/dl-49, y Hw $m^2 g^4$
72 Df(2) Px^2, bw sp/Cy L
73 T(2;3)Cy

CLERMONT-FERRAND, FRANCE: LABORATOIRE DE ZOOLOGIE ET DE BIOLOGIE CELLULAIRE

Wild Stock	Chromosome 1	Chromosome 2	Chromosome 3
Oregon-R-C	B	vg	e
	w		

SEOUL, KOREA: SEOUL NATIONAL UNIVERSITY
Department of Zoology

Wild Stocks

1 Canton-S
2 Daekwanryung
3 Damyang
4 Heuksando-1
5 Heuksando-2
6 Hongdo-1
7 Kanghwa-1
8 Kwangju-1
9 Oregon-R
10 Oregon-R-C
11 Oregon-S
12 Samarkand
13 Seoul-1
14 Seoul-2
15 Seoul-3
16 Seoul-4
17 Suwon
18 Swedish-C
19 Yangdong
20 Yonsei

Chromosome 1

101 B
102 bi ct^6 q^2
103 bo
104 br
105 Bx^3
106 cm
107 ec
108 fa
109 rg
110 sc av v eq
111 sc cv v f
112 t
113 t^2 v f
114 v
115 w
116 w^a
117 w^{bf2}
118 w^{ch}
119 w^{co}
120 w^{col}
121 w^e bbl/ClB
122 y
123 y ac v
124 y_2 sc mf^2
125 y^2 cv v f
126 M-5/y sc^8 y

Chromosome 2

201 a px or
202 a px sp
203 ab
204 al
205 al bc sp^2
206 al dp b pr Bl c Px sp/ SM1 al^2 Cy sp^2
207 al dp b pr c px sp/Cy, pr
208 b lt wxt bw
209 bw
210 bw ba
211 Bl/Cy, bw^{45a} sp^2 or^{45a}
212 Bl L/SM5, al^2 Cy lt^v sp^2
213 c
214 c wt px
215 cn bw
216 Cy/Pm
217 ex
218 he
219 L_4
220 L^4
221 Pd
222 pr
223 vg
224 wt

Chromosome 3

301 aa h
302 bul
303 ca
304 cu
305 D/GL
306 gl
307 Gl sb/LVM
308 h
309 jv
310 p
311 Pc/TM1, Mé ri
312 ra
313 ro
314 ru
315 ru h st p^p ss e^s
316 Sb/In (3LR) Ubx^{101}
317 Sb/TM3, Ser ri p^p
318 se
319 se h
320 ss
321 st
322 st in ri p^p
323 th

Chromosome 4

401 ci
402 ci gvl bt
403 ey
404 pol

Multichromosome

501 bw; st(2;3)
502 Cy/Pm; D/Bd (2;3)
503 Cy/Pm; Sb/Ubx (2;3)
504 M-5, Cy/Pm; Sb/Ubx (1;2;3)
505 ptg; px pd; su-pd (1;2;3)
506 v; bw(1;2)
507 vg; se (2;3)
508 w; vg (1;2)

Attached-X

601 br ec/y^{3d}

Duplications

701 Dp (2;3)S

Inversions

801 Muller-5
802 vg^{nw} Hia/SM5 al^2 Cy lt^L sp^2
803 vg^u/Roi, bw sp or

Translocations

901 Su Cy/l(j)chro-2
902 T(1;2)Xa/Sb bx^D

LYON FRANCE, UNIVERSITÉ DE LYON
Section De Biologie Générale et Appliquée, Biologie Cellulaire

Wild Strains

Lyon
Algerie

SÃO PAULO, BRASIL: UNIVERSIDADE DE SÃO PAULO
Faculdade De Filosofia, Ciencias E Letras

Wild Stocks

Oregon-R
Oregon-R-C
Swedish-C
Samarkand

Chromosome 1

B/C(1)DX, y f

Chromosome 2

al dp b pr c px sp
Pin Y^t/SM1, al^2 Cy sp^2
Cy L/Pin

Chromosome 3

ru h th st cu sr e^s ca
TM3, ru Sb Ser/ru Pr

Multichromosomal

1) SM1, al^2 Cy sp^2/In(2LR)bw^{V1},bw^{V1}; Sb/In(3LR) Ubx^{130}, Ubx^{130} e^s
2) Y; In(2L)Cy + In(2R)Cy/In(2LR)bw^{V1}, bw^{V1}, Sb/ In(3L), D
3) y T(1;2)^6/B^S Y
4) al^2 Cy lt^3 L^4 sp/Pin ds^{33k}; Ubx^{130}, e^s/Sb (CyL/Pin; Ubx/Sb)
5) y v bb/O
6) X•Y, v f B/O

PÔRTO ALEGRE, BRAZIL: UNIVERSIDADE FEDERAL DO RIO GRANDE DO SUL
Seccão de Genética

Wild strains from: Buenos Aires, Oregon, Leningrado, Brisbane and Eldorado (Rio Grande do Sul, Brazil)

Chromosome 1

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

Chromosome 2

pr
ltd
cn
bw
pd
vg
L
dp
st bw
vg st

Chromosome 3

p
e
se
e se

JERUSALEM, ISRAEL: THE HEBREW UNIVERSITY
Laboratory of Genetics

Additions to DIS 42:21

pn stocks

y pn.= /FM6
pn^2
pn^2/FM6/w^+Y; ca K-pn
pn^{59j}
pn^{59j} f od l^{A33}/FM6
pn^{59j} spl l^{3des}/Y.pn^- w^+/y pn.=
pn^{66x2} cv
pn^{66x4} cv f/y f:=
pn^{AA1}
pn^{AW1}
pn^{AW3}
y pn^d
pn^{FG}
y^2 pn^{FG} cv l^{B57}/FM6(1)/Y.ma-l^+
pn^{FS1}
pn^{FS2}
pn^{MS1} sn
pn^{MS4}
y ac sc pn^R/y f:=
y ac sc pn^S

Lethals covered by Y.ma-l^+

42 X-ray induced lethals
1 Di-ethyl-sulfate induced lethal
5 Ethyl-methane-sulfonate induced lethals

Lethals covered by Y.w^+

37 X-ray induced lethals
2 Di-ethyl-sulfonate induced lethals

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

Chromosome 1

Muller-5
ClB
y^2 w cv f

Chromosome 2

vg

Chromosome 3

se cu

KEELE, STAFFORDSHIRE, ENGLAND: UNIVERSITY OF KEELE
Department of Biology

Chromosome 1

B
m
Muller-5
ras^2
v
w
w^a
w m B
y

Chromosome 2

bw
cn
Cy/+
dp b cn bw
vg
vg bw
dp
dp b cn

Chromosome 3

e
se
ve

Chromosome 4

ey^2

Multichromosomal

bw; st
vg; se
bw; e
cn; ve
cn; e
bw; ve
dp; se
dp; e
Cy L^4/Pm; H/Sb

Attached-X

w/ywf:=

STRASBOURG (BAS-RHIN), FRANCE: UNIVERSITÉ DE STRASBOURG
Laboratoire de Zoologie

Wild Stocks

Oregon-R-C

Chromosome 1

B
v
w
y

Chromosome 2

b

Chromosome 3

DcxF/Dfd
e
se

Chromosome 4

ey

Multichromosomal

w; e
y; e
y; se

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

Wild Stocks

1. Oregon-K

Inbred Lines

2. light (F198)
3. straw (F198)

Chromosome 2

4. a px
5. a px pd bw
6. b
7. bw
8. cn
9. Cy/Bl L^2
10. dp
11. el
12. el b
13. el b pr lt ltd cn/Cy
14. el b pr lt ltd cn a px pd bw/Cy
15. lt
16. ltd
17. ltd cn
18. pd
19. pr
20. pr lt
21. pym/Cy
22. vg

Chromosome 3

23. e

NORFOLK, ENGLAND: JOHN INNES INSTITUTE

Wild Stocks

1 Bayfordbury
2 Hampton Hill
3 Samarkand
4 Teddington

Inbred Lines

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

Chromosome 1

10 v
11 w
12 y w

Chromosome 2

13 b pr
14 vg

Multichromosomal

15 Cy L^4/Pm; H/Sb

OBNINSK, USSR: INSTITUTE OF MEDICAL RADIOLOGY
Department of General Radiobiology and Genetics

Appendix to the Stock List presented in DIS 42.

Wild Stocks

Oregon-R
Sevelen

Chromosome 1

w sn
y In dl-49
y sn

Chromosome 2

al b c sp^2

Chromosome 3

D/Sb
$In(3R)\ P^{Fla}$ (homozygous)

BELGRADE, YUGOSLAVIA: UNIVERSITY OF BELGRADE
Department of Zoology

Chromosome 1

w
y/f
ClB

Chromosome 2

vg
bw
dp b
b cn vg
L^2 Cy/Pm

Chromosome 3

e
st

Multichromosomal

bw;e
bw;st

The wild populations from Sremski Karlovci and Beograd.

VALENCIA, SPAIN: VALENCIA UNIVERSITY
High Technical School of Agriculture, Genetics Department

Chromosome 1

y
cm
lz
f
B
Muller-5
fu/ClB
w m f

Chromosome 2

al
dp
vg
pr px
Bl L/Cy

Chromosome 3

se
ss
ro
Ly/Sb

Chromosome 4

ey

Multichromosomal

b; cu se
dp; k

SAPPORO, JAPAN: HOKKAIDO UNIVERSITY
Zoological Institute

X-Chromosome

w B
w

Wild - Sapporo, Japan

Chromosome 2

Cy
cn
vg;se (2;3)

UMEÅ, SWEDEN: UNIVERSITY OF UMEÅ
Department of Genetics

Wild Stocks

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

Chromosome 1

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

Chromosome 2

201. bw
202. al b c sp
203. al b c sp/al^2 In(2L)Cy
204. bw^D
205. Cy/Pm
206. Cy/S
207. fes Alu lt/al^2 Cy lt^3
208. nw^2/Cy RNS
209. pr
210. S^2 Cy pr Bl cn^2 L^4 bw_{sp}/In NSL In NSR px sp
211. S sp Bl bw^D/al^2 Cy lt^3 L^4 sp^2
212. vg
213. vg bw

Chromosome 3

301. ca
302. cd
303. D^3/InP
304. Inv Cx F_1 D^3/Sb In(3R)
305. e^{11}
306. lz s-u (=spontan Uppsala)
307. kar^2
308. ri_2 ss
309. ri^2
310. ri^2 ss
311. ro
312. ru h st p^p ss e^s
313. ry_2
314. ry^2
315. ry^2 cd
316. se
317. ss
318. st
319. st p
320. st ry
321. st ss e^{11}

Chromosome 4

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

Multichromosomal

501. w^{ch}; Su-w^{ch}/Cy (1;2)
502. w^{col}; bw (1;2)
503. w^e; cr-u/Cy (1;2)
504. y w spl; Cy; Ubx^{130}/Xa (1;2;3)
505. bw; cd (2;3)
506. bw; st (2;3)
507. cn bw; e^{11} (2;3)
508. Cy/S; D/InP (2;3)
509. L_2 sp; th (2;3)
510. L^2/+, sp; th (2;3)
511. sp; th (2;3)

Deficiencies and Duplications

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

Translocations

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

Triploids

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

CAMBRIDGE, ENGLAND: CAMBRIDGE UNIVERSITY
Department of Genetics

Only stocks unavailable elsewhere, or not readily available in U.K.,are listed.

Thoday's high sternopleural chaetae stocks

vg4
vg6
dp1
dp2
PAD

Chromosome 2

l(2)gl a px or/SM5

Chromosome 3

$gl^{2\,4}_{e}$
gl^{63}f6
gl^{63}a14
C(3)tr/Ubx^{130}
ve h eyg cp
ve h eyg cp (Oregon background)

Multichromosomal balancers

FM6; Pm; Ubx^{130}; on Oregon: (Selected)
SM5; TM3; on Oregon : (Selected)

Wild Stocks

Dronfield
Histon
Barton
Aries
Southacre

CALCUTTA, INDIA: UNIVERSITY OF CALCUTTA
Department of Zoology

Wild Stocks

a1 Canton-S
a2 Oregon-R
a3 P Ceylon
a4 Nai-C
a5 Samarkand
a6 Bishnupur

Chromosome 1

b1 B
b2 Bx^J
b3 ct
b4 $oc_3 ptg^3$/ClB
b5 sn^3
b6 sn^{36a}/y f:=
b7 sta/y f:=
b8 sx vb^2 sy/FM4
b9 **w**
b10 w^a
b11 w^{bo}
b12 w^{co}
b13 w^e
b14 w^e sn/ClB
b15 w m f
b16 y
b17 y oc/y f:=
b18 y^2 cv v f:=
b19 y ac sn^3 sx vb^2 sy/y sc^1 In dl 49 B v w^a sc^8

Closed X (Stable)

c1 w^{vc}/y w lz^s dl 49/sc^8·Y (Floating Y)
c2 X^{c2} y B & y f:=
c3 y w/w^{vc}/w

Chromosome 2

d1 al dp b bw l(2) ax/SM5, al^2 Cy lt^v sp^2
d2 b cn beta
d3 Bl L^2/SM5, al^2 Cy lt^v sp^2
d4 bw
d5 cg c/U
d6 dp
d7 ds S G b pr/Cy, al^2 lt^3 L^4 sp^2
d8 ds $ft_2 dp^{v2}$ l(2)M b pr/SM5, al^2 Cy lt^v sp^2
d9 fj wt/SM5, al^2 Cy lt^v sp^2
d10 ft
d11 lt bw
d12 lt stw^3
d13 pr en
d14 rdo^2 pr
d15 sm px/SM5, al^2 Cy lt^v sp^2
d16 vg

Chromosome 3

e1 D/dsx red
e2 e^s
e3 red e
e4 ("rucuca") ru h th st cu sr e^s ca
e5 ("ruprica") ru h th st cu sr e^s Pr ca/T (2;3)Mé
e6 se h
e7 th st Pc Scx p^p ss/TM1, Mé ri
e8 th st Pc Scx p^p ss/D
e9 Sb Ubx/Xa

Chromosome 4

f1 ci^w
f2 ey^2

Multichromosomal

g1 Cy/Pm; D/Sb
g2 (M1) y^{S1} sc^8 InS y^{3P}; al^2 Cy lt^3 cn^2 sp^2 $/dp\ b\ Pm^1$; ru h D InsCXF ca/Sb
g3 tu^1 with modifiers
g4 tu^2 with modifiers
g5 y:=; D/tra red
g6 y^2 sc w^a ec:=/FM4; (3N)

Altered Y chromosomes & attached X·Y chromosomes

h1 FR1 Y^S·y cv v f/y f:=
h2 X·Y^S (A-3), oc cv·Y^S/y v f:=
h3 X·Y^S(P-8b) In(1) sc^{S1} EN y^+ f y·Y^S/Y^{cL}/f:=
h4 Y^S/g^2 B·Y^L & y f:= (Stern) (dp^T)
h5 Y^S w y·Y^L y^{55f10}/O/y w:=
h6 y Hw·Y^S y^+/Y^{cL}/y w^a:=
h7 Y^S·X InEN v cv y·Y^L y^+ & y^2 su-w^a w^a bb:= (no free Y)
h8 Y^S·Y^S/sc^8 sc^8·Y^L/y^2 su-w^a w^a bb:=
h9 Y^S·Y^S # 2/y v f·Y^L & y f:=
h10 Y^S·Y^L/sc w oc_2 f·Y^S &y f:=
h11 Y^S X·Y^L ($FR\text{-}1^L$, $U\text{-}8d^R$), Y^S y w^a cv v f·Y^L/ y^2 su-w^a w^a bb:= (no free Y)
h12 Y^S·Y^S·Y^L ($FR\text{-}1^L$, $115\text{-}9^R$), Y^S y cv Y^S·Y^L y^+ & y^2 su-w^a w^a bb:= (no free Y)
h13 Y^{bb}/v
h14 Y^{-bb}/ w sn bb & y v f:=
h15 ("Maxy") $lJ1^+$·Y/lJ1 $sc^{J1(+)}$ In 49 ptg oc B^{M1}/ y sc^{S1} car odsy f g^2 dy v ras^2 sn^3 cm rb ec w pn l sc^8
h16 Y^L/f·Y^S & sc v f:=
h17 Y^L:bb^+?/y ct^6 f·Y^S/y v bb:=; bw
h18 sc^{S1}·Y^L # 2/y f· Y^S/y w:=
h19 y w f Y^L·Y^S/O/y w :=
h20 y w Y^L·Y^S/O/y w:=
h21 Y^L ac^+ y^+ (sc^8 EN c.o. Y B-2)/X·Y^S, y w & y v f:=

h22 X·Y^{L} (K-7), sc^{8} InEN y^{+} f v cv y·Y^{L} & y:=/Y"
h23 X·Y^{L}(P-8b), sc^{8} InEN, y^{+} f y·Y^{L}/Y" & y:=
h24 X·Y^{L}(C-2), y cv v g car bb^{-}·Y^{L} & RA, (ND-27) v f/Y"
h25 X·Y^{L}(U-8e), y w·Y^{L}/Y" & y:=

Deficiencies and Duplications

i1 Df(X)N$^{264-39}$, w^{ch}/FM4, y^{31d} sc^{8} dm B
i2 Df(X)N$^{264-105}$/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
i3 Dp(X;f)107, In(X) sc^{8}, Df(0+ac)w^{a} sc^{8}; Dp(X;f)107
i4 Dp(X;f)118 In(X)sc^{8}, Df(0+ac)w^{a} sc^{8}, Dp(X;f)118
i5 Dp(X;f)112 = Del(X)112/lJ1^{259} y w Y^{L}·Y^{S} & y v f:=
i6 y ac Dp(w^{a})2/ y f:=

Inversions

j1 In(X)N$^{264-84}$, y/FM6, y^{31d} sc^{8} dm B
j2 y In dl-49 sn^{X2} B^{M1}/y f:=
j3 y In dl-49 sn^{X2} bb/y f:=
j4 y^{S1} sc^{8} B InS sc^{S1}/w sn^{5a} bb

Translocations

k1 T(1;2)sc^{19}/y f:=; fes sc^{19i} b pr/Cy, dpTh pr
k2 T(1;4)B^{S} (16A1), y^{2} cv v B^{S} car/y f:=
k3 T(1;4)(4CS)/y f:=
k4 T(1;4)w^{m5} /y f:=
k5 T(1;4)w^{m5} /ey^{D}
k6 T(2;4)a/Cy, pr; ey^{2}
k7 T(2;4)ast^{V}/al^{2} Cy lt^{3} L^{4} sp^{2}
k8 rn T(2;3)/Cy Bl cn^{2} L^{4} sp^{2}
k9 T(3;4)e/Payne, Dfd ca
k10 T(3;4)A13, ve ca/Mé, ca

HELSINKI, FINLAND: UNIVERSITY OF HELSINKI
Department of Genetics

Wild Stocks

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

In addition 49 wild stocks collected from different localities in Finland.

Chromosome 1

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

Chromosome Y

35 X·Y^{L}/Y^{S} (Neuhaus)
36 In(1)w^{m4} (extra Y)
38 In(1)w^{m4}; rl (extra Y)

Chromosome 2

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

Chromosome 3

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

68 ru h th st cu sr e^S ca
69 tra/Mé, T23
70 W Sb/InsCXF

Chromosome 4

71 ci^W
72 spa

Multichromosomal

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

Deficiencies

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

Translocations

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

SEOUL, KOREA: CHUNGANG UNIVERSITY
Department of Biology

Wild Stocks

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

Chromosome 1

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

Chromosome 2

201 a px sp
202 ab
203 al
204 al bc sp^2
205 b
206 b lt wxt bw
207 bw
208 bw ba
209 Bl/Cy, bw^{45a} sp^2 or^{45a}
210 c
211 cl
212 cn bw
213 Cy/Pm
214 ex
215 ho
216 L^1
217 L^4
218 pd
219 pr
220 vg
221 wt

Chromosome 3

301 aa h
302 bul
303 ca
304 cu
305 gl
306 h
307 p
308 ra
309 ro
310 ru
311 se
312 ss
313 st

Chromosome 4

401 bt
402 ci
403 ci gvl bt
404 ey

Multichromosomal

501 v; bw (1;2)
502 w; vg (1;2)
503 M-5;Cy/Pm; Sb/Ubx (1;2;3)
504 Cy/Pm;Sb/Ubx (2;3)
505 Cy/Pm; D/Bd (2;3)
506 vg; se (2;3)

Attached-X

601 br ec/y^{3d}

Duplications

701 Dp (2;3) S

Inversions

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

Translocations

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

HAREN, GRONINGEN, NETHERLANDS: UNIVERSITY OF GRONINGEN
Genetisch Instituut

Wild stocks

1 Bannerdale
2 Davis Oregon
3 Dronfield
4 Groningen I
5 Kolmar
6 Leiden

Inbreds

50 Bayfordbury
51 Iso-Canton
52 M-Oregon
53 Oregon I
54 Pacific
55 "
56 "

Chromosome 1

101 B
102 ClB/sc ec cv ct^6 v s^2 f car bb
103 ClB/fu
104 f^{55f}
105 g
106 lz^{cl} y:=
107 sc^{s1} B In S w^a sc^8 Muller-5
108 sc ct v f car y:=
109 su^2 - s v
110 su^3 - s cv v f y f:=
111 v
112 w
113 w^a
114 w^e
115 w m
116 y
117 y f car
118 y pn
119 y_2w spl
120 y^2
121 y^{59b}
122 w cv sn
123 2^{58g}

Chromosome 2

224 al dp b pr c px sp
225 al dp b pr c px sp/Cy
226 al dp b pr Bl c px sp/Cy
227 a px or
228 a px sp
229 b
230 b cn vg bw
231 bw_D
232 bw^D
233 bw^{75}
234 cn
235 crc/Pm
236 Cy L^4/Pm
237 dp
238 dp - 1
239 dp b cn bw
240 net
241 px sp
242 SD-5/SM1, al^2 Cy sp^2
243 SD-72/SM5, al^2 Cy lt^4 sp^2
244 Su-H/Cy pr
245 vg
246 vg^p

Chromosome 3

301 bx^3 Cbx Ubx bxd pbx/Xa
302 ca K-pn
303 Cbx
304 e
305 e^{11}
306 e^{ug}
307 pbx/Xa
308 ri
309 Ly/In
310 ro
311 ru h th st cu sr e^s
312 ru h st cu sr e^s Ca
313 ru h th st cu sr e^s Pr ca/TM1 Me ri
314 se
315 se cp e
316 ss bx Su^2 ss
317 st
318 su-ve ru ve h th
319 ve
320 ve^2
321 ve^3

Chromosome 4

401 ci^D/ey^D
402 ci^{D-G} spa^{pol}/spa^{Cat}
403 ci^w
404 ci^{57g}
405 ci ey^R/spa^{Cat}
406 Df(4)M-4/ey^D
407 ey^R
408 spa^{pol}

Multichromosomal

501 b(Su-er^+) bw; st er
502 w^a; Cy"O"/Pm; Ubx^{130}/Sb; ci^{D-G} spa^{pol}/spa^{Cat}
503 Cy/Pm; Me/H
504 Cy/Pm; D/Sb
505 dor/FM6; TM3/Sb
506 H^{57c}/Ubx^{61d}; spa^{pol}
507 SM5/Bla; TM3/Sb
508 su er tu bw; st er su-tu
509 T(2;3) bw^{VDe3};Ubx bxd/In(3 RL)Cx
510 v;bw^D
511 v;cn
512 vg spa^{pol}
513 y;bw;st
514 y ec w^{ch} spl; Cy; Ubx^{130}/Xa

ROMA, ITALY: CITTA' UNIVERSITARIA
Istituto di Genetica

Wild Stocks

A1 Oregon
A2 Canton-S
A3 Marzi

Chromosome 1

B1 car bb
B2 en w^e/y f:=
B3 gt v
B4 pn
B5 r^R/y f:=
B6 sc cv v f B/y f:=
B7 sc z ec
B8 sw
B9 w
B10 w^a
B11 w^{Bwx}
B12 w^{cf}
B13 w^{cf}/y f:=
B14 w^{cp}
B15 w^e dy/y f:=
B16 w^{bl}
B17 y
B18 y^1 59
B19 y ac sc pn/y f:=
B20 y cv v f car
B21 y cv v g/y f:=
B22 y fa^n sn^3
B23 y w
B24 y w^a spl rb
B25 y^2 v ma-l
B26 y^2 w^{cf}
B27 y^{31d} y^{Of} w^a f sin/y f:=
B28 y^1 ^{259}/s-5/sc^8·Y

Chromosome 2

C1 b cn c bw
C2 b cn vg bw
C3 Bl stw^{48} blt tuf sp/SM5 al^2 Cy lt^v sp^2
C4 B L^2/SM5, al^2 Cy lt^v sp^2
C5 bw_D
C6 bw^D
C7 cn bw
C8 cv -e
C9 SM5
C10 L^2/SM5
C11 M/Cy
C12 Pin/Cy
C13 Pin L/Cy
C14 pr cn/pr cn
C15 px bw sp/px bw sp
C16 S sp al^2 ltd/SM5, al^2 Cy lt^v sp^2

Chromosome 3

D1 ca K-pn
D2 D^3 H/In(3L)P, Mé
D3 eg/In(3LR)Cy
D4 eg^2/In(3LR) Cy
D5 Gl Sb/LVM
D6 H/Sb sr In(3R)Mé
D7 H^2/Tp(3R) Vno, Vno
D8 Ly Sb/LVM
D9 R Ly/In(3L)P, gm
D10 se ss k e^s ro
D11 st c3G (ca)/In(3LR) Ubx^{130}, Ubx^{130} e^s

Chromosome 4

E1 ci ey^R
E2 ey
E3 pol

Multichromosomal

F1 al L^4 Cy Sp/Pm; H Sb sr In(3R)Mé (2;3)
F2 bw; st (2;3)
F3 bw; e (2;3)
F4 lys rc; ss (2;3)
F5 y; bw; st (2;3)
F6 y;pol (1;4)
F7 y pn/y pn; ci ey/O
F8 y; ru h th st p^p cu sr e^s (1;3)
F9 y^2; bw (1;2)
F10 y^2 cho^2; lys rc (1;3)
F11 y/y; T(2;3)e/SM1; Ubx; pol/ pol (1;2;3;4)
F12 y/y; T(2;3)e/Cy; Ubx; pol/pol (1;2;3;4)
F13 y/FM6; T(2;3)e/SM1; Ubx; pol/pol (1;2;3;4)
F14 y pn v·y^+; T(2;3)e/SM1; Ubx; pol/pol
F15 y w;T(2;3)e/SM1; Ubx; pol/pol (1;2;3;4)
F16 XX/Y·sc^8;T(2;3)e bw/SM1; Ubx; pol/pol (1;2;3;4)

Translocations

H1 T(1;4) B^S(16A_1), y^2 cv v B^S car/y f:=
H2 T(1;4)w^{m5}(3C3), w^{m5}

Inversions

I1 In(1) dl-49, w lz/y v bb:=
I2 In(1) dl-49, y Hw m^2/fa N^{J24a}
I3 In(1) dl-49, y Hw m^2 g^4/Df(1) N^8
I4 In(1) dl-49, v^{Of} f
I5 In(1) sc^{4L}, sc^{8R}, y^{sc4+8} sc v f/y f:=
I6 In(1) sc^7, AM
I7 In(1) sc^8, dl-49, y car v^{Of} f
I8 In(1) sc^8, dl-49, y^{31d} w^a
I9 In(1) sc^8, sc^8
I10 In(1) w^{m4}, w^{m4} /Df(Y) y^{bb}
I11 In(1)w^{m4L}, rst^{3R}, y w^- rst^3/Dp(1;2R) w^{51b7}/y w f:=

I12 In(1) 481 (12 E-F;14B), y_8 bb l^{481}/ FM6, y^{31d} sc^8 dm B/sc^8·Y
I13 Ins(1)sc^{S1}, dl-49, y^{62k} v f car/Y/y f:=

Deficiencies and Duplications

L1 Df(2) bw^5, sp^2/Xa
L2 Df(2) bw^V De^{2L}, In(2R)Cy^R/Gla
L3 Df(2) MS4/SM1, al^2 Cy sp^2
L3 Df(2) MS8/SM1, al^2 Cy sp^2
L4 Df(2) MS10/SM1, al^2 Cy sp^2
L5 Df(2R) Px/Df(2R)P; Dp(2;3)P/In(3R)Mo, sr; w^e
L6 Df(2) rl^{10a} lt cn/Pm, ds^{33K}
L7 M(2) S5/SM5, al^2 Cy lt^v sp^2
L8 M(2) S11/Cy, bw^{v34}
L9 Dp(1;1) B^S(RMG), y w^a·B^S/sc^{S1} dl49, v
L10 Dp(1;f) z^9, Df(1) sc^{J4R}/y f:=

X Chromosomes with a Y arm attached

M1 X·Y^L(C-2), y cv v f car bb⁻ Y^L/RA, (ND-27) v f/Y"
M2 Y^S X· (FR-1) Y^S y cv v f Y^S/y f:=/Y
M3 Y^S X· (P-7), In(1) EN, y f Y^S/y v f/Y

Attached XY Chromosomes

N1 XY^S·Y^L (110-8 Parker), y^2 su-w^a w^a Y^S·Y^L y^+/y v bb/O
N2 XY^S·Y^L (115-9 Parker), y^2 su-w^a w^a Y^S·$Y^L$$y^+$/y v bb/O
N3 XY^S·Y^L (129-16 Parker), y^2 su-w^a w^a Y^S·Y^L y^+/y v bb/O
N4 Y^SX·Y^L, Ins(1)EN, dl-49, Y^S car f v y·Y^L
N5 Y^SX·Y^L, Ins(1)EN, dl-49, Y^S car f v y·Y^L/y^2 su-w^a w^a bb/O

Altered Y Chromosomes

P1 Y w^+ (Y 900)/y w^a
P2 Y w^+ (Y 303)/y w^a
P3 y^+ Y/ yw^a
P4 y^+ Y w^+ (11a)/y w^a
P5 sc^8·Y/X^{c2} y B/y f:=
P6 B^S Y y^+/y^2

MANCHESTER, ENGLAND: PATERSON LABORATORIES
Department of Cytogenetics

Wild Stock

Birmingham

Inbred lines (number of generations)

Oregon v (302)
b pr (205)

Chromosome 1

v/y v f car:=
sc^{S1} B InS w^a sc^8 (Muller-5)
sc ec cv ct v g/In (1) dl-49, y Hw m^2 g^4
sc v f car/ClB
sc ec cv
ct v g
y^2 cv v f
y^2 sc car, Dp sc^{V1}, y^+
y^2 sc v f, Dp sc^{V1}, y^+/B^S Y
In(1) sc^8, y^{31d} sc^8 w^a
In(1)sc^8, sc^8 v f car
In(1)sc^8, sc^8 cv v f/In dl-49, y Hw m^2 g^4
y^2 w^a $(1)^K_m$

Chromosome 2

b pr vg
dp b cn bw
dp^{Th} Cy cn bw/S Sp cn bw
al dp b pr c px sp/Cy
2L dp; 2R px
CyL^4/Pm

Chromosome 3

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

Multiple stocks

dp; e
Cy L^4/Pm; H/Sb
bw^{VA}/Bl L^2, st
v; bw^{VA}/Bl L^2
y^2/FM6 or y^2/Ysc^8;SM1/+
y^2 AM/FM6/ or y^+ Y; SM1/Bl;
Ubx^{130}/Vno; pol

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

Wild Stocks

1. Oregon-K

Chromosome 1

1. w
2. y
3. sc^{S1} B In S w^a sc^8 (Muller-5)

Chromosome 2

1. vg
2. dp b cn
3. dp b cn bw

Chromosome 3

1. e

Multichromosomal

1. bw; st
2. y sc^{S1} In 49 sc^8; bw;st

Attached X Chromosome

1. sc^{S1} B In S w^a sc^8/yvf
2. sc cv v f B/yvf

LONDON, ENGLAND: UNIVERSITY COLLEGE LONDON
Department of Zoology

Wild Stocks

Oregon

Chromosome 1

w
v
f
m
w^a
w^a sn
w m f
ClB/w m f
yy/w^{bl} wy f
N^8/dl-49 y^{Hw} m^2 g^4

Chromosome 2

cn
bw
cu bw
vg
dp

Chromosome 3

Sb/+
Sb ss^a
e ss^a
ss
se ss
se
ve

Chromosome 4

ey^2

Multichromosomal

dp;ey^2
bw;e

TURKU, FINLAND: UNIVERSITY OF TURKU
Department of Genetics

The stock list remains essentially the same as that appearing in DIS 42: 23-24 with the following changes:

Deletions

107 cm ct^6 sn^3 & y f:=
115 ec cv
801 y^2 sc w^a ec/FM4, y^{31d} sc^8 dm B; cn; h

Additions

Wild stocks

5 Turku-1
6 Turku-2

Chromosome 2

dp b pr

Chromosome 3

cu e^s

NEW MUTANTS

Report of J. Puro

spy: spready Puro 61e. Inseparable from T(2;3)spy. Found as a recessive visible viable mutation from the first brood of an X-irradiated wild-type male. Wings horizontally spread in homozygous flies; they often tend to be drooping, hence are likely to get stuck in the food medium. Good viability and fertility. The study of the translocation (see the report by Puro and Arajärvi, this issue) shows the exchange of the left arms of the 2nd and 3rd chromosomes, the points of the exchange being, in 2L, to the left of B1 and, in 3L, between ri and the centromere. Salivary chromosome analysis shows the breaks to be in 2L at 33D or E and in 3L at 79A (to the right of the two thick doublets in 79A but to the left of 79B1 of Bridges' revised map). One or very few bands in 33E may be missing. Heterozygous translocation reduces crossing over in 2L to about 3.4% between al and dp, 2.3% between dp and b, and 0.2% between b and pr; crossing over is also reduced in 3L to about 0.3% between th and st, 0.4% between st and cp, 0.2% between cp and in, and 0.02% between in and ri. However, crossing over is normal or slightly increased in the proximal part of 3R. A study of the linkage relations in translocation homozygotes gave the following crossing-over intervals: th-st, 1.4 ± 0.2; st-cp, 3.1 ± 1.0; st-in, 12.9 ± 0.4; in-ri, 1.0 ± 0.2.

Report of G. Ribó

ca: caramel 2 - 71.2±. Spontaneous in the offspring of a female inseminated in the wild (Prat de Llobregat, Barcelona). Eye colour on emergence brownish orange darkening with age, but lighter than sepia. Interaction with cn, giving an orange colour on emergence, darkening also with age. Not allele of sf and dke. Not tested the allelism with oo (Buzzati-Traverso, D.I.S. 20). RK1. Viability good.

ants: antenas 3 - 49.4±. Spontaneous in the offspring of a female inseminated in the wild (Prat de Llobregat, Barcelona). Malformation of the antennae which are longer or sometimes very reduced in males. Good expressivity in males but less in females. Viability good.

qf: quetas finas 3 - 60.7±. Spontaneous in the offspring of a female inseminated in the wild (Prat de Llobregat, Barcelona). Macro and microchaetae thinner and shorter. Low fertility. Viability good.

Report of H. J. Becker

rb^{66a}: $ruby^{66a}$ 1-7.5 X-ray induced in Berlin wild male. Fertility and viability good. w^a rb^{66a} have white eyes with slight yellow tinge.

vs^{66a}: $vesiculated^{66a}$ 1-16.3. X-ray induced in Berlin wild male. Fertility and viability good.

$Tp(1)f^{67a}$ X-ray induced in Berlin wild male. Phenotype forked; fully viable homo- and hemizygously. Four breaks in or near 12E, 15E, 18B and 20. New arrangement most probably 1-12E/18B-20/kinetochore/15E-12E/18B-15F/20. Occasionally both ends of right arm attached to chromocenter. Of nine free right arms in two cases the 15F end was attached to chromocenter; slight possibility, therefore, that right arm is arranged in reverse order. Mitotic stages showed no extra element. Crossover w-g 24.7%; g-f 0.1%.

Report of D. J. Komma

M^{Zw} (Modified glucose-6-phosphate dehydrogenase). X, near Zw^A-Zw^B locus. Komma, 1966. G6PD from females migrates abnormally fast in starch-gel electrophoresis, that from males abnormally slow. Male G6PD is slow because of binding to a "sex peptide". Dominant, i.e., both bands affected in double heterozygotes with Zw^A and Zw^B. Publ. with figures Biochem. Gen. 1 (3), December 1967.

Report of Ross MacIntyre

Acph-1^C - acid phosphatase-1^C MacIntyre, 1967. 3-101.3±. This is a codominant allele of Acph-1^A and Acph-1^B. The form of the acid phosphatase found in Acph-1^C/Acph-1^C homozygotes migrates to a position about 3.7 cm. from origin under the conditions specified by MacIntyre (DIS 41: 61). In zymograms, Acph-1^A/Acph-1^B or Acph-1^B/Acph-1^C heterozygotes show three band patterns which are characteristic of homomultimeric enzymes. Acph-1^C, discovered in stock 441 from Cal. Tech. is the third allele to be found at this locus (see MacIntyre, Genetics 53: 461). The order of the enzymes, in terms of increasing electropositive migration rates at pH 8.7 is AA, AB, AC, BB, BC, CC.

Report of S, J, O'Brien and R, J, MacIntyre

α GPDH-1^A: α-glycerophosphate dehydrogenase-1^A O'Brien, 1967.
α GPDH-1^B: α-glycerophosphate dehydrogenase-1^B O'Brien, 1967.
These are two codominant alleles which control the structure of electrophoretic variants of an NAD linked, soluble α-glycerophosphate dehydrogenase. The buffer system of Wright (Genetics 47: 787-801, 1963) for starch gels and the staining procedure of Sims (Nature 207: 757-758, 1965) were employed. A voltage drop of 6 v/cm for four hours at 4°C is sufficient to separate the variants. Homozygous αGPDH-1^A produce a single anodal band 3.7 cm from the origin. Individuals homozygous for the α GPDH-1^B allele produce a single slower band 3.0 cm from the origin. α GPDH-1^A/α GPDH-1^B heterozygotes produce both parental bands plus a more intensely staining intermediate or hybrid enzyme. α GPDH-1^B was found in polymorphic populations from Cores, New York, Pleasant, Ohio, and Oxford, North Carolina. All other stocks tested were monomorphic for α GPDH-1^{AA}. The structural locus for α GPDH-1 has been identified and mapped to 20.5 ± 0.75 on Chromosome II.

Report of R. Valencia

Ch^V: Chubby of Valencia. 1964. Dominant mutation induced by X-rays in a chromosome 3 of normal order bearing red and stubbloid. Locus not yet determined, but seems to be closely linked to red sbd in 3R. Phenotype of heterozygote and homozygote apparently identical and very similar to that described for chubby in chromosome 2. Larvae, pupae and adults short and thickset. Especially easy to distinguish in larvae and pupae. Homozygous females and males viable and fertile. Salivary chromosomes normal.

Report of G. Maroni

Clv^3: $Cloven^3$. Maroni, 1967. Induced by X-rays in a Binsc X-chromosome. Thorax has a longitudinal dorsal cleft. Phenotype constant with full penetrance. Fully dominant, semi-lethal and sterile in males. When homozygous with a similarly inverted chromosome, crossing-over is suppressed between v and w. Locus of Clv^3 is apparently in this region, possibly at the locus of clv-2 (42.0). Cytological observation by W. Kirschbaum shows a translocation X;2L with breakpoints at 11A7/8 and 27E2/3.

Report of B. Nilsson and J. Valentin

vs^{66j} : vesiculated 66 j Nilsson 66 j 25. From offspring of X-rayed Canton-S male (3600 r) mated to Muller-5 female. Proven to be allelic with original vs mutant. The degree of expression at 25°C was 93.0% (n=687).

Report of William Biggin

ey^{67b}: eyeless-67b. Arose spontaneously in an Oregon-R stock which had been inbred by brother-sister matings for 98 generations. First observed in an outcross with y w females and isolated by Lesnik from F98 Oregon-R. Penetrance and expressivity variable. After five generations of selection penetrance is 98% with 80% of flies showing extreme eye reduction. Penetrance reduced to 15-20% at 17°C. Allelism shown with ey^2 and ey^4. RK2

Report of B. Burnet

$l(2)gl^B$. A new spontaneous mutation for lethal giant larva. The lethal was isolated during a selection experiment carried out on thiamine deficient aseptic synthetic medium, summer 1966. Third instar larvae are bloated and fail to pupate. Penetrance on live yeast medium is excellent. Allelism was established by crossing $l(2)gl^B$ heterozygotes to l(2)gl a px or/SM5, al^2 Cy lt^v sp^2 (obtained from M. Ashburner).

Report of Sister M. Joan Rowan

$(sc^{SJ})scute^{SJ}$: From X-rayed C.S. males. Complete absence of scutellar, postvertical, ocellar, anterior and middle orbital, and posterior postalar bristles. Reduction in sternopleurals. Homozygous lethal. Associated with inversion whose right break is in $1B_{3/4}$ and whose left break is in $2E_3$.

Report of J. Masterson

Dp (1;Y)1E: 1961. Found as 6 males of the phenotype y,sc^+. The distal portion of the X has inserted into a functional Y chromosome. These males are fully viable and fertile. The duplicated X now carries the wild type alleles for su-v, su-apr, ac and sc. The effect of this Y with the mutant deep-orange (Merrill) gives a variegated eye phenotype, indicating a variegated-type position effect of the dor locus. (For a description of the way in which this was synthesized see Research Notes.)

Dp (1;Y)2E: 1961. Derived from an irradiated X·Y chromosome and covering the lethal (1-64, Stern). This duplication is similar to the one described above except that it covers the deep-orange region. It is also believed to have a piece of the proximal end of the X. (For a description of the way in which this was synthesized see Research Notes.)

Report of G. D. Hanks and L. Newlin

bab: brown abdomen 1=1.3. The normally black portion of the male abdomen is dark brown. It was first found at a frequency of a few percent in a stock bottle. This mutant can only be detected with assurance in males and is of high viability and fertility. There is no overlap with wildtype at 25°C.

Report of Milton Pasternak and W. D. Kaplan

Hk^{1P}: Hyperkinetic-1 1-30.9 ± 0.6. From feeding of adult male with EMS. Following etherization legs vibrate rapidly. Non-etherized flies respond to hand moving above vial by falling about as if completely uncoordinated. Heterozygous females shake but to lesser degree than homozygote and lose response to moving object. Normal flies do not respond to movement. Fully viable and fertile, RKI.

Sh^{5P}: Shaker-5 1-58.2 ± 0.6. From feeding of adult male with EMS. Appears to be allele of Shaker first described by Catsch (Z. Vererb., 82: 64-66, 1944). Following etherization legs shake rapidly, also is marked scissoring of wings. Fully viable and fertile, RKI.

Report of W. E. Trout and W. D. Kaplan

Hk^{2T}: Hyperkinetic-2 1-30.4 ± 0.6. From feeding of adult male with EMS. Shows same behavior as Hk^{1P} with movement response less well-developed. Compound. Hk^{1P}/Hk^{2T} gives strong response to movement. As with Hk^{1P}, is fully viable and fertile, RKI.

Report of R. E. Rayle

sa: sparse arista Rayle, 67a9. 1-1.4. Spontaneous in y w spl chromosome. Probably allelic to the Fahmy's cramped-like and swollen-antenna mutants. Antennae swollen. Aristae with short shaft and fewer, finer branches. Wings slightly divergent. Small additional sex combs on second tarsal segments of the prothoracic legs and less frequently on the basitarsi of the mesothoracic legs in males. sa;Pc/+ males have large sex combs on all six basitarsi and small sex combs on all six second tarsal segments in many cases. sa flies usually lack one or both postverticals, with other bristles occasionally missing or doubled. Body pigmentation slightly darker than wild type. Both sexes usually sterile. Viability reduced and extremely sensitive to crowding.

sa^2: sparse arista-2 Green 67. Repeated spontaneous occurrences as premeiotic events in a y $w^{dc}sn^3$ stock independent of premeiotic changes at the white locus occurring in the same individuals. Identical to sa except for frequent incision of inner wing margins, which also occurs in sa/sa^2 females.

w^{gd}: white-garnetoid Rayle 64. Spontaneous as one male in a w f;vg;e^{11} stock. Located to the right of w^a and acts as a zeste - suppressor. w^{gd} is non-compensating, but this can be recognized only in the presence of an enhancer (see below) due to the dark eye phenotype, inseparable from g^2, in non-enhanced individuals.

en-w^{gd}: enhancer of white-garnetoid Rayle 66. 1-(near centromere). Present in a stock of a cinnabar allele derived from a wild Urbana, Illinois population. Has only slight effect on w^{gd} females, but changes eye color of w^{gd} males to a light brown at 25°C. Enhancing effect reduced at 18°C and vanishes at lower temperature, at which en-w^{gd} acts as a suppressor of forked.

Report of G. Lefevre

In(1)w^{mB}: inversion white-mottled-B Barnes, 65a7. A radiation-induced X chromosome inversion, with the left breakpoint between 3C2 and 3C3 and the right breakpoint in the proximal heterochromatin to the left of bb. Males and homozygous females exhibit typical white variegation, resembling that of w^{m4}. In XO w^{mB} males, variegation for rst and vt (vertical) is also expressed.

In(1)w^{-64d}:inversion white-deficiency 64d Lefevre, 64d8. A radiation-induced X chromosome inversion, accompanied by a short deficiency, with the left breakpoint immediately following 3B1-2 and the right breakpoint in the proximal heterochromatin to the left of bb. The region from 3B3 to 3C2 (possibly 3C3), inclusive, is deleted. When covered by Dp w^{Vco}, w^{-64d} males express variegation for rst, as well as for w.

Dp(1;4)w^{m65g}: duplication white-mottled-65g Lefevre, 65g7. Following irradiation, a short section of X chromosome containing the w locus has been inserted in the heterochromatic region of the fourth chromosome. The probable extent of the inserted segment, as determined genetically, is not greater than from 3B2 through 3C4. The associated deficiency was lost before cytological analysis could be completed, and the short extent of the duplication, coupled with its chromocentral location, has made it impossible to determine the extent of the duplication cytologically. It covers the deficiency of w^{m4L}-rst^{3R} and w^{-64d} (see above), the latter covered males being poorly viable and expressing bristle abnormalities like those described by Gersh for deficiency of 3C1.

Df(1)w^{-64b};Dp(1:2L)w^{+64b}: Deficiency white-64b; Duplication white+64b. Lefevre, 64b13. Following irradiation of In(1)w^{m4}, a piece of X chromosome extending from 3C2 (accompanied by an appreciable amount of proximal heterochromatin) through 5A1-2 was deleted; the deleted fragment, broken into 2 pieces at 4E3, was inserted in 2L at 26D7 in the following order: 2L tip • 4E1-2 -- 3C2 (heterochromatin) 5A1-2 -- 4E3 • 2L base. Males containing both the deleted X chromosome and the inserted (rearranged) fragment are phenotypically normal, viable, and fertile. In the XO condition, Df-Dp males survive, but exhibit white variegation similar to that of XY w^{m4} males. Females heterozygous for the deficiency (without the duplication) are poorly viable, express Notch, and may produce a few offspring. This deficiency, 88 bands in length, is the longest (in the neighborhood of the w locus) so far identified that survives in the heterozygous condition. Males hyperploid for the duplication do not survive, but hyperploid females are viable and fertile.

Dp(1;2L)$(w\text{-}ec)^{64d}$: Duplication of white through echinus-64d. Stafford, 64d20. (Publ. Genetics 53:175-187) Corrected description: following irradiation of wild-type males a piece of X chromosome extending from 3C2 to 3F1-2, inclusive, was inserted in region 37D of 2L. The duplication should contain both the w^+ and ec^+ loci, but the duplication produces no change of phenotype when added to w ec males or females. Further, neither the duplication nor its associated X chromosome deficiency complements with sp-w. Nonetheless, z; Dp$(w\text{-}ec)^{64d}$ males show a mottled phenotype suggestive of duplication of the w^+ locus. Thus, the duplication contains a white locus in such a condition (or position) that it is unable to complement with sp-w. In addition, spl/spl; Dp$(w\text{-}ec)^{64d}$ females express split, as though the spl^+ locus in the duplication is not fully dominant; however, spl: Dp$(w\text{-}ec)^{64d}$ males approach wild type in phenotype, having only slightly roughened eyes.

Dp$(w\text{-}ec)^{64d}$ will cover the lethality of $w^{258\text{-}21}$, although Dp w^{+51b7}(3C2-3D6) will not, indicating that the lethality of $w^{258\text{-}21}$ is associated with its translocation breakpoint, which Dp$(w\text{-}ec)^{64d}$ covers, not with a position effect on the N^+ locus, which Dp w^{+51b7} covers. Further, Dp$(w\text{-}ec)^{64d}$ covers N^8, though poorly, which is quite unexpected in view of the published extent of N^8.

(In our laboratory, Dp$(w\text{-}ec)^{64d}$ is commonly referred to as Stafford-Notch, and may have been so designated in correspondence.)

Report of John Erickson

y v •bw^+, X•Y^L Arose spontaneously in stock Oregon N-5, y v/ bw^+Y; bw. Cytologically, it appears as an X chromosome with a prominent arm (Y^L) beyond the centromere. Tested with Brosseau's stock L-4, which carries a Y lacking all fertility factors of the long arm, the bw^+ X•Y^L was shown to possess these factors. Stock is in the following form:

y v•bw^+/ B^SY; bw/bw ♂♂ & XX, y f / B^SY; bw/bw ♀♀

Report of A. Schalet

From X-rayed y ct^{6} f•Dp(sc^{V1}y^{+}) male crossed to y^{2} v f car females.

1(1)5: lethal 5 Schalet, 1961. To the right of car; to the left of 134 and not covered by y^{+}Yma-1^{+}.

1(1)20: lethal 20 Schalet, 1961. Between 1114 of Novitski and 122 of Schalet. Allelic to 197 and 1137 of Novitski. 1 crossover in 3,507 between lethal 20 and su-f in experiments which gave 5.4% crossing over between car and su-f and 0.06% between su-f and Dp(sc^{V1}y^{+}).

1(1)22: lethal 22 Schalet, 1961. Between 120 and su-f. Deficient for su-f (see my research note this issue). Viable and normal with 120 and bb^{13a}. Viable with y^{+}YB^{S} and Dp(1;f)3. Lethal with Dp(1;f)52 which covers su-f. Lost. See Df(1)y^{x15} below.

1(1)34: lethal 34 Schalet, 1961. To the right of M(1)n; to the left of ot. Most distal locus of proximal X covered by y^{+}Yma-1^{+}.

Females from X-rayed y^{+}Yma-1^{+}/sc^{8} B male crossed to y sc^{S1} ma-1^{F2} sc^{8} female.

Df(1)y^{x2} Deficient for 1J1, y, ac and part of bb locus. Males normal, viable and fertile with sc^{8}Y.

Df(1)y^{x7} Deficient for 1J1, y, ac, bb, su-f, 120. Viable with 1114. Viable with sc^{8}YB^{S}, y^{+}Yma-1^{+}.

Df(1)y^{x9} Deficient for 1J1, y, ac, bb, su-f, 120, 1114. Lethal with sc^{8}YB^{S}, viable with y^{+}Yma-1^{+}.

Df(1)y^{x15} Deficient 1J1, y, ac, bb, su-f. Viable with 120, sc^{8}YB^{S}, y^{+}Yma-1^{+}. Lethal with sc^{8}Y. Lethality with Dp52 which covers 1J1, y, ac, bb and su-f suggests the absence of a lethal locus between 120 and su-f. See 1(1)22 above.

Df(1)y^{x16} Deficient for 1J1, y, ac, bb, su-f, 120, 1114. Viable with 1DCB2-19. Lethal with sc^{8}YB^{S}, viable with y^{+}Yma-1^{+}.

Report of John R. Merriam

FM7: first multiple seven constitution: In(1)sc^{8} + 15D-E; 20A-E + dl 49, y^{31d} sc^{8} w^{a} v^{Off} B.

synthesis: recovered as f^{+} crossover from FM6/In(1)sc^{8} + dl 49, y^{31d} sc^{8} w^{a} v^{Off} f B females.

cytology: salivaries of females heterozygous with In(1)sc^{8} + dl 49 show complete synapsis, dl 49 region homozygous, and inversion loop from 15D-E to chromocenter.

genetics: FM7/In(1)sc^{8} + dl 49, y^{31d} females have apparently normal crossing-over in w^{a}-B interval.

properties: male viable and fertile; homozygous female should be viable and fertile. Should effectively eliminate X recombination in FM7/+ females that are also heterozygous for In(2LR)SM1 and In(3LR)Ubx^{130}, since FM7 includes the best crossover-suppressing properties of FM1 and FM6.

Report of P. T. Ives

net^{38j} b^{38j} cn^{38j} bw^{38j} Ives, 38j. These mutants, which resemble their standard mutant alleles in separate phenotypes, were put into this combination in 1942 after having been found several times each in chromosomes extracted from the natural population in South Amherst during 1938-1941. Not until 1953 was it observed that the yellowish eye color in this combination differs strikingly from standard cn bw which is white. In this respect the effect of bw^{38j} is like that of bw^{37g} (Clancy, DIS 10:55). Recently C. W. Clancy chromatographed cn^{38j} bw^{38j} and found it produces most if not all of the pteridine components of wild type, which is not true in cn bw. This stock has been sent to several laboratories over the years without any superscripts on the symbols. In view of Clancy's observation it seems desirable now to differentiate these alleles in this manner. Since bw^{37g} was not distributed and was lost some 20 years ago any laboratory stock of net b cn bw with a yellowish eye color is probably this 38j group. The stock breeds well and is a useful alternative to al b c sp. An additional observation of interest is a lighter yellowish color in cn^{38j} bw^{38j}/cn bw, suggesting a simple dosage effect of bw^{38j} when heterozygous with other bw mutants.

Report of John H. Williamson

y^{67j} - In $B^{S}Y y^{67j}$ from EMS-treated $Y^{S}y$ cv v f/$B^{S}Y y^{+}$ males. $y/B^{S}Y y^{67j}$ males have yellow bodies and dark bristles like y^{2}/Y males. The usefulness of this Y chromosome is that now y^{67j} or even ac^{+} or $1(1)J1^{+}$ can be used to mark Y-short and y^{+} can be used in some other capacity, for example, on the left arm of the fourth chromosome (see Parker, this issue).

Report of A. Schalet and V. Finnerty

The list below includes previously undescribed or only sketchily mentioned male viable and male lethal mutants of maroon-like. All ma-l lethals have proved to be lethal or mutant with at least two adjacent loci. (See our research note in this issue for a map of the proximal X). We assume ma-l lethals are deficiencies and have listed the genetically determined limits of each. If reference to our map shows a locus within the limits of a breakage point for a particular deficiency, then that locus has not been tested with the deficiency. All ma-l lethals are viable and fertile as males with $y^{+}Yma\text{-}l^{+}$ and/or $y^{+}Yma\text{-}l^{106}$ unless otherwise noted.

ma-1: maroon-like 1-64.8

Females from X-rayed v B^{M1} male crossed to y v $ma\text{-}l^{2}$ female.

Df(1)$ma\text{-}l^{3}$ Left break between 134 and sw; right break between su-f and bb.
" 4 Left break between 134 and sw; right break between 1A7 and 1DCB1-35c.
" 5 Left break between 134 and sw; right break between 1t2-14a and 1LV7.
" 6 Left break between mel and ma-1; right break between 1A7 and 1DCB1-35c.
" 7 Left break between 134 and sw; right break between 1gluful-2, DCA3-19 and 1A7.
" 8 Left break between 134 and ot; right break between 1N5 and 1152.

Females from X-rayed $y^{+}Yma\text{-}l^{+}/sc^{8}$ B male crossed to y $sc^{S1}ma\text{-}l^{F2}$ sc^{8} female.

Df(1)$ma\text{-}l^{9}$ Left break between 134 and ot; right break between su-f and bb.
" 10 Left break between ot and sw; right break between ma-1 and 1152.
" 11 Left break between 134 and sw; right break ma-1 and 1152.
" 12 Left break between 134 and ot; right break in bb.
" 13 Left break between mel and ma-1; right break between su-f and bb.

$ma\text{-}l^{14}$ Male viable. - $ma\text{-}l^{14}$/Y and $ma\text{-}l^{14}/y^{+}Yma\text{-}l^{106}$ males are sterile. Sterility removed by replacement of region to the left of ma-1 (to the right in normal order). $ma\text{-}l^{14}/ma\text{-}l^{6}$; $ma\text{-}l^{14}/ma\text{-}l^{13}$ females show an additional wing, thoracic and abdominal phenotype similar to that of the mutant mel which lies to the left of ma-1. However, $ma\text{-}l^{6}$ and $ma\text{-}l^{13}$ are normal over mel. Few $ma\text{-}l^{14}$/0 males examined give ambiguous phenotype.

Df(1)$ma\text{-}l^{17}$ Female from X-rayed y sc^{S1} sc^{8} female crossed to sc^{8} Tu w^{a} male. Picked up as a lethal between y and Tu. Left break between 134 and ot; right break between ma-1 and 1N5. Deficiency at left tip, including 1J1 y ac and part of bb locus probably induced simultaneously.

Females from TEM treated $y^{+}Yma\text{-}l^{+}/sc^{8}$ f male crossed to y sc^{S1} $ma\text{-}l^{2}$ 1t2-4a f sc^{8}/y In49 sn^{x2} B^{M1} Df(1)$ma\text{-}l^{6}$ female.

$ma\text{-}l^{20}$ Y/$ma\text{-}l^{20}$ males viable and fertile.
21 Y/$ma\text{-}l^{21}$ males viable and fertile.

Female from TEM treated $y^{+}Yma\text{-}l^{+}/sc^{8}$ f male crossed to y sc^{S1} $ma\text{-}l^{2}$ 1t2-4a f sc^{8}/y In49 sn^{x2} B^{M1} Df(1)$ma\text{-}l^{6}$ female.

$ma\text{-}l^{23}$ Y/$ma\text{-}l^{23}$ males viable and fertile.

Female from EMS treated $y^{+}Yma\text{-}l^{+}/sc^{8}$ f male crossed to y sc^{S1} $ma\text{-}l^{2}$ 1t2-4a f sc^{8}/y In49 sn^{x2} B^{M1} Df(1)$ma\text{-}l^{6}$ female.

$ma\text{-}l^{24}$ Y/$ma\text{-}l^{24}$ males viable and fertile.

Female from EMS treated Y/sc^{8} f v cv male crossed to y sc^{S1} $ma\text{-}l^{F2}$ 1t2-4a f sc^{8}/y In49 sn^{x2} B^{M1} Df(1)$ma\text{-}l^{6}$ female.

$ma\text{-}l^{25}$ Y/$ma\text{-}l^{25}$ males viable and fertile.

Female from TEM treated Y/sc^8 f v cv male crossed to y sc^{S1} $ma\text{-}1^{F2}$ lt2-4a f sc^8/y In49 sn^{x2} B^{M1} Df(1)$ma\text{-}1^6$ female.

$ma\text{-}1^{26}$ Y/$ma\text{-}1^{26}$ males viable and fertile.
27 Y/$ma\text{-}1^{27}$ males viable and fertile.
28 Y/$ma\text{-}1^{28}$ males viable and sterile.
29 Y/$ma\text{-}1^{29}$ males viable and fertile.

Male from EMS treated y^+Yv^+/y^{S1} sc^8 male crossed to Y/y v f ma-l xx female.
$ma\text{-}1^{30}$

Males from X-ray treated $y^+Yma\text{-}1^+$/y ct^6 f 120•Dp($sc^{V1}y^+$) male crossed to y v $ma\text{-}1^2$ females.

$y^+Yma\text{-}1^{102}$ Deficiency of $ma\text{-}1^+$ segment. Left break to the left of 134; right break to the left of 1N5.
" 106 Mutation of ma-l which doesn't complement any X chromosome ma-l alleles and covers all lethals covered by $y^+Yma\text{-}1^+$.
" 113 Deficiency of $ma\text{-}1^+$ segment. Left break between 134 and sw; right break between lt2-14a and 1LV7.

Males from X-rayed $y^+Yma\text{-}1^+$/sc^8 B male crossed to y sc^{S1} $ma\text{-}1^{F2}$ sc^8 female.

$y^+Yma\text{-}1^{116}$ Mutation of ma-l which complements some X chromosome ma-l alleles and covers a number of loci on either side of ma-l including closest known loci.
" 118 Deficiency of $ma\text{-}1^+$ segment. Left break to the right of mel; right break to the right of su-f.
" 126 Deficiency of $ma\text{-}1^+$ segment. Left break to the left of sw; right break between lgluful-2 and 1A7.

Report of Ray Perez and W. D. Kaplan

wgo: wing-out 1-16.2 ± 0.6. From injection of H^3TdR into adult male. Wings divergent, slight thickening of veins, notching effect in only 10-15% of flies. Not allelic with vesiculated, RKI.

LINKAGE DATA

Report of W. W. Doane

Amylase (Amy): symbols, cytogenetics. Observations were made in support of Bahn's (DIS 42: 41, 84) view that Amy 'alleles' characterized by more than one major amylase band upon electrophoresis represent closely linked gene duplications (Doane, research note, this issue). He proposed that Amy symbols be changed to account for this; thus, $Amy^{2,6}$ would be designated Amy^2 Amy^6, etc. This is no doubt a suitable solution, but a simpler one would be to merely replace the period with a comma in the symbols already in use, until more is known about the fine structure of the region. Then we would have $Amy^{2,6}$, and other strains would become $Amy^{1,2}$, $Amy^{1,3}$, etc.

A linkage experiment was performed to estimate the position of Amy on the salivary chromosome map. The following cross was made: w; c $Amy^{4,6}$ adp^{60} ♀ x y w^a N^{62G1}/Y; Dp(1:2R)w^{+51b7} Amy^1/Amy^1 ♂. F_1 females were testcrossed to males from the parental female strain. A crossover analysis was done on the progeny with the duplication (detected as red eyes) used as a marker. It is inserted at band 52 F (Ratty, 1954). Amy genotypes were determined electrophoretically. The data indicates that Amy lies to the right of band 52 F. (Supported by N.S.F. grant GB 1718.)

MADISON, WISCONSIN: UNIVERSITY OF WISCONSIN
Department of Anatomy

D. ananassae
D. canapalpa
D. castanea
D. fulvimacula
D. fulvimaculoides
D. giberossa
D. melanopala
D. mercatorum mercatorum
D. mercatorum pararepleta
D. paranaensis
D. peninsularis
D. repleta
D. saltans
D. simulans
D. tumiditarsus
D. willistoni

LEXINGTON, KENTUCKY: UNIVERSITY OF KENTUCKY
Department of Zoology

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

AMHERST, MASSACHUSETTS: AMHERST COLLEGE

D. affinis Amherst 1964
D. algonquin Amherst 1966
D. buskii Amherst 1967
D. funebris Amherst 1955
D. hydei Amherst 1956
D. immigrans Amherst 1966
D. melanica Amherst 1952
D. persimilis ? Vancouver 1961
D. repleta Philadelphia 1966
D. robusta Amherst 1960
D. simulans Amherst 1961
D. similans Tennessee 1966
D. virilis ? Amherst 1967

St. LOUIS, MISSOURI: WASHINGTON UNIVERSITY
Department of Biology

D. melanica: St. Louis, Mo.
D. euronotus: St. Louis, Mo.
D. micromelanica: Cave Creek, Ariz.
Florida
D. melanura: St. Louis, Mo.
D. paramelanica: St. Louis, Mo.
D. nigromelanica: Terra Haute, Ind.
D. pengi: Japan
D. colorata: Lake Itasca, Minn.
Petosky, Mich.
D. sordidula: Sugadaira, Japan
Hokkaido, Japan
D. lacertosa: Hizashiyama, Honshu, Japan
Nopporo, Ebetsu, Japan
D. robusta: New York
D. pseudosordidula: Nopporo Forest, Japan
D. immigrans: Hawaii
D. pararubida: Marshall Atoll
D. virilis: Texmelucan
D. funebris: Minneapolis, Minn
D. macrospina: St. Louis, Mo.
Mallen, Ga.
D. subfunebris: Willow Creek, California
D. carsoni: Vermont
D. polychaeta
D. gibberosa: Mexico
D. multispina: Sapporo, Hokkaido, Japan
D. daruma: Iriomote & Ryuka Islands, Japan

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

D. affinis: Florida, Minnesota, Nebraska, Ontario
D. algonquin: Minnesota, Ontario, Vermont
D. athabasca: British Columbia, Colorado, Massachusetts, Minnesota, New Mexico, Ontario, Oregon, Pennsylvania, Vermont, Washington.
D. azteca: Arizona, California, Distrito Federal (Mexico)
D. narragansett: Nebraska
D. tolteca: Bolivia, Colombia

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. (6 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. funebris

Poughkeepsie, N. Y. (2 strains)

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. (2 strains)

D. melanogaster

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

NEW YORK, NEW YORK: THE ROCKEFELLER UNIVERSITY

-new strains in addition to ones previously listed in DIS.

D. tropicalis

1. Santa Marta, Colombia
2. Cordoba, Colombia
3. Piojo, Colombia
4. Turbo, Colombia
5. Grand Cayman

D. willistoni

1. Santa Marta, Colombia
2. Piojo, Colombia
3. Turbo, Colombia
4. Grand Cayman

D. equinoxialis

1. Cordoba, Colombia
2. Piojo, Colombia
3. Turbo, Colombia
4. Grand Cayman

D. paulistorum

1. Santa Marta, Colombia
2. Piojo, Colombia
3. Turbo, Colombia
4. Choco, Colombia

COLD SPRING HARBOR, NEW YORK: CARNEGIE INSTITUTION OF WASHINGTON
Genetics Research Unit

D. buskii: Wild-type B

D. virilis: China-a

EAST LANSING, MICHIGAN: MICHIGAN STATE UNIVERSITY
Department of Zoology

D. funebris (Yale)
D. immigrans (East Lansing, 1964)
D. melanica (East Lansing, 1964)
D. pseudoobscura (Rochester)
D. pseudoobscura (Yale)
D. virilis (California)
D. virilis (Yale)

CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGO
Department of Zoology

D. americana

Wild Stocks

1 Independance
2 Anderson

D. texana

3 New Orleans

D. virilis

Wild Stocks

4 Pasadena lethal-free
5 Texmelucan

Chromosome 1

6 ap^{40e}
7 w^{50112}

Chromosome 2

8 b bk dt
9 va

Chromosome 3

10 gp^2
11 S/+
12 sv t tb gp^2

Chromosome 4

13 cd

Chromosome 5

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

Multichromosomal

22 b bk dt; t tb gp^2
23 b bk dt; t tb gp^2; pe
24 b; cn; pe
25 b; cn; B^3 pe
26 b; pe^{Jap}
27 b; tb gp^2; cd; pe
28 b; sv t tb gp^2; cd; pe
29 b; sv t tb gp^2; pe
30 cd; pe
31 cn; pe
32 gp; pe
33 gp^2 S/gp^2 +; ru st mh
34 pe; gl
35 "scute"(II); pe^{m3}
36 t; cd
37 v^{48a}; pe
38 v^{48a} w; pe
39 v^{48b}; pe
40 w; pe
41 y; tb; px; st
42 y^{40a}; pe

BUFFALO, NEW YORK: STATE UNIVERSITY COLLEGE
Department of Biology

pseudoobscura

Chromosome 3

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

Zaprionus

multistriata
tuberculatus
vittiger

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

D. willistoni

Wild strains from: Florida, Perú, Cuba, Guatemala, Jamaica, Equador; Brazil: Serra do Navio (Amapá), Manaus and Tabatinga (Amazonas), Pôrto Velho (Guaporé) Belém (Pará), São Luiz (Maranhão), Maranguape (Ceará), Salvador, Cassarongongo and Pedras 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

w
w^h
w^e y sn ru (Inv)/lethal

Chromosome 2

S Hk abb bw (Inv)/lethal
S Hk abb bw (Inv)/cn Em ph
S Hk abb bw (Inv)/Em

Chromosome 3

pink (Inv)/lethal

D. paulistorum:

Wild strains from: Costa Rica, Boquete (Panamá), Caripe (Venezuela), Bucaramanga and Santa Marta (Colombia), Georgetown (British Guiana); Brazil: Tabatinga (Amazonas), Belém (Pará), Xingú (Mato Grosso), Brasília (D.F.), Pocos de Caldas (Minas Gerais), Florianópolis (Santa Catarina), São Pedro (Rio Grande do Sul).

D. equinoxialis: wild strain from: Tefé (Amazonas, Brazil)
D. cubana: wild strain from: Trinidad
D. tropicalis: wild strain from: Palma (Goiáz, Brazil)
D. pawlovskiana: wild strain from: Apoteri (British Guiana)
D. capricorni: wild strain from: Morro do Ferro (Minas Gerais, Brazil)
D. nebulosa: wild strains from: Tingo Maria and Lima (Perú); Brazil: Pedras de Una (Bahia), Posto Duque de Caxias (Santa Catarina), Eldorado (Rio Grande do Sul), Brasília (D.F.).
D. polymorpha: wild strains from: Brazil; Maranguape (Ceará), Morro do Ferro (Minas Gerais), Eldorado (Rio Grande do Sul).
D. prosaltans: wild strain from: Maranguape (Ceará, Brazil).
D. simulans: wild strains from Brazil: Maranguape (Ceará), Pascoal (Rio Grande do Sul).

BHAGALPUR, INDIA: BHAGALPUR UNIVERSITY
Post-Graduate Department of Zoology, Drosophila Laboratory

D. ananassae

Salivary gland chromosomes

Chromosome 2

ST^2/ST^2
AL/AL
GA/GA
ST^2/AL
ST^2/GA
AL/GA

Chromosome 3

ST^3/ST^3
DE/DE
ST^3/DE
ST^3/ET
DE/ET

CAMBRIDGE, ENGLAND: CAMBRIDGE UNIVERSITY
Department of Genetics

D. anannassae (Haren)
D. funebris
D. simulans (Berkely)
D. busckii (Cambridge)
D. hydei (Leiden)
D. subobscura (Cambridge)

SÃO PAULO, BRASIL: UNIVERSIDADE DE SÃO PAULO
Faculdade De Filosofia, Ciencias E Letras

D. willistoni

Wild

U.S.A. - 1 strain
Guatemala - " "
Equador - " "
Cuba - " "
Jamaica - " "
Brazil - 24 strains

Mutants

Chromosome 1

se
w^e Y sn ru
W

Chromosome 2

S Hk abb bw/lethal
S Hk abb bw/cn
Em/abb bw

Chromosome 3

Δ pink (Inv.)/lethal
ebony
Not located
orange
taxi

Other Species

wild

D. ananassae - Brazil (1 strain)
D. anstrosaltans - Brazil (1 strain)
D. imigrans - Brazil (1 strain)
D. montium - Brazil (1 strain)
D. neocardini - Brazil (3 strains)
D. palidipenis - Brazil (1 strain)
D. prosaltans - Brazil (1 strain)
D. saltans - San Salvador, El Salvador (1 strain)
D. septentriosaltans - Sevilla, Colombia (1 strain)
D. sturtevanti - Brazil (1 strain)

HELSINKI, FINLAND: UNIVERSITY OF HELSINKI
Department of Genetics

bifasciata (1 strain)
busckii (1 strain)
funebris (11 strains)
hydei (1 strain)
obscura (5 strains)
subobscura (7 strains)

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

TURKU, FINLAND: UNIVERSITY OF TURKU
Department of Genetics

D. simulans

Wild

Chromosome 1

v
y w

Chromosome 2

net

Chromosome 3

H^h pe
jv se st pe
st pe

SAPPORO, JAPAN: HOKKAIDO UNIVERSITY
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)

TOKYO, JAPAN: TOKYO METROPOLITAN UNIVERSITY
Department of Biology

D. ananassae

Wild Stock

001 Texas
002 TL_3
003 Barro Collorado, Panama 69 (low elevation)
004 Turrialba, Costa Rica 101 (high elevation)
005 Christobal, Panama
006 Baton Rouge, Louisiana
007 $2L\text{-}A^H$
008 $2L\text{-}B^H$
009 Hawaii
010 D-rar
011 D-pp
012 L-pp
013 Cuba
014 Hawaii (Wh)
015 Niue
016 Panama
017 Tonga
018 Yukatan
019 IM-1
020 IM-2
021 IM-4
022 IM-5

Chromosome 1

101 $y^{51}f^{49}$
102 y (Hinton)
103 w^{65}
104 $w^{65}f^{49}$
105 $w^{65}y^{51}$
106 $w^{65}f^{49}y^{51}$
107 $w^{65}y^{51}sn^{65}$
108 kk

Chromosome 2

201 Arc
202 b
203 b^{65}
204 ba^{65}
205 bw^R
206 j
207 ma (Hinton)
208 se^T
209 Arc b
210 Arc bw^R
211 Arc ma
212 Arc se^T
213 b ma_T
214 b se^T
215 bw^Rba^{65}
216 cd ba^{65}-1
217 cd ba^{65}-7
218 cd ba^{65}-12
219 cd bw^R
220 cd se^T
221 cd se^Tba^{65}
222 j b
223 j b se^T
224 ma ba^{65}
225 se^Tba^{65}-1
226 se^Tba^{65}-2
227 se^Tba^{65}-6
228 se^Tba^{65}-11
229 se^Tba^{65}-12
230 se^Tba^{65}-13

Chromosome 3

301 bri (Hinton)
302 M^{65}
303 px
304 px^2
305 Rf
306 ru
307 ru^2
308 sm^{66}
309 bri px
310 bri Rf
311 bri Rf px
312 bri ru
313 Rf px
314 M^{65} px
315 M^{65}bri px
316 M^{65} px E^+
317 M^{65} px E^-

Multichromosomal

501 f; cd
502 f;cd (Hinton)
503 f; cd se^T
504 f y^{66}; 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}; $_T$px
531 b se^T; px^2
532 ba^{65}; M^{65}
533 bw^R; bri
534 bw^R; ru
535 j b se^T; ru
536 ma; bri
537 se^T; ru

Undetermined

701 pxd^{50}
702 pxd (Hinton)
703 Bn (Hinton)
704 ms^{65}
705 sk^{66}
706 ab-a

CALCUTTA, INDIA: UNIVERSITY OF CALCUTTA
Department of Zoology

D. ananassae

Wild Stocks

1 a66 Calcutta (a6)
2 a77 Behala (a7)
3 a88 Kolaghat (a8)
4 a99 College Street
5 a111 Dakshineswar
6 a222 Port Blair, Andaman

Chromosome 2

7 bw
8 bw vs ss^a

Chromosome 3

9 pc

Multichromosomal

10 se px pc fu

Special Selected Strains with Crossing Over in Males

11 px pc (6a)
12 px pc (3)

Other Species

D. Kikkawai (2 strains)

13 Brazil
14 Bishnupur

D. bipectinata
D. hydei
D. simulans

N.B. Numbers within the parenthesis denote our old Stock Nos.

BARQUISIMETO, VENEZUELA: CENTRO EXPERIMENTAL DE ESTUDIOS SUPERIORES
Departamento de Biologia

latifasciaeformis
ananassae
capricorni
willistoni & sibling species
mesophragmatica, Colombia
pavani, Chile

simulans
bandeirantorum
hydei
immigrans
gaucha, Brazil
viracochi, Columbia

griseolineata
viracochi
gasici
brncici, Colombia

READING, ENGLAND: THE UNIVERSITY OF READING
Department of Zoology

D. simulans

yw

VARANASI, INDIA: BANATAS HINDU UNIVERSITY
Department of Zoology

Wild Stocks

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

Mutants of D. ananassae

Chromosome 1

y
w^a
vs

Chromosome 2

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

Chromosome 3

px pc
stw pc
stw px
stw
px
pc

Unlocated mutants

dct
sp
ci
arch

LONDON, ENGLAND: UNIVERSITY COLLEGE LONDON
Department of Zoology

D. subobscura

Inbred lines

B
K
O
NFS

Fifth Chromosome

gs

D. simulans

Wild Stocks

Edinburgh
Galilee

SEOUL, KOREA: SEOUL NATIONAL UNIVERSITY
Department of Zoology

D. angularis	2 strains
D. unispina	3 strains
D. auraria Race A	8 strains
D. auraria Race B	2 strains
D. auraria Race C	3 strains
D. nigromaculata	4 strains
D. lacertosa	2 strains
D. sordidula	4 strains
D. virilis	6 strains

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)

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

Wild Stocks

D. americana
D. subobscura (Belgium)
D. virilis

D. subobscura (Küssnacht)
(Standard Homozygous)

STOCKHOLM, SWEDEN: UNIVERSITY OF STOCKHOLM
Institute of Genetics

D. pseudoobscura

ST	1 line
SC	1 line
TL	1 line

AR	2 lines
CH	1 line
OA	1 line

(all phenot. wild)

BRIGHTON, SUSSEX, ENGLAND: UNIVERSITY OF SUSSEX
School of Biology

D. simulans

Wild Stocks

1. AG
Jer. X

D. subobscura

Wild stocks

B (inbred)
K (inbred)

ROMA, ITALY: CITTA' UNIVERSITARIA
Istituto di Genetica

D. funebris
D. immigrans

D. simulans
D. subobscura

MANCHESTER, ENGLAND: PATERSON LABORATORIES
Department of Cytogenetics

D. ananassae

Non Cross-over males
Cross-over males

UMEA, SWEDEN: UNIVERSITY OF UMEÅ
Department of Genetics

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

DROSOPHILA SPECIES

NEW MUTANTS

subobscura

Report of H. Burla

1) Mutant stocks. Stocks with asterisk are described below. The superscript ^{Eu} stands for Eugene, Oregon, where these mutants were isolated from stocks 1 and 2, after treatment with EMS. The superscript is used only with mutants that appear to be allelomorphic to mutants obtained by former workers. Allelism has not yet been tested.

chromosome A
7: y
9: Oc
14: Bx
23: Bx^{Eu}
37: ct^{Eu}
55: $ct^{fr\ Eu}$
25: dw^{Eu}
31: w^{Eu}
10: pm ct sn
12: m ct v bnt sc

chromosome I
52: mj^{Eu}
69: ni
65: th ma
66: th ma mop
67: int ey wt
68: ma int mj
64: s th int ey mj
63: s th int ey wt

chromosome E
13: ho
80: dp^{1}
*53: $dp^{2\ Eu}$
41: $dp^{tr\ Eu}$
*32: oi
*47: dck
*56: qm
11: pl pp pt
8: sj pl pp otp

chromosome U
*36: ltr
*34: abb
70: nt
99: nt
71: fd^{Mi} nt

chromosome O
*27: cdc
74: Va
72: ch cu
73: ch ar
75: ch/Ba st
35: rn^{Eu}

unplaced
*19: shsc
*20: shv

Wild type stocks.

78: Galilaea
79: Edinborough

3) Homokaryotypic stocks (Chromosomes not mentioned are St/St, chromosomes mentioned are homozygous for the respective gene arrangement.)

1: Küsnacht
2: Zürich: U_{1+2}
3: Zürich: U_{1+2}
4: Zürich: I_1
17: Zürich: I_1
5: Tunis: I_1, E_{1+2}, U_{1+2+8}, O_{3+4+8}
6: Tunis: A_{2+6}, I_1, $E_{1+2+9+4}$, U_{1+2+8}, O_{3+4+8}

4) Diagnosis of some of the mutants

dp^2 Eu, dumpy^2 Eu. Wings rather uniformly shortened to about half normal length. Vein pattern slightly different from dp^1. Compound dp^1/dp^2 Eu intermediate. Very closely linked with pt, but not allelomorphic with it. dp^2 Eu/ pt segregates phenotypically into three classes: dumpy, pointed and mosaics.

oi, oily. Body surface shining black as if wetted by oil, probably due to the fact that microtrichia are lacking almost completely. Branches of arista short. Wings delicate, whitish, bent downward, often torn distally and along posterior margin.

dck, dackel. Legs shortened, the character being most obvious in femora and tibiae which become relatively thick, the hind tibiae, in addition, being arched. Wings slightly shortened. Scutellum shortened, the scutellar bristles twisted and standing up. Body size slightly reduced.

qm, quasimodo. Face irregularly protruding, with warts and holes, the margins torn. Antenna with arista crumpled. Lower eye border furrowed in strongly expressed phenotypes. Expression asymmetric. Anal papillae, external copulatory apparatus as well as tergites similarly affected. Up to three additional spermathecae. In male, mid and hind legs with incomplete sex combs. Expressivity variable. Fertile.

ltr, light red eye. Eye colour cadmium red, lighter than in wild type. Difference in hue striking in newly hatched flies, becoming obsolete with aging.

abb, abbreviated. Macrochaetae on head and mesonotum minute, mostly about half normal length in females, longer in males.

cdc, close dorsocentrals. The anterior and posterior macrochaetae stand very close to each other, the distance at their bases measuring about 13.5 percent of the length of the scutum, compared to about 20 percent in wild type flies. Middle orbital bristle enlarged, measuring about 68 percent of the first orbital. One or both of these bristles may point outwards. Some flies carry an additional orbital bristle. In most of the flies, the orbital phenotype is present on one side only, or is expressed in different ways on the two sides.

shsc, short scutellars. One to four scutellar bristles reduced in size to half of length or less, and proportionally thin. Character present in about 90 percent of males and about 70 percent of females.

shv, short vein. Fourth longitudinal vein not reaching wing margin. Expression asymmetric and variable. In a few flies, second and/or fifth veins are similarly affected. Probably identical with longitudinale staccate (Buzzati), short-vein (Gordon) and vli (Prevosti).

5) Marker stocks of Drosophila subobscura, obtained by the courtesy of Dr. Maynard Smith and Mrs. Jean M. Trent, were analyzed cytologically in order to determine their karyotypic composition. From mass cultures, individual males were crossed with a virgin female of the homokaryotypic stock Küsnacht, whose gene arrangements are standard. From the offspring of every pair, 8 larvae were studied. The symbols for chromosomes and gene arrangements are those proposed by Kunze - Muehl and Sperlich.

stock	marker genes	markers in chromosome	number of males analyzed	stock homokaryotypic for A	I	E	U	O	stock heterokaryotypic for
9	Oc	A	20	St		St			$I_{St},I_1,U_{St},U_{1+2},O_{St},O_{3+4}$
14	Bx	A	16	St	St	St	St	St	
10	pm ct sn	A	20	St	St	St	St		O_{St},O_{3+4}
12	m ct v bnt sc	A	20	St	St	St		3+4	U_{St},U_{1+2}
65	th ma	I	20	2	St	St		St	U_{St},U_{1+2}
64	s th int ey mj	I	19	St	St	St			$U_{St},U_1,O_{St},O_{3+4}$
13	ho	E	20	St	St	St			$U_{1+2},U_{1+2+8},O_{St},O_{3+4},O_{3+4+6}$
11	pl pp pt	E	20	St	St	St		St	U_{St},U_{1+2}
8	sj pl pp otp	E	20		St	St		St	U_{St},U_{1+2}
71	fd^{Mi} nt	U	20	St			1+2		$I_{St},I_1,I_{3+4},E_{St},E_{1+2},O_{St},O_{3+4}$
72	ch cu	O	20	St		St	St	3+4	I_{St},I_1

The linkage group symbols used by the Haldane - Spurway group of workers correspond to the chromosome symbols given above, in the following way:

linkage group	chromosome
1	A
2	I
3	U
4	E
5	O

This key is identical to the key given by Hipsch in DIS 26, with the exception of chromosomes U and E, their assigned symbols being now exchanged crosswise. Sondhi (in DIS 31) already pointed to the correct reading.

affinis

Report of R. A. Voelker

X Chromosome (loci unknown)

bl: blistered Wings are blistered and/or strap-like, often extending from body at 45° lateral angle. Viability and fertility considerably reduced. X-ray induced.

br: bristle Bristles are fine and one-half to two-thirds wild type length in br/br females and br males. +/br females are intermediate between +/+ and br/+. Fully viable with fertility very slightly reduced. Perhaps an allele of bobbed (Sturtevant, 1940), though not the same.

Pctd: Polychaetoid Doubling or tripling of bristles at many bristle loci, but usually not affecting all such loci on the same fly. Occasionally overlaps wild type. Homozygous lethal. X-ray induced.

sn: singed The larger body bristles are thin, twisted and somewhat blunt at tips. Arose on same sequence as bristle. Both sexes fertile. X-ray induced.

Autosomal (Locus unknown)

cr: crumpled Wings crumpled and cupped down over abdomen. May overlap wild type. Viability and fertility good.

ananassae

Report of D. Moriwaki

(X-chromosome)

sn^{65}: singed-65 (sn*) Moriwaki 65i27. 1.110.0. Recovered as single ♂. Likely reoccurence of sn. Bristles twisted or gnarled.

w^{65}: white-65 (w*) Ebitani 65e14. 1-16.7. Spontaneous in 2A315 wild stock. Inseparable from w.

f49: forked-49 Moriwaki 49129. Appeared as single ♂ from a cross +T x +L. Allelic to f.

y^{51}: yellow-51 (y*) Moriwaki 51b19. Spontaneous 1 ♂ in HH wild stock. Reoccurrence of y.

y^{66}: yellow-66 Tobari, Y.N. 66h. 1-93.7. Spontaneous in f cd ba^{65} stock. Inseparable from y.

kk: kinky Moriwaki 67a11. 1-73±. Recovered as single ♂ in Delta(?) stock. Bristles slightly bent or forked. Gives wild type F_1 with f.

l-1; lethal-1 Moriwaki 67e. 1-33± (between w and f: locus tentative). Spontaneous in y f stock.

od: outstretched Ito, S. 67b. 1- . Appeared as single ♂ in BBBBG$_1$ wild stock. Wings very divergent, often at right angles to body, and slightly depressed. Mostly expressed in males.

f: forked (from CALTECH as f cd, 1950) 1-44.4

(2-chromosome)

Arc: Arc Moriwaki 66i23. 2-L (between In2L and SFA). Spontaneous 1 ♂ from a cross sm^{66} x b se. Wings bent downward. Crossveins absent or traces present. Dominant. Homo. viable, almost sterile.

b^{65}: black-65 Moriwaki 65h20. 2-L (included in In2LB). Spontaneous in HU13♂1 wild stock. Black body color. Allelic to b.

gr: grey Moriwaki 65h20. 2- (in In2LA?). Spontaneous in HU13♂45 wild stock. Less dark than b. Gives sometimes intermediate type F_1 with b.

ba^{65}: balloon-65 (ba*) Moriwaki 65h20. 2-R. Spontaneous in HU13♂34 wild stock. Wings warped with blisters. Different from ba (Moriwaki 33g 8 - 3 - cht.).

b: black (from India 1956). 2-L (in In2LB).

bw^{R}. brown-R (bw*) (from India as orange, 1956). 2-L (in In2LA). Brownish eye color. Ocelli colorless.

j: jaunty (from India as curly^2, 1956). 2-L (in or near In2LB).

se^{T}: sepia-T (se*) (from Texas as "brown eye color", 1950). 2-R. Eye color sepia. Ocelli remain wild type in color.

cd: cardinal (from CALTECH, 1950). 2-L (in In2LA. Previously 2-30.0, Moriwaki, 1940). Eye color yellowish vermilion Ocelli white.

ma: maroon (from Dr. Hinton's, 1966). 2-R (right half ?, left of se in 2R).

(3-chromosome)

M^{65}: Minute-65 (M^{*}) Moriwaki 65j21. 3-R. Recovered as single female in Hu13♂17 wild stock. Dominant. Homo. lethal.

px^{2}: plexus^{2} Moriwaki 66h23. 3-R. Spontaneous in IM-1 (Madras, India) wild stock. Allelic to px. More pronounced in females. Semidominant in some Minutes.

sm^{66}: small bristles-66 Moriwaki 66e12. 3- (locus not determined). Spontaneous in IM-1 wild stock. Bristles, not so extremely as, M.

Rf: Roof Moriwaki 66h3. 3-R? From X-ray treatment of wild-type $BBBBG_3$ male, as 1 female. Roof-shaped wing. Dominant, emerge as normal, (Rf) appears almost after 12 hours (Oguma & Mitamura, unpub.) The same as Rf of melanogaster.

ru: roughoid Moriwaki 49112. 3-R. Spontaneous in a cross +T x +L. Slight smudge of blackened facets in lower parts of eye. Expression varied by modifying genes (Ono, A., unpub.)

ru^{2}: roughoid^{2} (from Dr. Hinton's as "rough", 1966). Allelic to ru.

px: plexas (from India, 1956) 3-R.

bri: bright (from Dr. Hinton's, 1966). 3- (in In3LA?) Eye color bright red. Ocelli colorless.

*Symbol in parentheses will be used for convenience' sake, but for special need.

tropicalis

Report of J. I. Townsend

g: garnet Townsend 59d. Sex-linked recessive. Spontaneous in Cuban stock. Eye color on emergence more dilute than wild type, resembles Scarlet (Ridgway plate I), becoming darker with age to Brazil Red (Ridgway plate I). Viability excellent. Homology uncertain.

pch: peach Johnson 66a10. Sex-linked recessive. Spontaneous in Guatemalan stock. Eye color on emergence between Peach Red (Ridgway plate I) and Coral Red (Ridgway plate XIII), darkening with age to Dragon's blood (Ridgway plate XIII). Viability excellent. Homology uncertain.

pr: prune Townsend 61e. Sex-linked recessive. Spontaneous in Puerto Rican stock. Eye color on emergence reddish orange, very similar to Coral Red (Ridgway plate XIII), darkening with age to Morocco (Ridgway plate I). Viability excellent. Probably homologous with prune in D. willistoni.

metzii

Report of Norman L. Jordan, Jr.

sd: scalloped EMS induced in male, recessive. Margins of wings between veins II, III, IV, and V are scalloped. Phenotype variable, sometimes approaching wild type. Viability and fertility good. RK1.

gld: glider EMS induced in male, recessive. Wings held out from sides at an angle varying from 45° to 90°. Sometimes only one wing shows lateral divergence. Wings may also be elevated above the body. Viability and fertility good. RK1.

V-less: five less EMS induced in male, recessive. Distal part of vein V varies in length. Variation is from phenotype approaching wild type to no extension of vein V past the posterior crossvein. A posterior crossveinless condition can also occur. Viability and fertility good. RK1.

LINKAGE DATA

hydei

Report of W. W. Doane

The Amylase (Amy) locus in hydei is associated with linkage group V (Doane, research note, this issue). Two electrophoretic variants, each characterized by a single major band, were distinguished by disc electrophoresis (Doane, DIS 41: 74). These codominant alleles are named Amy^7 and Amy^8 in accordance with the system of nomenclature used for melanogaster. Linkage data indicates the following c.o. frequencies: 17.2% between sca and cn, 5.5% between cn and Amy, and 8.9% between Amy and vg. In hydei, the linear arrangement of these loci appears to be sca cn Amy vg (or the reverse order), unlike the situation in melanogaster where the sequence of homologous loci in IIr is cn sca vg Amy.
(Supported by N.S.F. grant GB 1718.)

RESEARCH NOTES

Clancy, C. W. University of Oregon, Eugene, Oregon. Correction

The females used in the matings referred to in paragraph 4 of my note in DIS 42: 57 all bear attached-X chromosomes homozygous for the markers indicated. The underline ordinarily used to designate the attached-X condition was inadvertently omitted on retyping. Without this correction, the sense of the argument is lost.

McCrady, W. B. The University of Texas, Arlington, Texas. Injection of concentrated extract from delayed recovery D. melanogaster.

Previous work (McCrady and Sulerud, 1964, Genetics 50:509-526) has shown that it is possible, although difficult, to transmit CO_2-sensitivity to resistant flies by injection of cell free extracts prepared from Texas Delayed Recovery (TDR) Drosophila melanogaster. TDR flies are known to be homozygous for gene Dly which is the primary factor responsible for the delay in recovery from CO_2 exposure of these strains. However, the above mentioned transmission of CO_2-sensitivity suggests an infectious agent which is suspected of being causally related to gene Dly. The poor success of transmission of CO_2-sensitivity attained in many injection series suggests a very low titer of infectious material within TDR flies. In attempting to determine if low titer is responsible for the lack of success of CO_2-sensitivity transmission, concentration of TDR extracts by high-speed centrifugation prior to injection was accomplished in several experiments. In each experiment several hundred TDR flies were crushed in 0.25M sucrose (200-250 flies/ml) buffered at pH 7.5 with Tris-HCl. Centrifugation of the crude extract at 1200XG for 15 minutes was followed by centrifugation of the supernatant at 6000XG for 10 minutes. The supernatant was then centrifuged at 40000XG for one to one and one-half hours with all centrifugation being completed in a Sorvall Superspeed RC2-B automatic refrigerated centrifuge at 0-3°C. The pellet was resuspended in a small volume of sucrose solution and then injected into resistant Oregon D. melanogaster.

All control flies injected with CO_2-sensitive fly extracts prepared in the above manner became CO_2 sensitive while none of those injected with plain sucrose solutions or heat-treated TDR extracts did. While control flies gave the expected results, the CO_2 response of flies injected with the concentrated TDR extracts was often not clear cut in a manner similar to the situation encountered when non-concentrated TDR extract is injected. Most TDR-injected flies did not develop classical CO_2-sensitivity, however in all three injection series some sensitivity was induced with death of the affected flies following CO_2 exposure. For example, in one test 13 of 54 injected flies did not recover within 15 minutes after CO_2 exposure completed 31 days after the injection. Some recovery occurred later, but 8 flies died within 24 hours. In most cases there were some flies which recovered slowly in a manner suggestive of true delayed-recovery. Other evidence supporting the conclusion that some transmissible agent is involved appears when comparisons of control and test flies which survived CO_2 exposures are made. Flies injected with TDR extracts were consistently weaker after CO_2 treatment than those injected with sucrose or TDR heat-treated extracts and were much less likely to survive for any given length of time after exposure. Following each CO_2 exposure many of the weakened flies of the TDR-injected test groups became stuck when returned to culture vials and died while few of the controls did. One striking example was the third injection series. After 45 days the 18 living control flies of a total of 26 injected with TDR heat-treated extract were quite healthy and active while only 13 of 108 TDR-injected flies of the test group were still alive and all were weak and inactive. The difference in survival percentages (69% survival for controls to 12% survival for the test group) as well as the observable differences in general health of the two groups indicates a difference in the two extracts injected. The fact that the two extracts were the same except for heat treatment prior to injection into the control flies must mean that the different responses are related to some heat-liable transmissible substance in the TDR extracts.

One conclusion which can be drawn from these observations is that the concentrated TDR extracts contain an infectious agent, not necessarily in small quantities, but essentially of a "weak" nature which results in chronic detrimental effects following CO_2 exposure that eventually kill most of the flies injected with such extracts. The differences between flies injected with TDR extracts and flies injected with extracts from CO_2-sensitive flies could be the result of a less virulent form of virus σ which normally kills infected flies immediately after CO_2 exposure. The possibility of a mutant form of σ in TDR flies may be related in some unknown way to the possible integration of the virus in the Drosophila chromosome as suggested by the presence of gene Dly.

Neeley, John C. University of Portland, Portland, Oregon. The interchromosomal effect of triploids upon crossing-over.

Regular diploid daughters and sons of 3n females marked with $\overline{XX}$, Df(1)60g, Ins(1)sc^8, dl-49, y w ec•B f, v m, w^a sc^8/ ct^6 s f (a reversed-acrocentric, see DIS 37:113) were analyzed for recombination products. Of 66 females mated, 36 produced no progeny whatsoever and 24 produced less than ten F_1 each; only six 3n mothers (9.1%) produced ten or more scorable offspring. The distribution of scored F_1 is given in tables A and B. Table C compares these values with those obtained by Beadle (1934. J. Genetics 29:227-309), by Schultz and Redfield (1963. DIS 38: 46-48), and with the diploid map unit values.

In an effort to gain similar data from the proximal arm of the RA, a transposition of dl-49 to the left arm was attempted. Fifty-eight separate matings failed to accomplish this. Consequently, the procedure was altered so that the proximal arm retained the dl-49 and the free-X homolog contained sc^8, dl-49 inversions, viz. $\overline{XX}$, y w ec•B f, v m, w^a sc^8/Ins(1)sc^8, dl-49, y^{31d}.

Unfortunately, exchanges between the scute and apricot loci (high distal exchange region 1) go unrecognized. Of 33 crosses, only 17 females produced a total of 63 sons (2n daughters were not counted here). Highly tentative results from these exchanges are: white-miniature, 27%; miniature-vermilion, 1.4%; vermilion-forked, Bar, 21.6%; distal to forked, Bar, up to 10%. The value of such data is highly doubtful.

The quantitative significance of the distal data is reduced, due to the high infertility rate (54%) and the low family size (mean family size for all 66 females is 2.61, for the six females producing over ten F_1, 18.2). Nevertheless, qualitative conclusions can be drawn from these recombination events:

1. Corroboration of the pattern of interchromosomal effects--an increase of exchange at the distal tip with a reduction near the middle portion of the chromosome;
2. Further delineation of this distal regional increase--from the yellow-echinus region into the yellow-white and the white-echinus regions;
3. Not only does triploidy per se increase these exchanges (Beadle's and Schultz and Redfield's data in table C), but the configuration of the chromosomes apparently superimposes additional effects.

Confirmation of this last conclusion can occur only with the accumulation of additional data. This 3n stock has recently died out, so that such confirmation must come from other sources.

Table A. Distribution of progeny according to sex and recombination type.

sex	non-cross-overs	Single exchanges					Multiple exchanges									SUM
		y-w 1	w-ec 2	ec-ct 3	ct-s 4	s-f 5	1-3	1-4	1-5	2-4	2-5	3-4	3-5	4-6	1-2-3	
♀♀	46	6	8	14	5	4	1	0	1	0	1	2	2	1	0	91
♂♂	36	4	8	10	10	4	4	1	1	1	0	0	1	0	1	81
SUM	82	10	16	24	15	8	5	1	2	1	1	2	3	1	1	172

Table B. Frequency of exchanges classified as to regions of exchange.

REGION	0	1	2	3	4	5	6
Numbers	82	19	19	35	20	14	1
Frequencies	47.7%	11.0%	11.0%	20.3%	11.6%	8.1%	0.5%
2n map intervals	---	1.5	4.0	14.5	23.0	13.7	9.3

Table C. Comparison of various cross-over frequencies

Regions involved	Standard 2n map interval	Beadle (1934) $\overline{XX}$,RM	Schultz & Redfield (1963) free-X	Neeley (this paper) $\overline{XX}$,RA
y - ec	5.5	7.1-8.4*	8.0-12.1*	22.0
ec - f	51.2	32.7	23.1	40.5

*values indicate 2X1A and 1X1A gametes, respectively

Anders, F., A. Altmaier, A. Anders and R. Prüssing. Genetisches Institut der Justus Liebig-Universität Giessen, Germany. Investigations about the bar- and antibar-effects.

In connection with our investigations about the bar- and antibar-effects, we have analyzed the free amino acids of a standard, of a bar, and of an ultrabar strain of D. melanogaster (homogenates of larvae, which were in the first part of the last stage, temperature = 24°C). The result (average of 6 quantitative determinations) is to be seen in Fig. 1.(opposite page)

In general, it seems to be the rule that the ultrabar strain has a lower concentration of amino acids than have the other strains (see the right part of Fig. 1).

Only Ala and - probably - Gly have a distinct positive correlation between their concentration and the bar-effect. The correlation of the amount of Leu, Ileu and Met to the bar-effect is not significant. The concentration of Pro has a negative correlation to the bar-effect. Furthermore, it is striking that the basic amino acids, Lys, His and Arg, form a specific group. They reach their highest concentration in the bar and the lowest in the ultrabar strain.

As duplication and triplication of the 16A-region (=bar-region) represent a destructive principle (bar-effect), which can be reduced by adding certain amino acids to the food (Kaji 1958; Anders et al. 1967), we have incubated homogenates of the three strains at times with 5 µ Mol pro 1 g wet weight of the amino acids, which had been analyzed (see Fig. 1). The incubated homogenates were hatched at 24°C. The free amino acids were analyzed 30, 75 and 120 minutes after the beginning of the experiment. Homogenates without supplementation of amino acids were need for control.

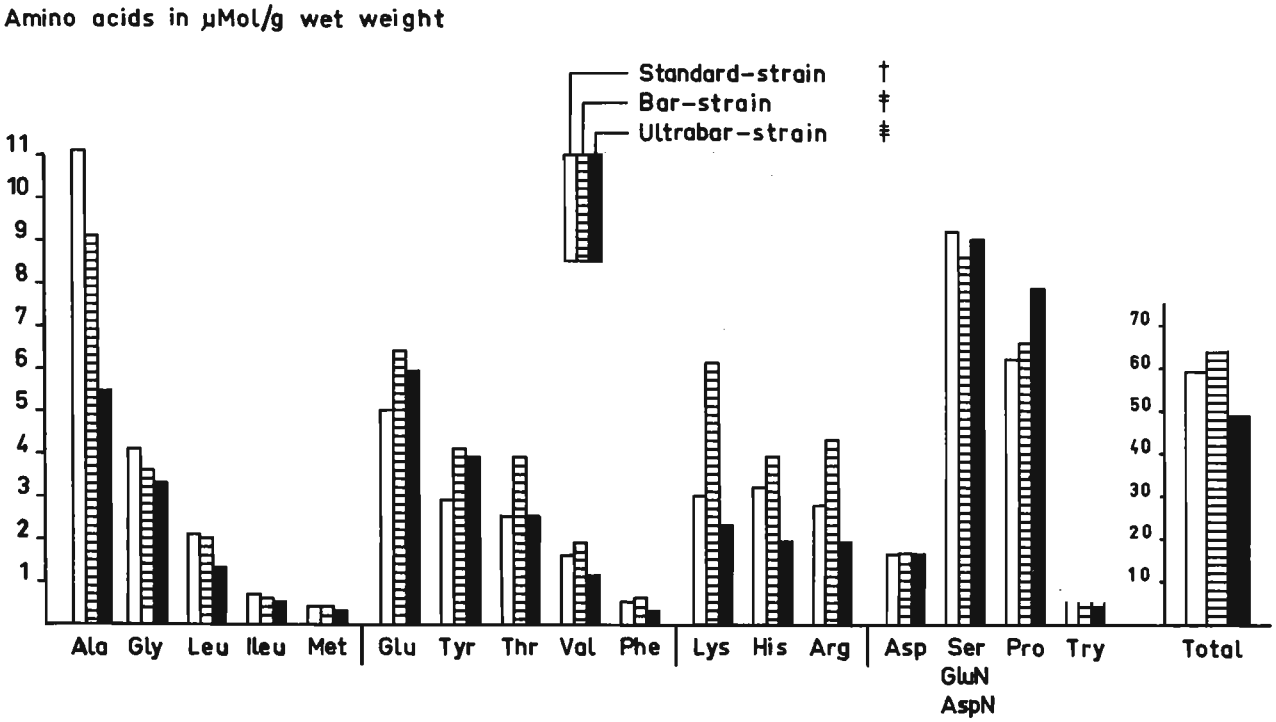


Fig. 1

For simplicity's sake, in the following only those substances shall be regarded, which have shown specific relations to the bar. In order to facilitate the comparison of alterations in homogenates with and without supplementation of amino acids, the amino acids which were added at the beginning of the experiment, were subtracted by calculation. The main results of these experiments are to be seen in Fig. 2.

In all the acid and neutral amino acids, it is striking that the concentration of Ala (and probably Gly) decreases (after supplementation of amino acids) in comparison with the controls (compare Fig. 2b with a). But it is of great interest that this is only to be seen in the standard and (not yet as distinct) the bar strains. In the ultrabar strain, the kinetics of Ala remains unchanged by incubation with amino acids.

In His, Lys and Arg (they are summarized in Fig. 2), the case is contrary. In this case, the standard-strain homogenate remains unchanged, while the bar shows a decrease, and the ultrabar even a deficit (compare Fig. 2 d with c).

In this paper, the fate of Ala (and Gly), His, Lys and Arg shall not be discussed. We only wish to state that in general, molecular His, Lys and Arg have the highest, and Ala (and Gly) the lowest antibar-effect of all amino acids proved. Furthermore, it is striking that the antibar-effect can be increased by adding mixtures of basic amino acids to the food. Of the greatest interest, however, is the fact that oligopeptides of Ala (and Gly) have a very high antibar-effect, while molecular Ala (and Gly) has only a very low one (see Kaji 1958). Mixtures of oligopeptides of Ala, Gly and molecular basic amino acids have the highest antibar-effect, we have ever observed in our laboratory. We suppose that the oligopeptides of Ala and Gly and the molecular basic amino acids influence two different functions of the bar-region. The oligopeptides may act against a natural proteolytic process initiated by the 16A-region, and the basic amino acids may repress the activity of this region.

Methods and more special results see Anders et al., Uber den Bar-Effekt bei Drosophila melanogaster, Verhandl. Deutsch.Zool.Gesellschaft in Heidelberg 1967, in print.
(This work is supported by Deutsche Forschungsgemeinschaft and Stiftung Volkswagenwerk.)

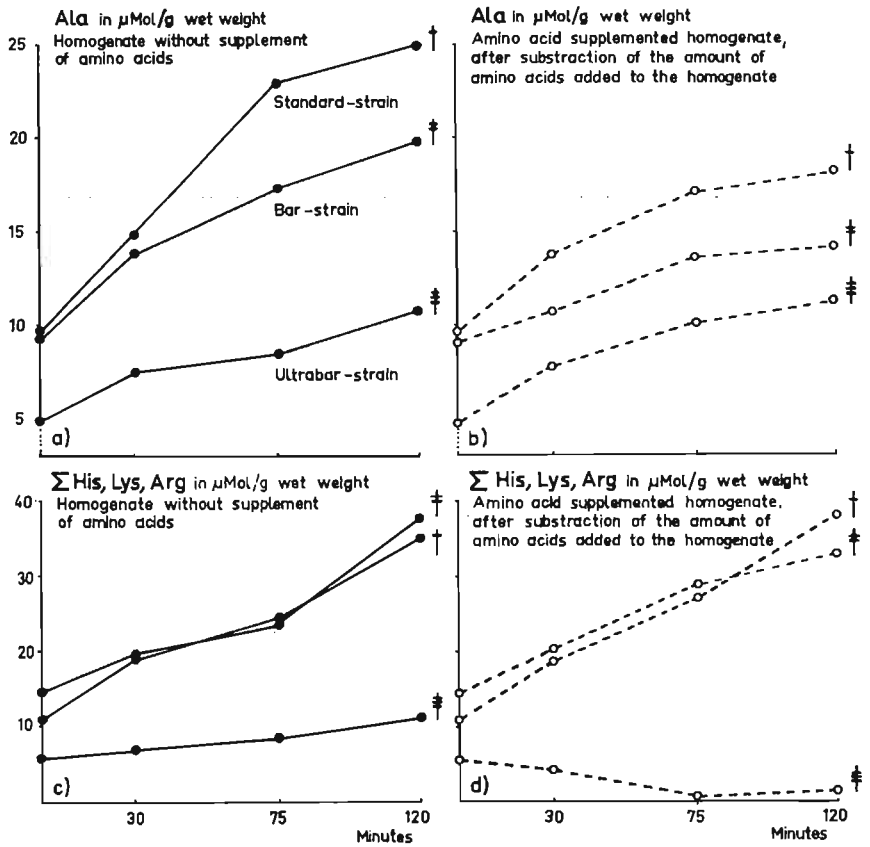


Fig. 2

Félix, R., V. Salceda and R. Villalobos-Pietrini. Comision Nacional de Energia Nuclear, Genetics Program, Mexico City, Mexico. Determination of the frequency of spontaneous recessive lethals in the sex chromosome of the Drosophila wild-type from México City.

The frequency of recessive lethals is determined by the Müller-5 technique in four test stocks. This is done as a preliminary to future experiments with addition of antibiotics to the culture medium before irradiation to test the protective effect of antibiotics.

The Müller-5 (Basc) stock was kindly sent to us by Professor H. J. Müller from Bloomington, Indiana. The X chromosome contains two inversions which avoid crossing-over in the total length of the sex chromosomes of heterozygous females. The markers w^a and B are very visible allowing the identification of the non irradiated maternal chromosomes along the experiment. The fertility and viability of the stock are high enough to permit the scoring of progeny and identification of recessive lethals.

The test stocks are: wild type from Mexico City (homogenized for 24 generations in the laboratory) and three stocks with known lethal frequency from previous experiments: Canton-S, Florida and w. The data for the last two are not included though we have not found any lethal until now in 4211 tested sperms. The procedure was performed in a small culture room with constant temperature regulated at 25° ± 1° C during all the experiment.

Procedure:

1. The isolated virgins (females with less than 6 hours after hatching are not fertilized by the males of the same culture) were mass mated to males aged from 2 to 4 days; 30 to 50 females were placed in each culture bottle, with 50 to 100 males.
2. After 10 or 12 days the emergence of the F_1 adults is starting. The adults are allowed to make free mating and 1 or 2 days after they are placed in aging bottles and left there for two more days.
3. The mature fertilized females (2 to 4 days old) start the oviposition immediately after its transference to vials. A couple or a female and two males are placed in each vial.
4. The absence of wild type males in a vial indicates the presence of a recessive lethal.
5. With the cultures containing less than 5% wild type males a new generation is started on. If the added adults from the two generations contain less than 5% wild type males the stock is classified as a lethal carrier.
6. The vials are placed in rows over a tray with a card containing the date of mating and the scoring of the progeny. The number of normal cultures is also written down as well as the number of lethal cultures and the ones which did not give any offspring (failures). In the cultures classified as "failures" a female stuck to the medium is usually found. Table 1 shows the summarized results obtained through six months. Table 2 contains the proportion of recessive lethals in a similar experiment from Spencer (1948).

Table 1. Frequency of spontaneous recessive lethals in the X chromosome of two wild type stocks.

STOCK	CHROMOSOMES TESTED	RECESSIVE LETHALS	PERCENTAGE
Wild type from Mexico City	3,962	8	0.20
Canton-S	1,469	1	0.07

Table 2. Frequency of spontaneous recessive lethals in the X chromosome of two wild type stocks. From Warren P. Spencer (1948).

STOCK	CHROMOSOMES TESTED	RECESSIVE LETHALS	PERCENTAGE
Pittsford, N.Y.	2,213	5	0.22
Canton-S	20,307	17	0.08

The test for spontaneous recessive lethal mutations has been done in several laboratories. After testing the effect of temperature on mutation frequency Plough (1941) obtained data from different stocks at a constant temperature. Demerec (1937) published an extensive study of wild type stocks from several laboratories, some of them obtained from places so widely separated that they were only distantly related. The results obtained from Plough, Demerec and from other authors on spontaneous mutability are summarized in Table 3.

Table 3. Frequency of spontaneous recessive lethals in chromosome 1 from wild stocks of Drosophila melanogaster.

STOCKS	DATA FROM	TESTED CHROMOSOMES	LETHALS	PERCENTAGE
Sukhum (Caucaso)	Zuitin	2039	24	1.18
Florida (U.S.A.)	Demerec, 1928	2108	23	1.09
Akhalcikh (Caucaso)	Duseeva	1300	10	0.77
Wooster 0 (U.S.A.)	Demerec, 1937	1266	8	0.63
Ticino (Italy)	Buzzati-Traverso	2335	13	0.56
Vladikavkaz (Caucaso)	Duseeva	1544	8	0.52
Florida (U.S.A.)	Shapiro y Volkova	9228	5	0.50
Formosa (Japan)	Demerec, 1937	2054	8	0.39
Ordjonikidze "Lab"	Zuitin, 1940	2348	8	0.35
Nalchik (Caucaso)	Sakharov	5169	18	0.35
Vladikavkaz (Caucaso)	Zuitin	2348	8	0.34
California-C (U.S.A.)	Demerec, 1937	708	2	0.28
Florida (U.S.A.)	Sakharov	81457	19	0.23
Birkina's	Birkina, 1938	9695	21	0.22
Pittsford (U.S.A.)	Spencer, 1948	2218	5	0.22
Florida (U.S.A.)	Olenov	2397	5	0.21
Lausanne (Switzerland)	Demerec, 1937	955	2	0.21
Mexico City	Félix, 1966 (this paper)	3962	8	0.20
London (England)	Timofeef-Ressovsky	5863	11	0.19
Swedish-B	Demerec, 1937	1627	3	0.18
Florida (U.S.A.)	Timofeef-Ressovsky	8963	15	0.17
Leningrad "Lab. line"	Zuitin	8614	14	0.16
Paris (France)	Timofeef-Ressovsky	7483	12	0.16
Florida (U.S.A.)	Buchman, Timofeef Ress. 1936	6495	10	0.15
Steglitz (Germany)	Timofeef-Ressovsky	8637	12	0.14
Merv (Turq.)	Lobashov	1424	2	0.14
Tashkent (Turq.)	Timofeef-Ressovsky	9972	13	0.13
Samarcanda (Turq.)	Magrzhikovskaya	4416	5	0.11
Kiev (Russia)	Timofeef-Ressovsky	12481	1	0.11
Madrid (España)	Timofeef-Ressovsky	5476	5	0.09
Canton-S (U.S.A.)	Spencer, 1948	20324	17	0.08
Canton-S (U.S.A.)	Félix, 1966 (this paper)	1469	1	0.07
Oregon-R	Demerec, 1937	3049	2	0.07
Oregon-R	Müller	3935	4	0.03

Félix, R., V. Salceda and R. Villalobos-Pietrini. Programa de Genetica, Comision Nacional de Energia Nuclear, Mexico City, Mexico. Induction of recessive lethals by x-rays in sex chromosomes during successive stages of spermatogenesis in the wild type of Mexico City Drosophila melanogaster.

The mutagenic effect of x-rays over the successive stages of spermatogenesis has been investigated by many authors. In this experiment the frequency of recessive lethals in the X chromosome is determined by the Müller-5 technique. The wild type from Mexico City was homogenized at the Laboratory for 24 generations. The general procedure consists in obtaining broods procedent from gametes in which the time and stage on which they were irradiated is known.

Auerbach (1954) obtained broods by mating a male with three females for three days, taking out the fertilized females and obtaining another offspring by placing three new female virgins with the same male. The offspring of each period is called brood which represents irradiated gametes in successive stages of spermatogenesis:

1st Brood: from 1 to 3 days after irradiation; stage during treatment: mature sperms and spermiogenesis.

2nd Brood: from 3 to 6 days after irradiation; stage during treatment: second meiotic division and early spermiogenesis.

3rd Brood: from 6 to 9 days after irradiation; stage during treatment: first meiotic division and probably some early stages in the second state of meiosis, late gonial cells.

4th Brood: from 9 to 12 days after irradiation; stage during treatment: spermatogonial cells and early meiotic cells.

Hoenigsberg (1961), obtained broods by mating a male with a female, and changing females every 24 hours. The highest proportion of dominant lethals was obtained in the 6 to 9 broods which belong to the second meiotic division and to the beginning of spermiogenesis at the moment of irradiation. Hoenigsberg's experiment shows that the stage of greater sensitivity comes after the first six days and even lasts until the ninth day. Both experiments should be explained in the same manner, but Auerbach's experiment reaches earlier the most sensitive stages due to the greater amount of spermatozoa transferred per day. In our experiment the transfer of spermatozoa is delayed by mass mating with the same number of males and females.

PROCEDURE

The experiment was performed as follows:

1. M-5 female virgins aged 3 to 5 days were mated to irradiated males (1 to 3 days old). The fertilized females were transferred to culture bottles with fresh medium and new emerged females were added every four days.
2. When the emergence of F_1 starts, the adults 1 to 2 days old were aged for 2 more days. The fertilized females (3 to 4 days old) lay eggs as soon as they are placed in test vials, in this way the F_2 emergence is assured.
3. Each test vial contains the male carriers of one irradiated X chromosome which has passed to the F_1 heterozygous females, just at the time the M-5 chromosome did. Vials without wild type males are scored as cultures with a lethal sex linked mutation induced by the x-ray treatment. Irradiation was applied in different doses to three groups of adult males.

A. Miller RT 250 apparatus was employed. It was operated at 250 kV and 8 MA with an additional 0.5 mm Cu filter. The total doses were delivered in the periods of time contained in table 1.

Table 1. Doses of x-rays applied to three groups of wild-type males.

Number of males	Doses	Duration of exposition
320	1,500r	1'21''
340	2,500r	1'35''
480	4,500r	2'43''

Male adults were mass mated in 14 cultures to M-5 females immediately after irradiation. They were transferred to new cultures after 4 days and two more broods were obtained by changing adults after two periods of 4 days. By this procedure the oviposition period of 12 days was divided in three equal periods. F_2 cultures without wild type males or with less than 5% of them were placed aside in order to obtain the 3rd generation. If the proportion of wild type males, putting the two generations together was less than 5% the culture was scored as a lethal carrier.

In the following table and graph the results of the experiment are summarized.

Table 2. Recessive Lethals induced by three different doses of X-rays.

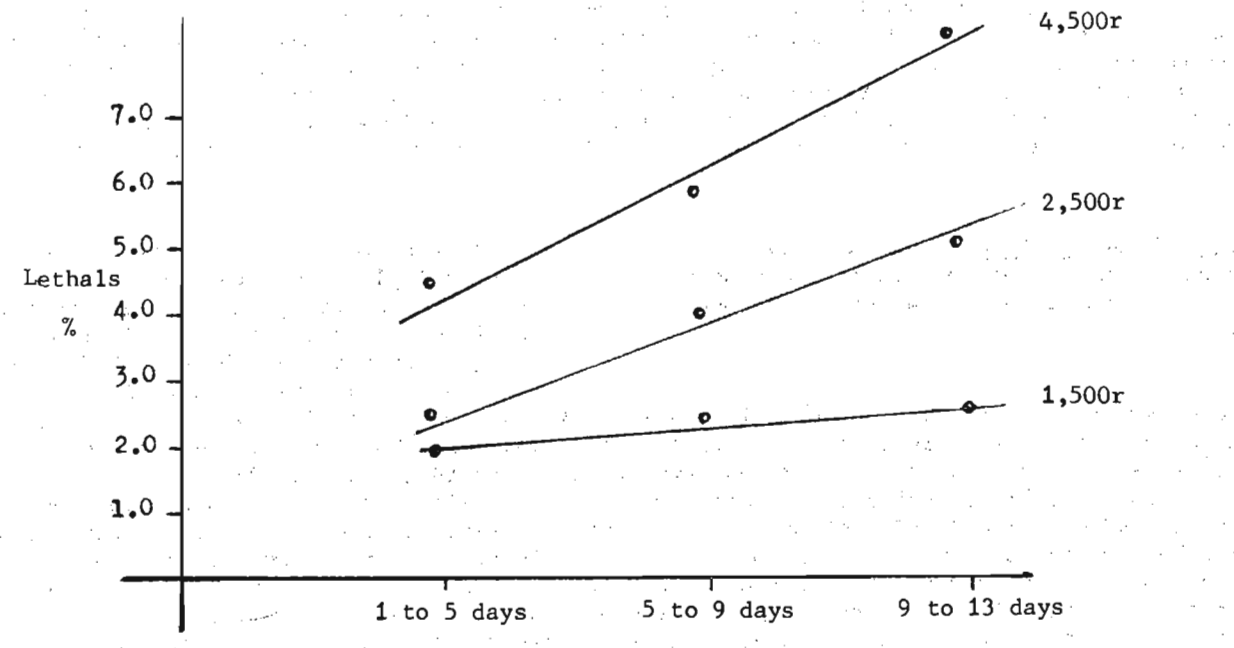
	First Brood (1 to 5 days)						Second Brood (5 to 9 days)						Third Brood (9 to 13 days)					
	1,500r		2,500r		4,500r		1,500r		2,500r		4,500r		1,500r		2,500r		4,500r	
	N	L	N	L	N	L	N	L	N	L	N	L	N	L	N	L	N	L
	277	5	310	6	282	11	288	6	322	11	198	10	298	7	219	10	270	20
%		1.81		1.93		3.90		2.08		3.41		5.05		2.34		4.56		7.41

r: roentgen units
N: normal offspring
L: lethals

The testes of the adult fly contains all the stages of spermatogenesis. Auerbach points out the greater sensitivity of the second brood which contains the higher proportion of spermatids and late spermatocytes. In this experiment the scoring of spermatozoa coming

from the irradiated stages of spermatogenesis were delayed because the number of females and males is the same in each of the four-day P mass cultures. By this procedure the second brood of Auerbach's experiment is reached only after nine days.

Graph 1. Lethals induced in successive broods with three doses of x-rays.



Puro, J. and P. Arajärvi. University of Turku, Finland. Localization of cp, in, and ri by means of T(2;3)spy.

A number of the 3rd chromosome genes of D. melanogaster between st (44.0) and p (48.0) are in a strategic position due to their intimate linkage with the centromere. Yet the evidence is contradictory as to how some of these genes are distributed into the two chromosome arms. In particular, the position of the in (47.0) and ri (47.1) loci in relation to the centromere has been in doubt. On the basis of a new translocation designated as T(2;3)spy (see New Mutants, this issue) we have been able to delimit the cp, in, and ri loci to the left of the band 79B1 in the salivary chromosome map.

T(2;3)spy is a homozygous viable reciprocal translocation with the recessive wing character (spready) inseparable from it. Linkage data suggest the breaks to the left of B1 and at the cp-ri region respectively in the 2nd and 3rd chromosomes. After introducing marker genes in the translocated chromosomes it could be shown that, in the new arrangement, ru and h (3L) belong to the same linkage group as bw (2R), whereas e (3R) and bw are in different groups. Salivary chromosome analyses of both heterozygous and homozygous larvae corroborate the genetic data showing the exchange of the left arms. The breaks are in 2L at 33D or E (probably just to the right of the thick doublet of 33D3-4) and in 3L at 79A (to the left of 79B1).

By studying crossing over in translocation heterozygous females the evidence was obtained that cp, in, and ri are to the left of the break point. Recombinants derived from females of the genotype th st cp in ri p^p/T(2;3)spy or th st in ri p^p e^s/T(2;3)spy were individually tested, by mating to translocation homozygotes, for the presence or absence of spy. The data indicate that crossing over at any of the regions st-cp, st-in, cp-in, or

in-ri always produced recombination of the left-hand markers and spy, or its complementary type without spy. On the other hand, crossing over at the region ri-p^p produced recombination of the right-hand marker (p^p) and spy, or its complementary type without spy. This suggests that the locus of spy (i.e., the break point) is very close to and probably to the right of ri. This was actually demonstrated by two cases of double crossing over derived from th st in ri p^p e^s/T(2;3)spy females after irradiation with X-rays; one of the type + st in ri + + showed recombination with spy but the other of the same type did not. This is to be expected, if crossing over at the ri-p^p region occurred, in the former case, between ri and the break point and, in the latter case, between the break point and p^p. It was further proved that + st in ri (spy) + + recombination retained the translocation. Linkage data obtained from translocation homozygous flies show the same linear arrangement of the genes from th to ri, but the crossing-over intervals have been increased to about 1.4 between th and st, 3.1 between st and cp, 12.9 between st and in, and 1.0 between in and ri.

Kaji, S. and Y. Hirosé. Kônan University, Kobe, Japan. Effect of nitromin and acid amides to the facet-formation of compound eye.

It has been reported that nitrogen mustard, methyl bis chloroethyl amine hydrochloride has strong inhibitory effect to the facet-formation of the wild type fly (Bodenstein and Abdel-Malek, 1949, Kaji and Ogaki, 1951, Bertschmann 1955). On the other hand, acid amides especially acetamide (1.8%) and lactamide (3%) were found to have the strongest facet-increasing effect to the Bar strain, so that the Bar eye was augmented to a size as large as that of the compound eye of wild type (Kaji, 1954, 1960).

In the present work has been confirmed that nitromin (methyl bis chloroethyl amine N-oxide hydrochloride) and lactamide have or not an antagonistic effect to the facet-formation both of the wild type and the Bar eye. For this purpose, the larvae of 70 hours age were exposed to nitromin for 1 hour and then exposed again for 1 hour with lactamide, or vice versa. Concentrations of both compounds were used at the same molarity, i.e., 0.108 mol.. After treatment in these chemicals, larvae were again transferred to the normal media until their emergence. The results of these tests are presented in Table 1 and 2.

Table 1. The effect of nitromin and lactamide on the development of the Oregon wild type eye.

treatment	n	number of facets in Oregon ♂♂		
		mean.	max.	min.
N M*	45	367.0	675	12
L A** ----> N M	31	622.1	697	406
N M ----> L A	26	661.2	692	480
L A	10	680.5	719	630
control	12	672.4	702	634

* N M : Nitromin (0.108mol., 1.5%)
** L A : Lactamide (0.108mol., 0.96%)

Table 2. The effect of nitromin and lactamide on the development of the Bar eye.

treatment	n	number of facets in Bar ♂♂		
		mean.	max.	min.
N M	23	42.1	78	10
L A ----> N M	19	54.3	82	30
N M ----> L A	10	49.4	72	26
L A	17	211.7	288	128
control	12	73.5	98	62

From the results of these experiments, nitromin was found to have the strong inhibitory effect to the facet-formation both of the wild and the Bar eye. On the contrary, lactamide was found to affect the facet number in the sense of increasing it. However, if attempt to consecutive exposure of these two substances respectively, no effect has been observed to the facet-formation. These two chemicals apparently have an antagonistic effect on the metabolic process of the eye development.

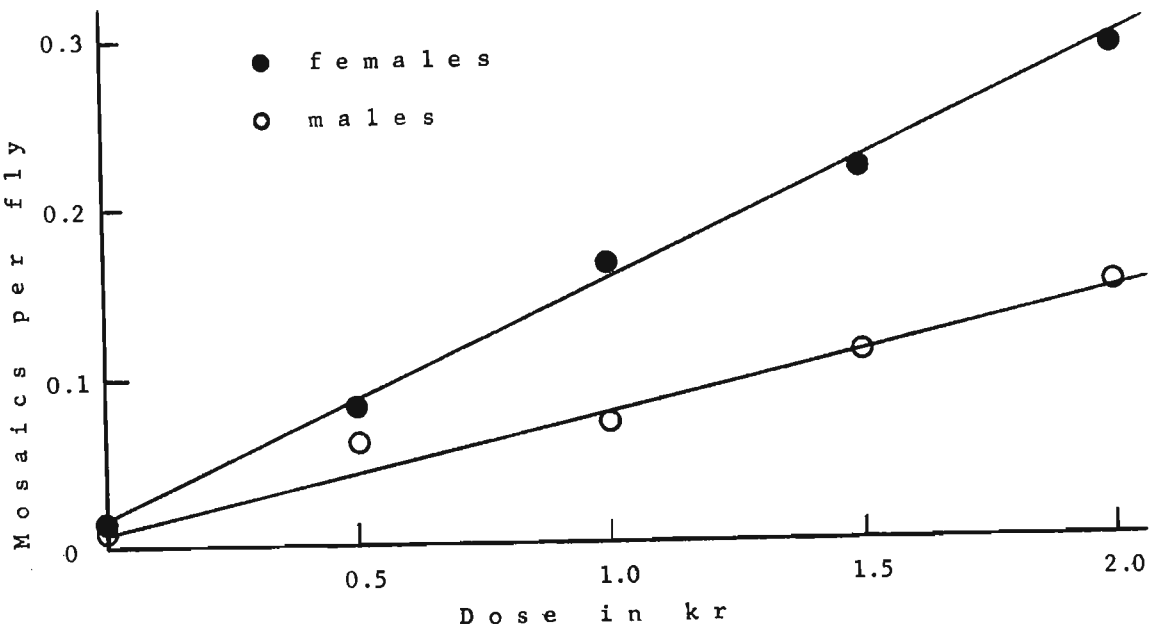
Bowman, J. T. Utah State University, Logan, Utah. X-ray-induced somatic reversion of the w^i mutant of D. melanogaster.

Previous studies of the white-ivory mutant (1 - 1.5) (Lewis 1959, Bowman 1965, Bowman and Green 1966) have shown that in contrast to spontaneous germinal reversions, X-ray-induced reversions occur in clusters. The two events are further differentiated by the effect of the homologue on the frequency of exceptional wild-type progeny. The frequency of spontaneous germinal reversion is essentially zero in males and in females that are heterozygous for w^i and a deficiency that includes the white region. These observations are interpreted to mean that spontaneous germinal reversion occurs primarily during the first meiotic prophase and is dependent on regular synapsis between homologues while the effect of X irradiation is restricted to premeiotic cells. It was of some interest, therefore, to determine if the somatic cells of males and females differ in their sensitivity to X-ray-induced reversion of the mutant.

Eggs from w^i parents were collected over a 12 hour interval on baker's yeast. Three days later, i.e. 72±6 hours after egg deposition, the larvae were collected by washing the yeast through a 60 mesh sieve. The larvae were transferred to filter paper and then irradiated with a Seifert "Isovolt 150" operated at 150 kvcp, 12 ma, with 1 mm Al filtration. The target to specimen distance was 50 cm and the dose rate, ca. 90 r/min. The treated larvae were then placed on standard medium to complete development. Throughout the experiment, the temperature was maintained at 25±1°C.

After eclosion, the eyes of the imagoes were scored for wild-type spots using 25X magnification. Under these conditions, subommatidial mosaics as well as larger spots are readily detected. The results are shown in the figure. It is immediately obvious that the frequencies of mosaics in both males and females are proportional to dose and that the slope of the line for females is about twice that of the line for males. In fact, the two regressions fit the equation $m = n\,(0.008 + 0.07\,X)$ where m is the number of mosaic spots per fly, n is the number of w^i loci per cell, and X is the dose in kiloroentgens. The coefficient of correlation is 0.95. Since the white locus is sex linked, n is 1 for males and 2 for females. These results show that there is no detectable difference in the frequencies of X-ray-induced reversion of w^i in the somatic cells of males and females and are consistent with the postulated premeiotic origin of induced germinal reversions.

Supported by National Science Foundation grant GB-4539.



Fahrig, R. and F. Anders. Genetisches Institut der Justus Liebig-Universität Giessen, Germany. The influence of glutamic acid upon the nucleic acid content of Drosophila melanogaster.

In earlier experiments we found that an excessive diet of glutamic acid enlarges the amount of DNA and free amino acids and the salivary gland chromosomes (Anders and Anders 1964; Drawert, Reuther und A. U.F. Anders 1965). The earlier experiments were for orientation only; exact determinations of DNA were still lacking. They are presented in this article. The determination of nucleic acid content of the larvae fed with glutamic acid has been carried out in five separate series of experiments. In this article, only the data of one of the five series will be presented (Table 1).

Table 1

	Control	
	No. of experiments	Mean with standard error
Weight of 100 larvae in mg	8	190.5 ± 0.94
Dry weight/100 g wet weight	8	15.46 ± 0.105
Nucleic acid content/100 g dry weight	8	5.30 ± 0.040
RNA content/100 g dry weight	8	5.02 ± 0.033
DNA content/100 g dry weight	8	0.190 ± 0.0070

	Experiment			
	No. of experiments	Mean with standard error	Mean deviation of the experiment from the control in %	Probability of error in % (t-test)
Weight of 100 larvae in mg	8	189.0 ± 0.38	- 0.79	ca. 25
Dry weight/100 g wet weight	8	15.49 ± 0.071	+ 0.19	ca. 50
Nucleic acid content/100 g dry weight	8	5.93 ± 0.040	+ 11.89	< 0.1
RNA content/100 g dry weight	8	5.56 ± 0.027	+ 10.76	< 0.1
DNA content/100 g dry weight	8	0.226 ± 0.0049	+ 18.95	< 0.2

At first one must observe that glutamic acid has no influence upon the weight of the larvae and the portion of the lipid-free dry-weight (table line 1 and 2). Upon the amount of nucleic acids, however, it has a significant influence. In total, the experimental objects have about 8 to 10% more nucleic acids than the control objects (table line 3).

RNA makes up the biggest part of the nucleic acids; therefore, here one finds about the same values as for the total content of nucleic acids (table line 4). But particularly spectacular is the influence of the glutamic acid diet upon the content of DNA. The experimental objects can possess about 50% more DNA than the control objects, the average is 25% (table line 5). Therefore, the DNA/RNA-ratio is changed considerably.

The extent of the salivary gland chromosomes depends in the same way on the glutamic acid diet (Fahrig, Sieger und Anders 1967). Hitherto existing countings of salivary gland cells gave no indication that their number is influenced by the glutamic acid diet. Therefore, the increase of the chromosomes can, assuredly, be led back to a raised content of DNA.

(This work is supported by Deutsche Forschungsgemeinschaft and Stiftung Volkswagenwerk).

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Anders, F. and A. Anders: Dietary chloramphenicol and glutamic acid as a medium affecting large and distinct stainable salivary gland chromosomes of Drosophila melanogaster. DIS 39 (1964) 87.

Drawert, F., K. H. Reuther und A. u. F. Anders: Über den Einfluss des Aminosäurenpools auf den Nucleinsäurengehalt bei Drosophila melanogaster. Experientia 21 (1965) 618.

Fahrig, R., M. Sieger und F. Anders: Über den Einfluss von Zuchttemperatur und Glutaminsäurefütterung auf den Nucleinsäurengehalt sowie die Chromosomengrösse und einige andere quantitative morphologische Phäne bei Drosophila melanogaster (Meigen.) Verh. d. Dtsch. Zool. Ges. (1967) in print.

Kalicki, H. G. and Thomas J. Trzaski.
Adelphi University, Garden City, New York. RNA synthesis in the Drosophila ring gland during pupal development.

The activity of the corpus allatum in the larva of the ebony mutant about to undergo puparium formation is higher than in the wild type (Canton-S) at a comparable time, and is manifested on the histophysiological level, by larger nuclei, a larger corpus allatum with evidence of secretory activity, and on the phenotype level, by a lack of tanning of the puparium (Kalicki, 1963; Wolsky and Kalicki, 1959). This higher activity suggests that there may also be a difference in the activity of the ring gland during pupal development.

Pupae, selected at five levels of development were injected with tritiated uracil in the posterior abdominal region and allowed to rest for two hours. They were then fixed, embedded in paraffin, sectioned serially, processed for autoradiography and stained with Methyl Green Pyronin. Grains over the prothoracic gland and the corpus allatum were counted in each section, and the counts for all the sections in the series were totaled to give the uracil uptake in the entire organ.

RNA synthesis, as indicated by uptake of radioactive precursor, in Canton-S prothoracic gland (Fig. 1-A) is high before pupation, and generally lower during pupation. There is a slight increase in synthesis during eye pigmentation which decreases only a little during bristle pigmentation, but this is followed by a sharp decline when the imago is ready to emerge. In the corpus allatum (Fig. 1-B) RNA synthesis is low during puparium formation and pupation, but increases markedly during eye pigmentation, after the corpus allatum has migrated into the thorax. Synthesis decreases during bristle pigmentation and in the ready-to-emerge imago.

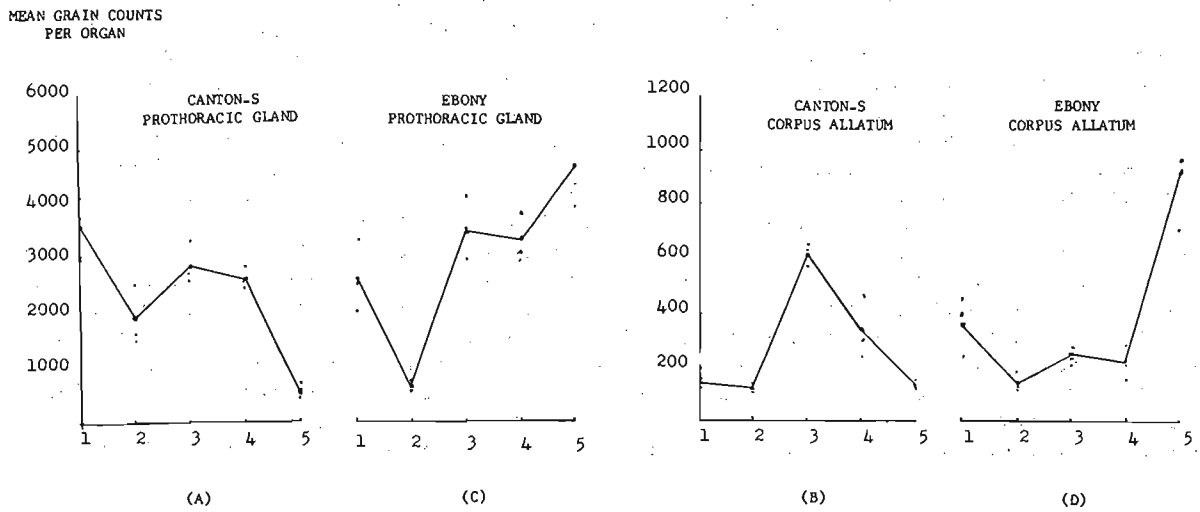

Fig. 1. Uptake of tritiated uracil in ring gland during pupal development:
(1) separation of prepupa from puparium
(2) pupation and secretion of pupal cuticle
(3) completion of eye pigmentation
(4) bristle pigmentation
(5) ready to emerge imago

RNA synthesis in the ebony mutant prothoracic gland (Fig. 1-C) is somewhat lower than wild type during puparium formation and drops to a lower level during pupation. Synthesis increases more sharply than wild type during eye pigmentation, remains high at bristle pigmentation, then, unlike the wild type, reaches a peak in the ready-to-emerge imago. In the corpus allatum (Fig. 1-D) RNA synthesis is higher at puparium formation, decreases to about the same level as the wild type at pupation and does not show the same increase of uptake at eye pigmentation. This low activity persists through the stage of bristle pigmentation, but uptake increases to a high peak at the period of pre-emergence.

A comparison of the activity of the ring gland during pupal development in the monogenic ebony mutant and in the wild type shows a dissimilarity in the patterns of synthesis. The wild type gene and its allele "ebony" are not equally susceptible to regulator influences. If RNA synthesis in the nucleus can be interpreted to reflect a derepression of the repressor locus of the chromosome, then the response of the mutant gene is quantitatively different from the wild type.

References:

Kalicki, H. 1963, Folia Histochem. et Cytochem. 1:423.
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Heed, W. B., J. S. Russell, and B. L. Ward.
University of Arizona, Tucson, Arizona.

Host specificity of cactiphilic Drosophila in the Sonoran Desert.

Arborescent cereus cacti of the Sonoran Desert provide specific habitats for different species of Drosophila. From samples of rotting limbs collected in the field from 1962 to 1967, we have reared the following species in the laboratory (see localities on map): Lophocereus schottii (senita) 19 localities in Jan. Feb. May June Nov. Dec. reared 2,380 D. pachea; Carnegiea gigantea (saguaro) 4 localities in Jan. Feb. Aug. Oct. Dec. reared 12,172 D. nigrospiracula; Pachycereus pringlei (cardon) 3 localities in Feb. May Dec. reared 353 D. nigrospiracula; Machaerocereus gummosis (agria) 6 localities in Feb. Mar. May Nov. reared 1,048 D. mojavensis; Lamaireocereus thurberi (organpipe) 5 localities in Jan. Nov. Dec. reared 113 D. mojavensis, 3 D. arizonensis, 1 D. longicornis; Rathbunia alamosensis (cina) 4 localities in Jan. Feb. Nov. Dec. reared 790 D arizonensis, 20 D. mojavensis, 13 D. pseudoobscura, 1 D. hamatofila.

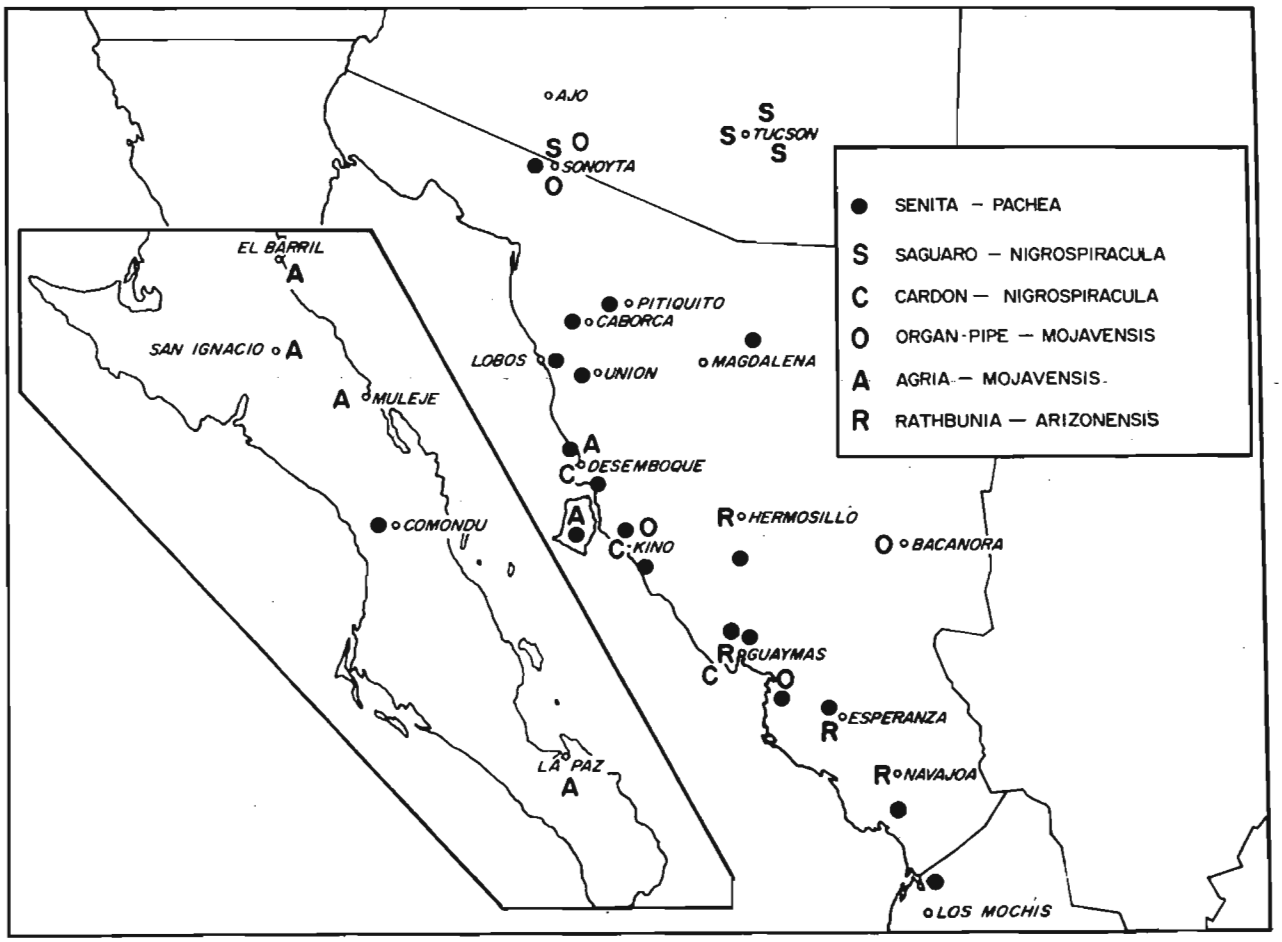
Of the four most abundant species, D. pachea is the only one truly monophagous. D. nigrospiracula has been bred also from Ferocactus. It is therefore classified as an oligophagous species. D. arizonensis is polyphagous having been bred also from Ferocactus, Opuntia, squash and citrus fruits. D. mojavensis has been bred also from Opuntia. It becomes more difficult to classify these species within the large genus, Drosophila, as each one becomes more monophagous. For instance, D. pachea (monophagous) belongs to a very small species group having only a few morphologically distant relatives. D. nigrospiracula (oligophagous) is a member of the large repleta species group but does not clearly fit any known subgroup. D. mojavensis and D. arizonensis (polyphagous) are closely related sibling species and have 10 other closely related members collectively known as the mulleri subgroup of the repleta group. The different degrees of host plant specificity may represent different levels of time with D. pachea being the oldest. We thank Dr. L. E. Mettler for checking D. arizonensis and D. mojavensis cytologically.

Heed, W. B. and J. S. Russell.
University of Arizona, Tucson, Arizona.

Inability of D. pachea to breed in cereus cacti other than Senita.

From our records D. pachea breeds only in the rot pockets of the arms of the cereus cactus, Lophocereus schottii (called senita) where it obtains the sterol, schottenol, necessary for its growth and reproduction. The question arises whether or not pachea can utilize other cereus cacti in the laboratory which grow sympatric with senita in the Sonoran Desert.

Cactus cube + banana medium	Progeny A	Progeny B	Progeny Total	Female Progeny Fertile	Sex Ratio F	Sex Ratio M	Av. no. filamented eggs/F	Range/F	No. females dissected
Lophocereus schottii (senita)	198	163	361	yes	55	47	21	(7-40)	10
Lemaireocereus thurberi (organpipe)	60	41	101	no	14	36	5	(0-16)	14
Rathbunia alamosensis (cina)	16	30	46	no	11	20	3	(0-9)	4
Machaerocereus gummosis (agria)	24	7	31	no	4	17	1	(0-4)	4
Pachycereus pecten-aboriginum (hecho)	10	11	21	no	2	10	0		2
Pachycereus pringlei (cardon)	6	3	9	no	1	5	0		1
Carnegiea gigantea (saguaro)	1	0	1	no	0	1	0		0
No cactus	39	64	103	no	19	49	5	(0-23)	10



The tests confirm our field observations that pachea is monophagous. The table summarizes the data from 14 test vials run concurrently with 7 kinds of cereus cacti and two control vials (no autoclaved cube of cactus added to banana media). Each vial was charged with 15 egg-laying females and 15 fertile males randomized from 5 cultures of different geographic origin. The parents, which were as yet alive, were discarded 22 days later to prevent overlap with the progeny, which began emerging 3 days after discard. Many eggs were deposited by the parents on senita, organpipe, and agria cactus cubes. Only a few eggs were deposited in the vials with saguaro. All progeny were placed on fresh banana media having the same kind of cactus cube as their parents. Twenty-one days after the first emergence, the senita progeny had deposited many eggs, which later hatched, while all other cacti and the control progeny had deposited none. Male progeny from all vials were fertile. There were 2.7 times more male than female non-senita progeny among the flies selected for dissection. Even though eggs with filaments were found in some of the ovaries of non-egg laying progeny, the filaments were not as distinct as the senita progeny indicating that development in this case was arrested in a late stage. Several of the cacti, other than not containing the correct sterol, also appear to inhibit D. pachea in parental egg laying and larval development. Separate tests were run with Peniocereus greggii (night-blooming cereus). Both the tuberous roots and the thin stems were negative for supporting pachea. These 8 species of cacti represent the common cereus cacti of the subtribe Cereanae (arborescent cacti) in the Sonoran Desert. The genera Bergerocactus, Cephalocereus, Myrtillocactus and Wilcoxia have more local distributions in this desert and have not been tested.

Mather, Wharton B. University of Queensland, Australia. Chromosomal polymorphism in two marginal populations of D. rubida.

When D. rubida was discovered at Cairns, Queensland, Australia in 1958 it appeared to be monomorphic for chromosomal inversions (Mather, 1961) which fitted well with the hypothesis of da Cunha and Dobzhansky (1954) that inversion heterozygosity falls off towards the periphery of the range of a species. Cairns is certainly near the southern limit of the range of the species. Since 1958 it has been shown that D. rubida is highly polymorphic for inversions throughout Papua and New Guinea.

Two stations in the Cairns area were sampled with banana bait in June 1966 with the results shown in the table. Crystal Cascades is 7 miles S.W. of Cairns and Lake Barrine 21 miles S.S.W. of Cairns. Both habitats consist of tropical rain forest and were collected from within a few days of each other but whereas Crystal Cascades is at sea level Lake Barrine is on the Atherton Tableland at 2400 feet.

From the table it will be noted that both stations are polymorphic for inversions. However this polymorphism is not nearly so marked as at stations nearer to the centre of the range of the species in Papua and New Guinea.

Acknowledgements are due to Sheridan Butler and V. Baimai for technical assistance.

Chromosome	Crystal Cascades %		Lake Barrine %	
	♂	♀	♂	♀
II +	93.4	94.2	96.9	92.9
RC	6.6	5.8	3.1	7.1
III +	72.1	73.1	68.8	75.7
A	1.8	1.9	-	-
B	2.2	1.9	-	-
D	26.6	25.0	31.3	24.3
Flies scored	113	26	16	35

References:
da Cunha, A. G. and Th. Dobzhansky, 1954. A further study of chromosomal polymorphism in Drosophila willistoni in relation to the environment. Evolution, 8:119-134.
Mather, W. B. 1961. Chromosomal polymorphism in Drosophila rubida Mather. Genetics, 46:799-810.

Band, H. T. Michigan State University, East Lansing, Michigan. Persistence of increased developmental homeostasis in a natural population of Drosophila melanogaster.

Recent investigations on the South Amherst natural population of D. melanogaster have indicated the population is undergoing another genetic change (Band, 1964). This change has been manifest largely as a reduction in environmental variance noted among the random heterozygotes in 1962 in comparison with previous estimates of this parameter obtained in 1961 and 1960. Environmental variance is a measure of developmental homeostasis. The results suggest an increase in developmental homeostasis.

Analysis of the population was undertaken in 1964 to determine if the reduced environmental variance persisted. No change had been noted among lethal and semilethal frequencies. The estimate of 34% lethal and semilethals among 141 second chromosomes from the Sept. 1964 collection likewise failed to indicate any significant change in the frequency of these variants. Table 1 presents the viabilities of the random heterozygotes, all homozygotes and quasinormal homozygotes at 25°C (77°F) in the 1964 sample. Two replicates were counted for each chromosome or combination. The results are in agreement with those obtained in previous years.

TABLE 1

	$\bar{x}$(%)	σ	N	no. counted
random heterozygotes	27.7± 0.33	5.32	133	29,728
all homozygotes	15.7± 0.64	10.82	141	27,540
quasinormal homozygotes	22.6± 0.40	5.42	93	19,201

TABLE 2

	σ^2_{ges}	σ^2_e	σ^2_s	σ^2_g
random heterozygotes	33.15	14.08	9.35	9.71
quasinormal homozygotes	29.75	20.16	8.84	0.75

Table 2 shows the "total", environmental, sampling and genetic variances, respectively, obtained from the data. Environmental variances for both random heterozygotes and quasinormal homozygotes are in excellent agreement with 1962 estimates. In that population, these estimates were found to be 14.13 for random heterozygotes and 20.79 for quasinormal homozygotes. The frequency of subvitals in 1964 was only 10%, the remaining quasinormal chromosomes falling into the normal category.

Concurrently, as a further test of the hypothesis that genetic changes have occurred in response to a more unfavorable environment, experiments on random heterozygotes and homozygous chromosomes were undertaken at F64/90 (°F, wide range environment). Again, there were two replicates per chromosome or combination. (Parents were the same for all 4 replicates: 4 ♀♀ x 4 ♂♂, transferred 4 times every 3 days, A and C replicates being in 77°F and B and D replicates in F64/90). There was no evidence of statistically significant viability differences among the random heterozygous classes. Viabilities were similar at 77°F and F64/90. Significant differences in viabilities among heterozygous classes at F64/90 had been found using chromosomes from the 1960 population (Band, 1963). In the 1964 analysis random heterozygotes were partitioned into drastic/drastic (d/d), drastic/nondrastic (d/nd), and nondrastic/nondrastic (nd/nd) classes both on the basis of homozygous chromosome performance at 77°F and F64/90 since no homozygous 1960 chromosomes had been analyzed at F64/90.

As a whole, the 1964 data are consistent with the hypothesis that the population is undergoing a genetic change and that the change is in response to more unfavorable environmental conditions. They also show that population viabilities and environmental variances at 77°F are reproducible even when performed in different laboratories. Alternative hypotheses to account for the reduced environmental variance among random heterozygotes in 1962 would fail to account satisfactorily for its persistence in the 1964 population and the lack of significant viability differences among heterozygous classes at F64/90 with 1964 chromosomes.

(Supported by NSF grant GB-1775. The major portions of the calculations were completed during the tenure of a U.S.P.H. Special Fellowship 1-F3-CA-8040-01 when the author was a guest investigator in Prof. Thoday's laboratory at the University of Cambridge.) Band, H. T. 1963, Evolution 17: 307-319; _____, 1964, Evolution 18: 384-404.

Hanks, G. D. University of Utah, Salt Lake City, Utah. RD (Recovery Disrupter) activity associated with various wild type X chromosomes.

Males were taken from various wild type stocks and mated to attached-X females containing an RD background including the Y chromosome. After 6 backcross generations (8 for Oregon R) males were tested extensively by mating to 5 al, ru tester females. Results are given in Table 1. It is noteworthy that apparently some ordinary wild type stock X chromosomes have significant RD activity.

Table 1: Percentage of females obtained from individual tests of males containing X chromosomes from the ordinary wild type stocks listed.

Oregon R	Swedish C	Urbana S	Canton S	Control (RD)
60.4	63.0	53.3	53.7	65.2
65.0	63.0	54.1	50.8	70.1
57.6	64.0	53.1	52.4	65.0
65.2	65.5	51.6	50.4	67.1
62.9	66.7	50.0	48.0	67.6
61.3	62.0	51.2	50.6	67.9
64.4	69.6	51.6	49.6	63.9
60.1	68.5	51.6	53.3	68.2
62.5	65.3	54.0	50.8	
61.4	62.8	53.3	53.9	
$\bar{X}$ = 62.1	$\bar{X}$ = 65.0	$\bar{X}$ = 52.4	$\bar{X}$ = 51.4	$\bar{X}$ = 66.9
n = 10	n = 10	n = 10	n = 10	n = 8

Hanks, G. D. University of Utah, Salt Lake City, Utah. Rate of recovery of products from nondisjunction of the 4th chromosmme in RD strain males.

Since Erickson (1965) found cytological nondisjunction of the 2nd or 3rd chromosomes to be 7.3% of the cells resulting from the first meiotic division, and Erickson (personal communication) states that his cytological work suggests that the 4 is rather often nondisjunctional, it would seem to be of interest to compare the rate of genetic recovery of nullo-4 gametes. Males heterozygous for a stock 4th chromosome marked with ci ey^R or ey were mated to three spa^{pol} females. Nullo-4 gametes from the male would be recovered as sparkling polished progeny. Progeny were classified as to sex and the sparkling polished phenotype. In a total of 13,699 progeny 10 were sparkling polished and the grand mean percentage of females was 63.6%. The frequency of Haplo-4 males is only slightly greater than the 1 in 2000 reported by Morgan, Bridges, and Sturtevant (1925). Perhaps there is a mechanism that provides for selection against recovery of unbalanced gametes with respect to some autosomes at least.

References: Morgan, T. H., C. B. Bridges, and A. H. Sturtevant. 1925 The Genetics of Drosophila. Martinus Nijhoff, The Hague p. 140.
Erickson, John 1965 Genetics 51: 555-571.

Hanks, G. D. and S. Kimberling. University of Utah, Salt Lake City, Utah. Rate of recovery of nullo-XY sperm from males carrying the sc^8 Y with and without RD autosomal background.

Single males with the sc^8Y (marked with y^+) and with RD background including the X were mated to five y v females. 100 yv exceptional males were scored in a total of 2500 male progeny (4%). The percentage female value was 60.3. The controls were performed by mating males carrying the sc^8Y and an ordinary stock X without RD autosomal background to five y v females. Only 11 y v males were recovered in 7,972 male progeny (0.138%). This is compelling evidence that nondisjunction of the sex chromosomes has a genetic component in either the X or the autosomes or both.

Frankel, A. W. K. University of Iowa, Iowa City, Iowa. Exchange between Y chromosomes.

Crossing over in the male between sterile Y chromosomes (derived from y^+ Y, Brosseau, '60 Genetics 45:257) and an $X \cdot Y^L$ chromosome was examined in the process of generating $X \cdot Y^{KL^-} y^+$ chromosomes for another purpose. ycvvfcar$\cdot Y^L$/ y^+ $Y^{KL^-} \cdot Y^S$ ♂♂ were mated to RA yf/ Y^{BS} ♀♀. The expected cross overs, unmarked fertile Y's and $X \cdot Y^{KL^-} y^+$ were recovered as yf ♀♀ (RA yf/ O is lethal) and cvvfcar B ♂♂ respectively. The data are presented in Table 1.

Table 1. Exchange between marked free Y's and an attached $X \cdot Y^L$ chromosome.

RA yf/ Y^{BS} ♀♀ x ycvvfcar$\cdot Y^L$/ $y^+ Y^{KL^-} \cdot Y^S$ ♂♂

Stock	Loci Affected	Total No. Flies	Crossovers* yf♀	cvvfcarB♂	Frequency of Crossing over x 10^{-4}
L 3	kl 5	37,865	6	4	2.6
L 19	kl 3,4,5	11,023	2	2	3.6
L 24	kl 3,4,5	29,963	20	7	9.0
L 28+	kl 5	21,515+	34	12	10.7*
L 36	kl 4,5	34,865	9	5	4.0
L 38	kl 3,4	14,471	14	7	14.4
L 41	kl 3	36,882	17	6	6.2
4-15	kl 1,2	38,751	20	23	11.1
4-65	kl 3,4,5	29,383	11	12	7.8
4-93	kl 3	14,928	10	7	11.4

*Corrected for nondisjunctional progeny.

+The ycvvfcar$\cdot Y^L$ was found to contain a y^+ suppressed lethal therefore only female and recombinant male survivors were recorded.

*Calculated by doubling the number of females as an estimate of the total number of flies.

All of the free Y's used, except 4-15, show a variegation for male fertility (V-type position effects, Brosseau personal communication). It is therefore probable that these chromosomes contain intrachromosomal rearrangements which may account for the very different frequencies of crossing over as well as the differential recovery of the reciprocal recombinant classes of some chromosomes. Nonrandom recovery may reflect the occurrence of nonrandom disjunction of recombinant chromosomes.

It is likely that at least some of the crossing over occurs during premeiotic mitoses (Brosseau, '58 DIS 32:115). Since more than one male parent was used in each bottle the present data do not provide conclusive evidence for clustering.

Exchange between the free Y and the attached $X \cdot Y^L$ occurs with frequencies similar to the frequencies of exchanges between X heterochromatin and a Y (Lindsley '55 Genetics 40:24). One might expect that with more extensive regions of homology between the attached $X \cdot Y^L$ (probably containing a Y centromere and part of Y^S in addition to Y^L) and a free Y exchanges could occur in more than one place. This might be manifested by a higher frequency of exchange, however the frequencies are not substantially enhanced. The existence of multiple places of exchange is suggested by the additional finding that though the derived attached $X \cdot Y^L$ position effect chromosomes as a group often show different fertility levels from that of the free Y position effect chromosome, in some cases there exist two distinct classes of fertility levels among the recombinant chromosomes, perhaps reflecting qualitative differences in their basal heterochromatin.

(Work supported by Research Grant GM 06508-08 U.S.P.H.S.)

Mather, Wharton B. University of Queensland, Australia. The genus Drosophila in Sabah.

In August 1966 the genus Drosophila was sampled from fermenting banana baits at Jesselton, Sandakan and two stations at Tawau (Fig. 1). At Jesselton the baits were placed in patches of lush vegetation near the town, at Sandakan in a rubber plantation five miles from the town and at Tawau in a rubber plantation 10 miles from town (Tawau I) and in a cocoa plantation at the Agricultural Station 2 miles from town (Tawau II).

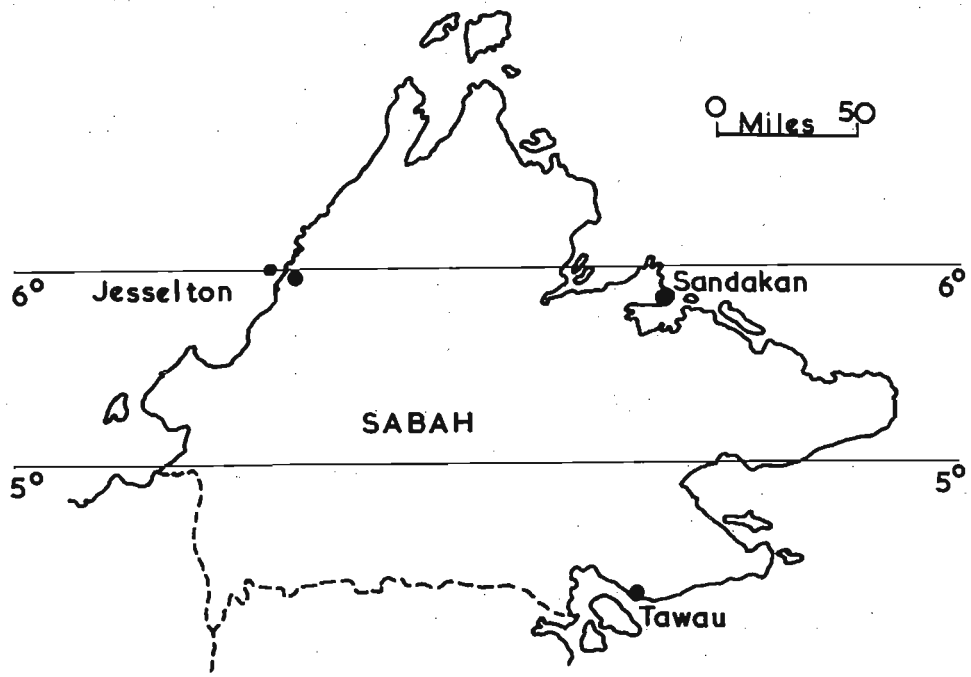


Fig. 1

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

Table I

Primary Sorting

	Jesselton		Sandakan		Tawau I		Tawau II	
D. setifemur	147	12%	346	26%	16	4%	37	2%
D. pararubida	10	1%	10	1%	15	3%	129	6%
D. tetrachaeta	-		-		2		-	
D. argentostriata	2		-		-		-	
D. silvistriata	-		-		-		-	
melanogaster group	1087	87%	966	73%	427	93%	1965	92%
Total	1246		1322		460		2131	

Table II

melanogaster group sample

	Jesselton		Sandakan		Tawau I		Tawau II	
D. ananassae	27	61%	24	43%	41	68%	5	12%
ananassae-like	-		-		-		2	5%
D. szentivanii	6	14%	12	21%	8	13%	34	81%
pseudotakahashii-like	4	9%	4	7%	2	3%	-	
D. dominicana	4	9%	15	27%	5	8%	-	
serrata-like I	1	2%	-		4	7%	1	2%
serrata-like II	2	4%	-		-		-	
serrata-like III	-		1		-		-	
Total	44		56		60		42	

It will be noted that at all stations the melanogaster group is very dominant. The immigrans group is represented by D. pararubida and D. setifemur.

Cultures of the species from the four stations have been preserved and are being studied in relation to cultures of the species from the Territory of Papua and New Guinea as regards chromosomal variation and reproductive isolation.

Acknowledgements are due to Research Assistant Sheridan Butler and Graduate Students V. Baimai and I. R. Bock for technical assistance.

Mather, Wharton B. University of Queensland, Australia. Chromosomal Polymorphism data in D. rubida from north eastern New Guinea.

One of the gaps in our knowledge of chromosomal polymorphism in D. rubida is in flies from the north coast of New Guinea. Some inversions have been recorded from small samples from Samarai, Popondetta and Lae (Mather, 1961 & 1963). This report records data from a sample of ten male flies at a new station - Madang. The new complex inversion J has limits of 7.1 and 14.1 in chromosome IIR on the giant chromosome photographic map (Mather, 1961).

Acknowledgements are due to Sheridan Butler and V. Baimai for technical assistance.

Chromosome		%
II	RC	62.5
	J	37.5
III	+	68.8
	A	18.8
	B	12.5
	D	6.3
	E	18.8

References:
Mather, W. B. 1961. Chromosomal polymorphism in Drosophila rubid Mather. Genetics, 46:799-810.
Mather, W. B. 1963. Patterns of chromosomal polymorphism in Drosophila rubida. Amer. Nat., 97:59-64.

Hirosé, Y. and S. Kaji. Kônan University, Kobe, Japan. ^{3}H-acetamide incorporation into eye discs of Bar strain.

Previous work has been shown that acetamide has strong effect to the facet-formation of the Bar eyes and increasing the number of facets as many as that of the wild type eye (Kaji, 1954). The sensitive periods of the eye discs to the chemical influence to lie between 60-80 hours after hatching (Kaji and Ogaki, 1953).

The present report describes studies on tritiated acetamide incorporation into eye discs of different larval ages.

In 55, 62, 70, 80 and 90 hours old larvae were treated with ^{3}H-acetamide (2%, 5.64 uCi/g) for 1 hour respectively, and then transferred to normal media for growth until the end of larval stage. Carnoy was used as a fixative, and sections ranged from 2 to 3 u. Autoradiographic exposure for tritiated acetamide was 3 days. The figure 1 shows photomicrograph of the preparation of Bar eye disc.

Comparison of ^{3}H-acetamide incorporation with different stages of eye discs showed that 62-70 hours disc was far stronger labeled than in before and after this stage. Within this period, the most effective point is found at 70 hours after hatching (Fig. 2). As is apparent from the experiments, quantitative differences of ^{3}H-acetamide incorporation can be seen in different larval stage,, and incorporation was marked at 70 hours discs. This stage of larval development is corresponding with the sensitive period of the eye discs to the chemical agents.

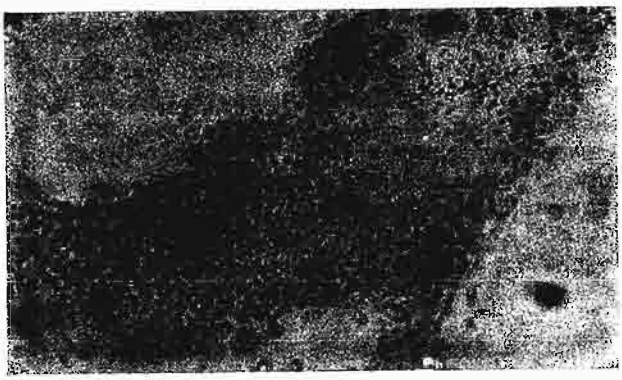


Fig. 1. The autoradiography shows that the tritiated acetamide incorporates mainly in the part of facet-forming region of eye disc.

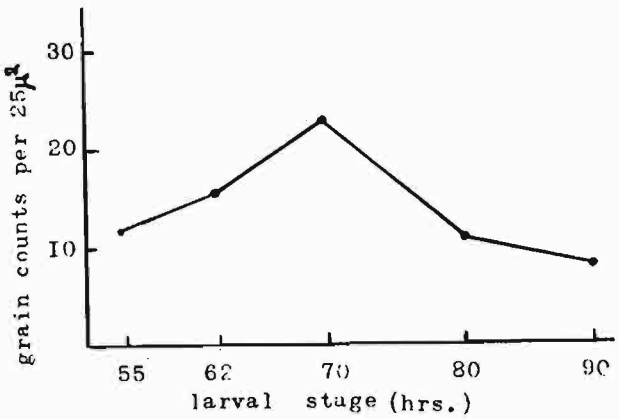


Fig. 2. Incorporation of tritiated acetamide into eye discs at different larval stages. The larvae treated with 5.64 uCi/g of ^{3}H-acetamide for 1 hour. The grain counts per 25u (taken as unit area) at the facet-forming region of eye disc.

Carver, J. E. University of Kentucky, Lexington, Kentucky, Ecological-Genetic study of South Amherst Drosophila populations.

In order to evaluate the influence of various ecological parameters on the genetic structure of Drosophila populations, studies were begun with Dr. P. T. Ives to determine whether or not Drosophila melanogaster (and other species) are continuous in the South Amherst, Massachusetts locale. If the flies do overwinter, an a priori expectation is that the process involves a high degree of genetic adaptation which may be reflected by a difference in frequency, viability, and variance values for drastic heterozygotes in overwintering versus peak summer-fall populations. Results thus far have revealed that Drosophila algonquin, funebris, putrida and quinaria and Chymomyza amoena over-winter in a non-adult stage in apple orchard soil at temperatures approaching but not reaching 0° C. The frequency of lethal-bearing second chromosomes in D. melanogaster was lower in samples of adults taken from the smaller spring population. Viability and variance analysis revealed an irregular pattern of statistically significant and nonsignificant differences between fall and spring samples. Population genetic analysis of the larger seasonal samples of D. melanogaster afforded by the less severe overwintering conditions of the area, as well as similar studies of the ecology of natural Drosophila populations are being continued at the University of Kentucky.

Perreault, W. J., B. P. Kaufmann and H. Gay. University of Michigan, Ann Arbor, Michigan. A DNA-associated RNA in Drosophila melanogaster adults.

A DNA-associated RNA isolated from pulse-labeled Drosophila melanogaster larvae was shown by Mead (1964) to possess many of the properties of messenger RNA. We have extracted a similar RNA from adult flies that had been labeled for only a short period of time with P^{32}.

Nucleic acids were isolated by Mead's procedure from five-gram lots of unlabeled adult Swedish b flies, and subjected to hydrolysis and separation of mononucleotides by the Katz and Comb (1963) technique. These procedures afforded the data contained in Table 1.

Table 1. Nucleotide analysis of RNA isolated from unlabeled XX, XY and unseparated (mixed) Drosophila melanogaster adults.

Source	N	AMP	UMP	GMP	CMP	A+U/G+C
DNA-associated RNA						
Mixed	4	28.2±0.3	28.5±0.6	24.2±0.8	19.1±0.7	1.31
XY Male	2	28.2±0.1	28.6±0.2	24.5±0.5	18.6±0.2	1.32
XX Female	4	28.2±0.2	28.4±0.4	24.7±0.2	18.7±0.2	1.30
Microsomal RNA						
Mixed	3	28.7±0.5	30.5±0.6	24.7±0.2	16.1±0.4	1.45
XY Male	2	28.6±0.6	30.6±1.1	23.9±0.3	16.9±0.7	1.45
XX Female	4	28.5±0.4	30.1±0.2	24.8±0.3	16.6±0.4	1.41

These experiments were designed to see if the Y chromosomes has any qualitative effect on the composition of the DNA-associated RNA. Microsomal RNA's were included as a control since they would not be expected to vary within a given species. The A+U/G+C ratios of DNA-associated RNA are nearly identical in the karyotypes analyzed. The nucleotide ratios obtained from microsomal RNA differ slightly but significantly from those of the DNA-associated RNA at the 5% level of confidence. Because of the slight difference in nucleotide composition between microsomal and DNA-associated RNA we attempted to determine whether the DNA-associated RNA might be an artifact resulting from binding of microsomal RNA to DNA during the isolation procedures. To investigate this possibility we isolated DNA-associated RNA from 5 grams of flies and included in the isolation medium 375 µg of P^{32}-microsomal RNA. Upon recovery of the DNA-associated RNA, only 2.4 µg out of a total yield of 270 µg carried the label. This results suggests that a small part of the DNA-associated RNA may be derived as an artifact of isolation, but that the major portion is not.

To incorporate label into the DNA-associated RNA of adults, Sw-b flies were kept in empty half-pint bottles for twelve hours at 25°C., after which a filter paper containing 0.5 mc P^{32}-orthophosphate in 1.0 ml of grape juice was introduced, and the flies were allowed to feed for one hour. The papers were removed, and the flies were frozen for isolation of nucleic acids. As many as one thousand flies may be treated in one bottle, so that 5 mc of P^{32} will suffice for ten thousand flies, or about ten grams wet weight.

An experiment conducted in this fashion demonstrated that the DNA-associated RNA incorporates P^{32} at several times the rate of either microsomal RNA or DNA (Table 2).

Table 2. Incorporation of P^{32} in a one-hour-feeding experiment.

	O.D. units* (260 mµ)	CPM	CPM/O.D. unit (Specific Activity)
DNA-associated RNA	6.70	16,540	2,469
Microsomal RNA	6.40	3,490	545
DNA	9.80	1,700	173

* One O.D. unit equals 45 µg nucleic acid/ml.

That the label actually resides in the RNA mononucleotides was demonstrated by an experiment in which the mononucleotides were separated, and optical density and radioactivity were plotted on the same set of axes (Figure 1). In this experiment 86% of the radioactivity was recovered within the optical density peaks of the mononucleotides. The nucleotide composition of this DNA-associated RNA from labeled flies is not significantly different from that given in the upper part of Table 1.

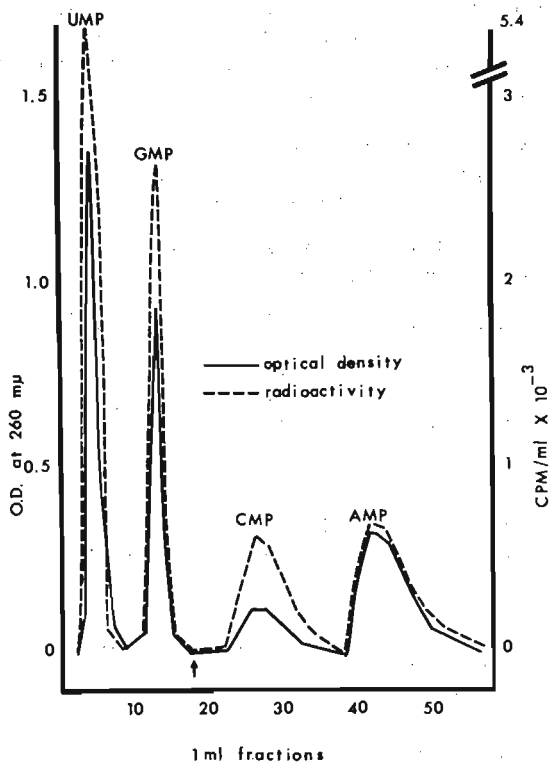


Figure 1. Separation of P^{32} labeled mononucleotides. UMP and GMP were eluted from Dowex 50 (H^+). At fraction 18 (see arrow) effluent was transferred to a Dowex 1 column and CMP and AMP collected.

The administration of actinomycin D (100 ug per filter paper) either fifteen minutes before addition of P^{32}, or concurrent with it, resulted in ca. 13-15% inhibition of P^{32} incorporation (Table 3). It is not clear whether this inhibition is due to interference with transcription, or simply a poisoning effect on the overall metabolism of the treated flies. In Table 3, experiment 1 involved the simultaneous administration of actinomycin D and P^{32} for one hour without any prior treatment, whereas experiments 2 and 3 featured a fifteen minute preliminary exposure of the dehydrated flies, namely, to AMD in the treated series and to water in the controls. The reduction in total P^{32} incorporation in those control flies subjected to the fifteen-minute pretreatment (experiments 2 and 3) indicates that dehydrated flies do considerable drinking during the first quarter hour that fluid is available. This fact should allow studies on adults in which the period utilized for incorporation of isotope is much less than one hour.

Table 3. The effect of actinomycin D on P^{32} incorporation.

	Treated with actinomycin D			Controls without actinomycin D			
Experiment	O.D.	CPM	Specific Activity	O.D.	CPM	Specific Activity	% Inhibition
1	12.3	46,310	3,764	11.4	61,220	5,347	14.5
2	9.0	15,350	1,696	6.2	15,460	2,493	14.7
3	12.5	20,840	1,669	10.2	23,857	2,150	12.9

We have concluded that adult D. melanogaster flies contain a species of RNA which is bound to DNA in an unknown fashion. This RNA is distinguished by a characteristic nucleotide composition and the rapid uptake of P^{32}. It seems probable that at least some of this RNA is mRNA, and that by further refinements of technique an adult messenger RNA fraction may be isolated.

References: Mead, C.G., J. Biol. Chem. 239:550 (1964)
Katz, S. and D. G. Comb, J. Biol. Chem. 238:3065 (1963)

(Supported by PHS Grant GM-10499)

Colot, Hildur[1], W. J. Perreault[2] and H. Gay. Carnegie Institution of Washington and University of Michigan, Ann Arbor, Michigan. The nucleotide composition of the DNA-associated RNA of Drosophila melanogaster larvae.

A type of RNA that is complexed with DNA in a ratio of 1:2 has been isolated from Drosophila melanogaster (Mead, 1964; Perreault, 1966; see also Perreault, Kaufmann and Gay, this issue of D.I.S.). The rapid uptake of P^{32} by this DNA-associated RNA suggests that at least some of it may be messenger (mRNA). Our previously reported data for the nucleotide composition of adult DNA-associated RNA indicated that it is somewhat different from DNA. The present series of experiments, using Swedish-b flies, was carried out to determine whether the larval DNA-associated RNA is similar to that of adults. The results presented in the accompanying table show that it is not, and reveal moreover that its nucleotide composition is quite different from the base composition of larval and adult DNA.

Table 1. Composition of larval and adult DNA-associated RNA.

	N	A(AMP)	T(UMP)	G(GMP)	C(CMP)	$\frac{A + T(U)}{G + C}$
DNA-associated RNA						
Larvae	9	26.7±0.5	27.4±0.4	25.1±0.5	20.6±0.4	1.18
Adults	2	28.9±0.2	29.9±0.4	22.6±0.0	18.7±0.2	1.42
Adults*	10	28.3±0.1	28.6±0.2	24.5±0.3	18.6±0.3	1.32
DNA						
Larvae	4	28.8±0.7	32.7±0.3	19.1±0.1	19.4±0.8	1.60
Adults*	88	29.3±0.4	31.2±0.4	19.5±0.3	20.0±0.3	1.53

*Data from experiments of W. J. Perreault (1966).

These results support the point of view that part of the DNA-associated RNA may represent messenger RNA; the difference between larval and adult forms might be a manifestation of developmental differences in the types of messengers being synthesized. Since it is still not known what proportion of the total DNA-associated RNA has the characteristics of messenger RNA, or whether some of it may be preformed structural or regulatory RNA, further work is in progress to determine the probable molecular heterogeneity of this class of RNA and the nature of its binding to DNA.

References: Mead, C.G., J. Biol. Chem., 239:550 (1964)
Perreault, W. J. (Reported by H. Gay in Carnegie Institution of Washington Yearbook, 65:585 (1966))

1. Supported by NSF Undergraduate Research Participation Grant to the Department of Zoology, University of Michigan.
2. Supported by USPHS Grant GM-10499.

Doane, W. W. Yale University, New Haven, Connecticut. Observations relating to duplications in the Amylase region of D. melanogaster.

I have data relevant to Bahn's contention (DIS 42: 84) that major isoamylase bands in homozygous Amy strains are indicative of gene duplications. A bennett population cage was set up in 1964 with 50 pairs of flies of the genotype Amy^1 $adp^{60}/Amy^{2,6}$ +adp. At F_{45}, the sample taken from the cage (N = 769) contained 5 exceptional phenotypes indicative of crossing-over within the Amy region. They were: $Amy^{1,6}$ +adp (2 females; 2 males) and Amy^6 $+^{adp}$ (1 female).

Bahn indicated the importance of distinguishing between stocks of independent origin which possess identical electrophoretic patterns. This becomes more evident from findings (Doane, in press) that amylases with the same mobility, but from different strains, may differ in their degree of sensitivity to heat and to various α-amylase inhibitors. Thus, homozygotes producing multiple bands may reflect duplications of the Amy region, but amylases with the same mobility from different strains need not represent the same duplication. (Supported by N.S.F. grant GB 1718.)

Thompson, J. N., Jr. University of Oklahoma, Norman, Oklahoma. Recessive mutants in a wild D. melanogaster population.

In a recent series of tests (summer, 1966) an attempt was made to analyze the frequency with which mutants causing morphological changes are carried in the wild populations of Drosophila melanogaster in and near Marshall County, Oklahoma. Special attention was paid to mutants affecting the wings, bristles, and body and eye color.

Two types of tests for mutations were employed. The first, denoted as "Section 1", utilized wild females which laid eggs in single vials. The offspring of these vials were then mated in ten single-pair matings. The second filial generation was counted and classified. Through this procedure, a recessive mutant would have a high probability of becoming homozygous and identifiable in the second generation. The second type of test, denoted as "Section 2", was similar to the first, except that wild males were trapped and mated to virgin females collected as larvae.

From successful tests of 82 original wild parents, 38 distinguishable mutants were recovered. Offspring in 11 other vials were killed by mold growth. Each successful pair of original parents carried at least one recoverable, visible mutant, and some carried as many as eight. Tables 1 and 2 indicate the number of individuals (F_2 offspring) carrying the mutant as well as the number of different tests (parents) it occurred in. A certain variably-expressed bristle mutant was found to affect approximately 97% of the sample flies. Counting each different occurrence of a mutant separately, 249% of the tested females in Section 1 and 206% of the original parent-pairs in Section 2 carried a recessive mutant. It should be noted that this only includes morphological mutations, with the exception of two affecting fertility and viability. It is also important to remember that they were concentrated in only three morphological areas. Although these are very representative and important characters, other mutations could have occurred which, because of their nature, were not recognized. Thus, the number of mutants reported here, though rather large, is almost undoubtedly only a small number of the actual existing mutants carried by the original parents and by the population at large.

A brief comparison of these data with those of earlier workers and several possible explanations of this phenomenon are included in the full paper to be published in the Proceedings of the Oklahoma Academy of Science, 47(1966): in press.

Table 1

Occurrence of Mutants: Section 1

Mutant	No. of sets involved	No. of indiv. involved
A. Wild-type (normal)	77	20,353
B. Bristles		
1. Irregularly stubbled	65	4,956
2. "Stubble"-like	2	327
3. "Singed"-like (?)	1	1
C. Color		
1. Orange eye color	5	55
2. Brown-streaked thorax	5	52
3. Yellow body	3	143
4. Brown body	3	61
5. Light red eye color	2	20
6. Red-brown eye color	1	46
7. "Plum"-like eye color	1	35
D. Wings		
1. Bent wing base	19	138
2. Fragments of extra veins present	12	106
3. "Dichete"-like wings	8	88
4. Notched wings	5	22
5. Vein V not touching wing margin	4	15
6. U-shaped wing notch	3	8
7. Wavy wings	3	indet.

Table 1 continued:

Mutant	No. of sets involved	No. of indiv. involved
8. Small, almost opaque, wrinkled wings	2	35
9. Vein LII broken	1	10
10. Folded	1	7
11. Thickened veins	1	14
12. Eave-like wings	1	6
13. Held out with rough veins	1	14
14. Vein LII forked	1	13
15. "Miniature"-like wings	1	20
16. Posterior crossvein half present	1	32
17. Added crossvein	1	4
18. Ballooned wings	1	5
E. Miscellaneous		
1. Abnormal abdominal segmentation	9	87
2. Sterility mutant	2	--*
3. Low viability factor	1	--*
4. Rough eye	1	4
F. Others (unsuccessful)	10	--

*These mutants are functions of the set, not of individuals.

Table 2

Occurrence of Mutants: Section 2

Mutant	No. of sets involved	No. of indiv. involved
A. Wild-type (normal)	16	2,678
B. Bristles		
1. Irregularly stubbled	14	927
C. Color		
1. Brown eye color*	1	21
2. Orange eye color	1	12
D. Wings		
1. Bent wing base	4	12
2. Fragments of extra veins present	3	20
3. Vein LII broken	1	15
4. Ballooned wings	1	8
5. "Rudimentary"-like wings*	1	6
6. Nicked wing tips*	1	5
7. Vein V not touching wing margin	1	4
E. Miscellaneous		
1. Abnormal abdominal segmentation	3	10
F. Others (unsuccessful)	1	--

*These mutants did not occur in Section 1.

Staub, M. Zoologisches Institut der Universität Zürich, Switzerland.

Larval stages dispensible for the development of giant chromosomes.

Anterior halves of 6 h old embryos (25°C) were transplanted into the abdomen of adult females (method E. Hadorn). After culturing time of 10-14 days, development of salivary gland cells was observed. Chromosomes can attain maximal size or even supergiant dimensions. Thus "larval life" is not required for the process of differentiation and polytaenisation.

Burnet, Barrie. University of Sheffield, England. The effect of culture medium on the composition of the free amino acid pool of Drosophila larvae.

Quantitative analyses of the composition of the pool of free ninhydrin-positive substances in larvae of the tu bw; st su-tu strain of D. melanogaster show that nutritional factors have a considerable influence on the levels of a number of components. Data are summarised in the table. Synchronised larvae cultured at 25°C were used in all cases. Non-germ-free first instar larvae were innoculated onto a standard live yeast-oatmeal-molasses medium, and germ-free larvae onto aseptic defined medium for which details are given by Sang, J. H., and B. Burnet, Genetics 48: 235-253, 1963. Amino acids were extracted in methanol at -30°C. following the procedure of Chen and Hanimann., Z. Naturforsch., 20b: 307-312, 1965, and estimated with an EEL automatic amino acid analyser.

Larvae obtain the bulk of their essential dietary amino acids from yeast protein when cultured on yeast medium, whereas bovine casein is the sole dietary source for larvae cultured on synthetic medium. The data show differences in relative composition, consistent in direction, affecting Tau., Urea, Meth. sulfox., Asp. a., Prol., Gly., Meth., Ileu, β-Ala., Orn., Ammonia, and Arg., which presumably reflect differences in the amino acid composition of the respective larval diets. More comprehensive data with experimental methods will be reported in detail elsewhere.

	YEAST MEDIUM		CASEIN MEDIUM	
LARVAL INSTAR:	second	third	second	third
Phosphoserine + cysteic acid	0.41	0.26	0.61	0.55
Glycerophosphoethanolamine + Tyrosine phosphate	3.79	9.12	3.22	13.9
Phosphoethanolamine	5.20	3.91	4.91	3.58
Taurine	1.74	1.22	1.20	0.87
Urea	2.46	2.33	trace	1.74
Methionine sulfoxide	0.73	0.64	3.66	1.80
Aspartic acid	2.92	3.79	1.75	1.11
Threonine	2.88	2.84	2.78	3.64
Serine	9.16	4.76	3.14	4.10
Asparagine + Glutamine	16.1	17.3	17.1	14.2
Proline	6.82	7.54	14.6	26.5
Glutamic acid	6.76	7.23	6.38	5.60
Glycine	4.45	3.79	2.76	2.88
α-Alanine	13.0	10.5	15.4	4.10
Valine	2.47	1.36	1.74	1.34
Cystathionine	0.13	0.18	0.23	0.20
Methionine	0.38	0.25	1.61	0.46
Isoleucine	0.87	0.46	0.45	0.28
Leucine	1.20	0.84	1.20	0.67
Tyrosine	3.40	2.25	3.07	3.34
Phenylalanine	0.36	0.21	0.31	0.34
β-Alanine	1.11	1.02	0.63	0.58
γ-Aminobutyric acid	0.32	0.24	0.31	-
Ornithine	0.53	0.75	trace	trace
Ethanolamine	0.22	0.13	0.27	-
Ammonia	2.18	4.46	1.48	0.84
Lysine	3.95	4.78	3.47	2.22
Histidine	4.65	4.29	7.60	3.33
Tryptophan	0.04	-	-	-
Arginine	1.85	3.61	-	1.93

Table I. Free ninhydrin-positive substances in second and third instar larvae of the tu bw; st su-tu strain. Each value is expressed as a percentage of the total of identified ninhydrin-positive substances recovered.

Barker, J. S. F. and M. C. Carrick.
University of Sydney, Australia. Effects of benzyl benzoate on components of fitness in Drosophila.

In long-term continuous populations of Drosophila maintained in population cages or bottles, infestation by mites is not uncommon, and benzyl benzoate is generally employed as an acaricide. A further component is thus added to the environment of the population which may differentially affect the genotypes or species being studied. Thus, in single-species populations of D. melanogaster (Oregon-R-C) and D. simulans (vermilion) maintained in population bottles, significant differences were found between populations with benzyl benzoate (B^+) and those without (B^-) (Barker, 1967). For D. simulans, mean population numbers were higher for B^+, while for both species, population survival was higher for B^+. These effects were apparently not mediated just through mite control, and have been studied further by measurement of some components of fitness.

Species	% Benzyl benzoate	Viability (%)	Developmental time (days)		Body weight (mg)		Sex ratio (Males/Total)
			♂	♀	♂	♀	
D. melanogaster (Or-R-C)	0	79.25	9.63	9.59	0.855	1.114	0.501
	4	79.64	9.87	9.89	0.858	1.100	0.468
	10	79.86	10.06	10.03	0.834	1.071	0.456
D. simulans (vermilion)	0	63.10	9.66	9.49	0.762	0.953	0.491
	4	69.69	9.86	9.71	0.734	0.911	0.474
	10	66.00	10.00	9.77	0.725	0.900	0.511

Source of variation	d.f.	Mean square					
		Viability	♂ D.T.x10^4	♀ D.T.x10^4	♂ Wt.x10^4	♀ Wt.x10^4	Sex ratio x 10^4
Species	1	1449.02***	11.13	2550.70*	932.25***	2400.47***	19.96
B.B. %	2	22.50	4575.86***	4435.39***	22.59*	66.12**	20.89
Error	30	48.45	255.80	380.80	4.68	9.64	26.12

* $P<0.05$; ** $P<0.01$; *** $P<0.001$.

Sheets of paper towelling (about 2 x 2 inches) were soaked in alcohol (0% benzyl benzoate), a 4% solution of benzyl benzoate in alcohol, or a 10% solution, allowed to dry and placed in vials containing 5 ml of dead yeast fortified medium (medium F of Claringbold and Barker, 1961). Larvae (0-1 hr. old) of Oregon-R-C and vermilion were collected by the technique of Podger and Barker (1966) and 400 put into each vial, using a predetermined random order of initiation over the treatment combinations. Seven vials were set up for each treatment with Oregon-R-C, but only 5 for vermilion (0%), and 4 each for vermilion (4% and 10%), because of a shortage of vermilion larvae. Emerging adults were counted and weighed (males and females separately) twice daily. The averages for each treatment combination, and analyses of variance are shown in the table. The interaction was not significant in any analysis, and has been pooled into the error. Increasing concentration of benzyl benzoate has significantly increased developmental time and decreased body weight for both species.

The larvae in this experiment were not exposed to severe crowding (evidenced by the viability and average body weight), and the effects of benzyl benzoate may be more marked at higher levels of crowding. Nevertheless, the differences in populations of these two strains between those with benzyl benzoate and those without (Barker, 1967), could be explained in terms of the extended developmental time, although other components of fitness (e.g. fecundity) may also be affected.(Work supported by Australian Research Grants Committee.)

References: Barker, J. S. F., 1967, Evolution, 21:606-619; Claringbold, P. J. and J. S. F. Barker, 1961, J. Theoret. Biol., 1:190-203; Podger, R. N. and J. S. F. Barker, 1966, D.I.S., 41:195.

Burdette, W. J. and J. S. Yoon. The University of Texas M. D. Anderson Hospital and Tumor Institute, Houston, Texas. Results of treatment with two strains of Rous-sarcoma virus.

Recently we were able to demonstrate that administration of Rous-sarcoma virus was associated with an increased incidence of mutations, chromosomal aberrations, and tumors in Drosophila. Both Bryan and Schmidt-Ruppin strains of Rous-sarcoma virus were compared in a subsequent study. Three-tenths ml of a 50-fold dilution of virus in Drosophila Ringer's solution without Ca was mixed with yeast and added to medium on which two-day-old Drosophila larvae were feeding. Germ cells of males remaining on the virus-yeasted food until imaginal stages were tested for genetic damage. Data are summarized in Table I. Results using the Bryan strain are in agreement with previous findings. On the other hand, the results with Schmidt-Ruppin strain differ very little from the control group. Translocations were not recovered in either group treated. Whether the results are due to differences in concentration or other factors such as the Rous associated viruses is the subject of current additional study.

Table I. Results of Treatment with Bryan and Schmidt-Ruppin Strain of Rous-sarcoma

	Control		Bryan		Schmidt-Ruppin	
Aberration	No./Total	Per cent	No./Total	Per cent	No./Total	Per cent
Sex-linked Lethals	0/ 883	0.00	12/1018	1.58	3/ 1088	0.28
Loss of Y	0/2972	0.00	5/3254	0.15	4/ 5889	0.07
Visible Mutants	0/9394	0.00	3/7484	0.04	1/13995	0.01
Translocations	0/ 790	0.00	0/ 914	0.00	0/ 886	0.00
F_1 Sterility	56/ 876	6.39	143/1057	13.53	98/ 984	9.96

Anderson, Wyatt, W. Yale University, New Haven, Connecticut. Elimination of the sex-ratio X chromosome in experimental populations of Drosophila pseudoobscura.

Eight experimental populations of D. pseudoobscura were set up in 1964 from the F_1 progeny of flies collected in nature. Each female from the collections was isolated in a separate bottle. Twenty female and twenty male progeny from each female from a given locality were used to begin the experimental population representing that locality. Thus, the populations had the same chromosomes, and in the same proportions, as did the samples from nature. The frequencies of the sex-ratio chromosome among the founding members of the populations are given below. Almost all of the offspring produced by a male carrying the sex-ratio X chromosome are female. This "meiotic drive" serves to increase the frequency of the sex-ratio chromosome.

Locality	Initial Frequency of Sex-Ratio Chromosomes (in %)
Okanagan, B.C.	0.0
Gunnison, Colo.	12.3
Hayden Creek, Colo.	8.8
Tucson, Ariz.	20.0
Raton, N.M.	16.0
Austin, Texas	19.7
Davis, Texas	14.9
Sonora, Mexico	25.0

After an initial period of about six months at 16° C, the cages were transferred to 25° C. They remained there for one and a half years. In a census of the chromosomes at the end of the one and a half years at 25° C, a special effort was made to score for the sex-ratio X chromosome. Not a single sex-ratio chromosome was found in the samples of 300 chromosomes from each experimental population. In no population was an equilibrium established. These results confirm the outcome of Wallace's (1948; Evolution 2: 189-217) experiment. Wallace found that the sex-ratio chromosome was eliminated from experimental populations of D. pseudoobscura kept at 25° C.

Hunter, Alice S. Centro Experimental de Estudios Superiores, Barquisimeto, Venezuela. Mating preferences of iso-female lines of D. mesophragmatica.

Certain iso-female lines of Drosophila mesophragmatica of Bogotá, Colombia differ from others in that they can be crossed with D. pavani and D. gaucha (Hunter & Hunter '64). Studies were made of several of these lines in order to test for possible isolating mechanisms between the "crossing " and "non-crossing" lines.

Male choice experiments were carried out with virgins of three weeks of age. In order to distinguish the two females, one anterior scutellar bristle was removed from one of them. The three unetherized flies were introduced into a vial at 9 AM and the vials observed till noon. When a pair copulated, the non-copulating female was discarded and the identity of the "selected" or "receptive" female was determined. The results are shown in Table 1.

Table 1

Female line		Copulas	Female line		Copulas	Male line		Chi Square
Cross	3	61	Non	57	16	Non	54	26.29
Cross	3	46	Non	57	18	Cross	3	12.25
Cross	3	30	Cross	45	21	Non	54	1.58
Cross	3	28	Non	53	20	Non	54	1.33
Cross	3	33	Non	60	24	Non	53	1.42
Cross	3	26	Non	60	27	Non	60	0.02
Cross	22	38	Non	53	21	Non	53	4.89
Cross	22	41	Non	53	40	Cross	3	0.01
Cross	22	20	Non	54	23	Cross	22	0.21
Cross	44	34	Non	53	17	Cross	3	5.78
Cross	44	23	Non	53	25	Cross	22	0.08
Cross	44	59	Non	60	29	Non	60	10.22
Cross	45	25	Non	54	27	Cross	44	0.08
Cross	45	32	Non	54	25	Non	57	0.86
Cross	22	22	Cross	45	29	Cross	45	0.96

It can be seen that in certain combinations there are preferences. However they are not simply preferences of "crossing" males for "crossing" females and vice versa nor are they simply homogamic preferences within a line. Female choice experiments were also carried out but no significant differences suggestive of preferential mating were found. Further experiments are under way in order to try to clarify these results.

Reference: Hunter, Alice S. and Robert A. Hunter, 1964. The mesophragmatica species group of Drosophila in Colombia. Ann. Entomol. Soc. Amer. 57:732-736.

Minamori, S. Hiroshima University, Hiroshima, Japan. A killing agent found in a natural population of D. melanogaster.

Some flies having a killing agent were found in sampled flies of D. melanogaster from a natural population in 1964. The number of their offspring was reduced distinctly, and the distorted segregation of second chromosomes in the offspring was observed by using dominant marker genes.

The killing agent was transmitted from both females and males to their progenies. The agent had been maintained by a carrier strain, though its all original chromosomes were substituted with marked chromosomes. However, most strains, having the killing agent originally, had lost it within one year. The killing agent seemed to have been transmitted by copulation from males to females and by contact of larvae. The development or multification of the agent at 30° C was faster than that at 20° C. Flies under two days old could not transmit the agent to their progenies. Such experimental results indicate that the killing agent might be an infective microorganism for flies.

The survival rates of flies carrying Cy, L, Pm or bw^D chromosomes respectively to the killing agent were observed to be different. The similar experiment was performed by using 184 second chromosomes isolated from a natural population. The survival rate of flies carrying chromosomes derived from the carrier strain was higher than that of flies having no infection. Flies carrying a lethal chromosome reduced the survival rate more than flies carrying a quasinormal chromosome .

Khan, A. H. Atomic Energy Agricultural Research Centre, Tandojam, West Pakistan.

The mutagenic effect of Nitosoguanidine in Drosophila.

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

An increase in the concentration of Nitrosoguanidine from 0.05% to 0.1% double the yield of complete sex-linked recessive lethals (Table 1). An indication of delay in the mutagenic effect of Nitrosoguanidine is seen in table 1, where 7.7% of the tested non-lethal F_2 cultures show F-1 lethal-mosaicism after 0.1% of Nitrosoguanidine treatment, compared with 3.6% from the control; the corresponding F-3 lethal frequencies are 5.8% for Nitrosoguanidine and 0.36% for the control.

Table 1: Complete and mosaic sex-linked recessive lethal mutation frequencies in Drosophila males after adult feeding treatment with Nitrosoguanidine.

	Concentration (%)	Control	0.05	0.1
	Duration of treatment (hrs.)	24	24	24
	Survival (%)	100	100	97
	Number of males examined	56	50	70
F-2	Average no. chromosomes examined/male	9	8	7
	Number of chromosomes examined.	510	400	490
	Number of lethal chromosomes	1	14 (from 11 ♂♂)	35 (from 27 ♂♂)
	Complete lethals (%)	0.2	3.5	7.1
	No. non-lethal F-2 cultures examined.	55 (arising from 55♂♂)		65 (arising from 65 ♂♂)
	Average no. female examined/ non-lethal F-2 cultures	10		9
F-3	No. non-lethal F-2 cultures yielding at least one lethal in F-3 set	2		5
	Cultures showing mosaicism (%)	3.6		7.7
	Total no. F-2 females examined	547		610
	No. of lethal bearing F-2 females.	2		35
	Lethals in F-3 (%)	0.36		5.8

This work was done at the Dept. of Genetics, University of Cambridge.

Schwinck, Ilse. University of Connecticut, Storrs, Connecticut.

Phenogenetic studies on control of drosopterin synthesis.

A wild type-like phenocopy of rosy or maroon-like genotype can be caused by phenylalanine, the mechanism is still unknown. Furthermore, rosy-like phenocopies of wild-type genotype could be obtained by feeding the xanthine dehydrogenase inhibitor 4-hydroxypyrazolo(3,4-d)pyrimidine to larvae or by exposing late pupal stages to this inhibitor in the "free pupae incubation" procedure (latter method described for the phenylalanine studies, Schwinck, I., Zeitschr. f. Naturforschung 20b, p. 322-326, 1965). The combination of feeding and incubation experiments and incubation with mixed solutions of various concentrations revealed an antagonism of the phenylalanine stimulation and XDH inhibitor as well as an antagonism of phenylalanine and hypoxanthine; the effect on drosopterin synthesis was determined in single head extractions spectrophotometrically.

A model discussing the indirect control of drosopterin synthesis by accumulated xanthine dehydrogenase substrates is proposed as unifying explanation for the pleiotropic pattern of the rosy and maroon-like mutants and their previously described plasticity in phenotypic expression, as revealed in the transplantation and phenocopy studies (Schwinck, I., Zoolog. Anz. 30th Suppl.Bd., p. 382-390, 1966).

Supported by PHS Grant GM-10256 and a Grant from the University of Connecticut Research Foundation.

Chen, P.S. and H. Weideli. Zoologisches Institut der Universität, Zürich, Switzerland. Free ninhydrin-positive components and proteins in the hemolymph of Drosophila larvae infected by Pseudeucoila bochei Weld.

The relationship of the parasitic wasp Pseudeucoila bochei to D. melanogaster has been extensively investigated in this laboratory (Jenni 1951, Schlegel-Oprecht 1953, Walker 1959, Meier-Grassmann 1962). It was found that there are Drosophila strains which are able to form a pigmented capsule around the parasitic embryo within the larval body cavity, and the encapsuled parasite subsequently dies. On the other hand, there are Pseudeucoila strains which can inhibit the process of capsule formation of the host larva. Detailed analyses indicated that the defensive reaction is strain-specific with respect to both the host and the endoparasite and appears to be under genic control. In order to get further information about this phenomenon we carried out a preliminary investigation of the free ninhydrin-positive components and proteins in the hemolymph of Drosophila larvae infected by Pseudeucoila. For comparison the hemolymph composition of non-infected larvae from parallel cultures was also analyzed. Drosophila larvae of the strain Hindelbank were raised on standard medium at 25°C and subjected to infection by Pseudeucoila (strain Erlenbach) at the age of 56-60 hrs. after oviposition. Hemolymph samples were taken at 10 hr. intervals until the host larvae reached puparium formation. The technique of 2-dimensional paper chromatography was used for the analysis of amino acids and peptides, and disc electrophoresis for the separation of proteins. We found that the total concentration of free ninhydrin-positive components of the infected larvae at 72 hrs. of age amounts to 10% higher than that of non-infected controls. According to Walker (1959) this corresponds to the time of the beginning of capsule formation through the aggregation of lamellocytes around the parasitic embryo. At 88 hrs., when the deposition of melanin in the aggregated lamellocytes takes place, the value of the infected larvae is even 25% higher. A total of 26 ninhydrin-positive substances was identified on 2-dimensional paper chromatograms. Among these phosphoethanolamine maintains a consistently higher concentration in the infected larvae than in the controls during the period from 60 to 80 hrs. The same is true for ornithine at 70 hrs. of age. On the other hand, the content of -alanine and one acidic peptide appears to be significantly reduced. Using disc electrophoresis at least 10 protein fractions migrating in the anodal direction could be separated. Significant differences between infected and normal larvae were detected in the gel region about 8 mm from the origin. Based on the staining intensity of the protein bands, one fraction (No.5) appears to be reduced in the infected larvae aged 60 to 80 hrs., whereas the opposite is true for another fraction (No.8).

In order to test to what extent the hemolymph of the host larva is related to the process of capsule formation, the following blood transfusion experiment was designed. As mentioned above, Drosophila larvae of the strain Hindelbank were infected by Pseudeucoila at 56-60 hrs. after egg-laying. According to Walker (1959) in this strain 97.6% of the infected larvae form a capsule around the parasitic embryo. About 10 hrs. after infection hemolymph was collected, and 0.1 to 0.2 µl of this was injected into infected Drosophila larvae of the strain Luxor which, in contrast to the donor strain, has an extremely low rate of capsule formation (2.9%). After injection the larvae were kept under normal culture conditions until the time of pupation, and the number of individuals which formed a pigmented capsule was determined. In two series of experiments we found that the rates of capsule formation were 11% and 29% higher in the injected larvae compared to those which received no hemolymph of the strain Hindelbank. It seems that some components in the larval hemolymph are probably essential to initiate the defensive reaction. Since we have so far carried out only a limited number of experiments, this result can not yet be considered as conclusive. It would also be of interest to see if the opposite is true by injecting hemolymph from the strain Luxor into infected larvae of the strain Hindelbank.

Up to this time we do not know how the observations reported here are causally related to the infection process. There is, however, no doubt that during the period of capsule formation significant quantitative changes in the chemical composition of the larval hemolymph take place.

Carrick, M. C. and J. S. F. Barker.
University of Sydney, Australia.

Marking of Drosophila eggs by feeding dyes to larvae and adult females.

In our current studies on interspecific competition between D. melanogaster and D. simulans, we intend to determine the effect of various factors on the fecundity of these species, both in single-species and mixed populations. As their eggs are indistinguishable, the latter would be greatly simplified if eggs of one species could be marked by a dye sufficiently to distinguish them on macroscopic examination. Using D. melanogaster Oregon-R-C and the vermilion mutant of D. simulans, 15 dyes have been tested.
Larval feeding: The dye was blended with hot liquid dead yeast fortified medium. Twenty pairs of adults were left in each bottle for 24 hours. Eggs produced by about 100 progeny from each bottle were collected daily and examined microscopically for at least 4 days post-eclosion.
Adult feeding: For each bottle, dye was added to a mixture of 30 ml cornmeal-agar-treacle medium, 2 ml treacle and 0.5 g dead yeast. Flies (<12 hrs old) were allowed to feed for 12 hrs. Eggs produced over at least the next 4 days were collected and examined daily.

Eggs were examined both whole and squashed. For all ether-soluble dyes, they were soaked in ether. For rhodamine B and fluoroscein, eggs were examined under ultra-violet light. Samples of females were examined for ovary pigmentation.

None of the dyes were apparently grossly toxic with adult feeding. With larval feeding, seven were harmful under some conditions. Where many dead larvae were observed, either toxicity or unpalatability may be involved.

No ovary or egg pigmentation, nor apparent toxicity with larval feeding was observed for the following dyes:

Ether soluble: Calco Oil Red Z-1700 - 0.1 and 1.0% w/v concentrations.
Water soluble: At 0.05 and 0.1% w/v concentrations - Rhodamine B, Fluoroscein, Congo Red, Orcein, Methyl Orange.
At 1.5 and 3.0 ml saturated solution in 30 ml medium - Chrysoidin.

No ovary or egg pigmentation was observed for the ether-soluble dyes Irgacet Scarlet GL and Irgacet Scarlet RL, at 0.1 and 1.0% w/v concentrations. The former was completely toxic to larvae at the higher concentration, while the latter (at 1%) delayed the onset of egg production by D. simulans by 2 days.

For the water soluble dyes, Methylene Blue, Nile Blue and Neutral Red at 0.05 and 0.1%, no egg pigmentation was observed, although all (D. simulans only for Neutral Red) showed ovary pigmentation with adult feeding and with larval feeding at 0.05% for the last two. For these two dyes at 1% and Methylene Blue at both concentrations, no adult females were obtained.

Three ether soluble dyes showed some egg pigmentation. Irgacet Orange GR gave both ovary and egg pigmentation when fed to larvae at 1.0 and 2.0%. Egg pigmentation ceased after 4 days. At 2.0%, emergence of Oregon-R-C was delayed by about 2 days, while no D. simulans were obtained. No pigmentation was observed with adult feeding (1.0%). Sudan III gave both ovary and egg pigmentation with both adult (0.1 and 1.0%) and larval (1.0 and 2.0%) feeding. The proportion of pigmented eggs was highest in the first eggs laid and decreased to zero after 3 days. At 2.0% larval feeding, the numbers of eclosing adults were reduced, and the onset of egg production by D. simulans was delayed by 2 days. Sudan Black fed to larvae (1.0 and 2.0%) also delayed egg production by D. simulans, and although ovaries of both species were pigmented, no pigmented eggs were laid. With adult feeding (1.0%), ovaries of both species again were pigmented, but Oregon-R-C produced pigmented eggs. The proportion was highest in the first eggs laid, and decreased to zero after 4 days.

However, for these three dyes, egg pigmentation was not consistent (only a proportion of early eggs were marked), and the intensity of pigmentation was never great enough to be detectable macroscopically. (Work supported by Australian Research Grants Committee.)

Berendes, H. D. Max Planck Institut f. Biologie, Abt. Beermann, Tübingen, Germany. Electron microscopical mapping of giant chromosomes.

Electron microscopical studies of selected regions of giant chromosomes which have been sectioned parallel to the chromosome axis permit the construction of chromosome maps from which the exact number of bands can be determined. The map made by light-microscopical investigation can be improved by the location of additional submicroscopic bands. Moreover, morphological changes in the submicroscopic structure of active regions (puffs and Balbiani rings) can be investigated at different phases of their active period.

The method for sectioning squashed salivary gland chromosomes developed by Sorsa and Sorsa (1) was modified to permit the selection of particular chromosome regions to be sectioned. Isolated glands are fixed in 6% glutaraldehyde, buffered 10% neutral formaldehyde, or Carnov's fixative for 10-20 min. and squashed on siliconized slides in 40% acetic acid or in the fixative. Directly after squashing, the coverglass is removed on dry ice and the preparations post-fixed in a methanol-10% neutral formaldehyde series (M:F, 1:0, 9:1, 6:4, 4:6, 1:9, 10 min. each step). Subsequently, the preparations are stained in filtered heamalum Mayer (Merck) for 10 min. and dehydrated in an ethanol series of which the last steps (80%-100%) are saturated with uranyl acetate. Capsules filled with Epon are placed on top of a well spread nucleus or group of nuclei selected light-microscopically and marked on the underside of the slide. After polymerization and removal of the capsules from the slide by dry ice, the capsules are placed in a holder (fig. 1) which is adapted to a Leitz Ortholux light-microscope. The chromosome region to be sectioned can now be selected (fig. 2), marked on the plastic and the pyramid can be made according to these marks.

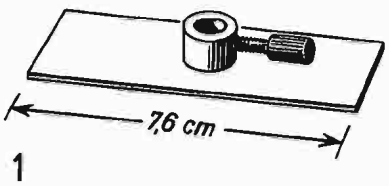


Fig. 1. Holder for capsules adapted to the Leitz Ortholux.

Fig. 2 Oil-immersion image of chromosomes on top of the capsule after trimming the pyramid. The X chromosome is indicated.

Fig. 3 Low power micrograph of the tip of a female X chromosome fixed with Carnoy's fixative. The white region is indicated. The bands 1A5,6; 1B3,4; 1E3,4 clearly demonstrate their compact singular nature.

This method was used to make a map of the banding pattern of section 1-4 of the female X chromosome of D. melanogaster (fig. 3). The pattern of bands is the same with all fixatives used. From 35 series of sections the maximum number of bands was determined and mapped. A comparison of this map with the revised map of Bridges (2) revealed characteristic differences. The electron microscopical map contains a maximum of 85 bands from X-1A to X-3F, whereas the map of Bridges contains 136 bands in this section. This discrepancy can partly be explained by the absence of double bands in the EM-map in 36 of the 38 band pairs indicated by Bridges (see Fig. 3). The remaining difference of 33 bands consists mainly of very thin bands on the map of Bridges.

It is evident that the reduction in the number of bands to about 60% of the number indicated by Bridges may have an important bearing not only on the correlation of known genetic factors with particular bands, but also on the calculation of the mean DNA content per band.

Lit: (1) Sorsa and Sorsa, Chromosoma 22 (1967); (2) Bridges, J. Hered. 29 (1938).

Hotchkiss, Sharon K. and J. K. Lim.
Wisconsin State University, Eau Claire, Wisconsin. Mutagenic specificity of Ethyl methanesulfonate affected by treatment method.

Preliminary experiments were conducted to determine if there is a difference in the mutagenic effects of Ethyl methanesulfonate (EMS) when fed and when injected into Drosophila. 0.025 M EMS in 1% sucrose solution was prepared by adding 0.24ml of EMS (Eastman Kodak) into 100ml of 1% sucrose solution and aerating for a few minutes with a hypodermic syringe. Two-day old males of the constitution $sc^8 \cdot Y \cdot B^S / y^2 w^i ct^6 f$ were injected with approximately 0.16 microliter of the EMS solution. Twenty-four hours after injection, the males were mated individually to two virgin females of the composition $ysc^{S1} ln49 sc^8$; dp bw; st p^p. Males of the stock used in the injection experiment were fed on the EMS solution for 24 hours and were mated individually to two virgin females of the stock used in the injection experiment. The matings in each experiment were kept for four days, after which males were discarded. Upon eclosion of F_1 flies, females were sib-mated for sex-linked recessive lethal detection and F_1 males were mated back to two virgin females of the composition $ysc^{S1} ln49 sc^8$; dp bw; st p^p for detection of translocations II-III, Y-II, Y-III, and Y-II-III.

If 25 or more wild type males were present in the sib-population, without any y w ct f male, a lethal was scored and retested for two generations. The presence of 40 or more flies in the expected classes was used as the criterion in screening for translocations (the actual number ranged from 41 to 156 with a mean of 73.7). The results are summarized in the following table:

Type of Treatment	Sex-linked recessive lethal			Translocation		
	Number tested	Number detected	Per cent	Number tested	Number detected	Per cent
Injection	508	29	5.7%	958	0	0%
Feeding	612	328	53.6%	760	19	2.5%

Of the 19 translocations detected, nine were T II-III and the remaining 10 were TY-III. None involving Y-II and Y-II-III were detected. A random sample of three from the nine T II-III was taken for salivary chromosome examination. All three samples showed reciprocal translocation between the second and third chromosomes, confirming the detection based on the genetic data.

The results indicate a quantitative difference in the effects of EMS when fed and when injected since the frequency of sex-linked recessive lethals induced by feeding was nearly ten times the frequency induced by injection. A qualitative difference, the production of translocations when fed but not when injected, is strongly suggested by these results, however, larger scale experiments will have to be conducted to eliminate the possibility that this, also is a quantitative difference only.

Cohn, R. H. and E. H. Brown. University of Illinois, Urbana, Illinois. The formation of alpha (proteoid) yolk spheres in the oocyte of D. melanogaster.

Developing oocytes of D. melanogaster undergo vitellogenesis during stages 8 through 14 of oogenesis. Alpha yolk spheres (containing protein, carbohydrate and lipid components) begin to accumulate in the ooplasm at stage 8 and beta yolk spheres (containing carbohydrates and lipids, but no protein) begin to accumulate at stage 13 (1).

In Hyalophora cecropia and other saturnid moths, at least some of the yolk sphere proteins are obtained directly from the hemolymph. Blood proteins apparently diffuse through the ovariole sheath and into the intercellular spaces of the follicular epithelium which surrounds the oocyte. When the proteins reach the oolemma they are taken into the oocyte via micropinocytosis, and the resulting vesicles become the proteoid yolk spheres (2, 3, 4).

In order to further elucidate the processes involved in the formation and maturation of the alpha yolk spheres in D. melanogaster, we have made light and electron microscopic observations of oocytes fixed in glutaraldehyde, post-fixed in osmium tetroxide and embedded in Epon.

Light microscopy was used to examine 2 micron, Epon-extracted sections stained with fast green (for proteins), Sudan black B or Nile blue sulfate (for lipids), or the periodic acid/Schiff reagent reaction (for carbohydrates). In stage 8 - 11 oocytes, all alpha spheres stain intensely and homogeneously with fast green and the majority are from 1 to 3 micra in diameter. In addition, however, stage 8, 9 and 10 oocytes contain numerous, tiny alpha spheres (0.2 to 0.5 micron in diameter) which are localized in the peripheral region of the oocyte, directly beneath the columnar follicular epithelium. At stage 12, some of the larger alpha spheres lose their homogeneous appearance and contain variable numbers of fast green positive subunits. In subsequent stages the number of homogeneous alpha spheres decreases and the number of alpha spheres containing subunits increases, and by stage 14 nearly all of the alpha spheres contain proteinaceous subunits. The homogeneous and subunit-containing alpha spheres presumably correspond, respectively, to the $alpha_1$ and $alpha_2$ yolk spheres observed by King, Bentley and Aggarwal (5). King (1) reported that alpha spheres accumulate a carbohydrate component during stages 8 - 12 and then lose their carbohydrate stainability during stages 13 and 14, at which time the carbohydrate stainability of the ooplasm increases. We have found the same relationship to apply to a lipid component of the alpha spheres and the ooplasm. Perhaps the formation of the proteinaceous subunits is related to the passage of lipids and carbohydrates from the yolk spheres into the ooplasm.

Electron microscopic examination of oocytes in stages 8 - 14 reveals that the oolemma is thrown into numerous folds and microvilli. During stages 8 - 10, the oolemma seems to engage in intensive micropinocytotic activity, and we have observed all stages of a sequence which is characteristic of the formation of micropinocytotic vesicles. The smallest vesicles which presumably pinch off from invaginations of the oolemma are about 120 millimicra in diameter and contain dense, granular material which resembles material present in the space between the oocyte and the follicular epithelium. These vesicles increase in size, and at least some of this enlargement seems to involve fusions of vesicles. The larger of these membrane bound vesicles in the periphery of the oocyte correspond to the smallest alpha spheres observed with the light microscope. Prior to stage 12, the alpha spheres contain a homogeneous electron-dense material. The subunits which appear in the alpha spheres during stages 12 - 14 are highly variable in size and number. However, under high magnification each subunit (presumably proteinaceous) exhibits a crystalline lattice having a periodicity of about 65 angstroms.

The oocyte of D. melanogaster thus exhibits the cytological features which have been implicated in the uptake of hemolymph proteins as a mechanism of proteoid yolk formation in several other insects. As a further test of this possibility we have injected adult females with a 1% solution of the semicolloidal vital dye trypan blue, which appears to be taken into insect oocytes via micropinocytosis (6). The ovaries were dissected from injected females at varying intervals and either examined in toto or fixed in glutaraldehyde, embedded in paraffin and sectioned at 6 micra. Within 10 minutes the dye is present in yolk spheres located in the peripheral region of the oocyte, beneath the columnar follicle cells. Similar results were obtained with ovaries immersed in trypan blue in vitro. Only oocytes in stages 8 - 10 incorporate trypan blue into yolk spheres. These are the stages at which electron microscopy

indicated micropinocytotic activity of the oolemma, and it is during stages 10 and 11 that the vitelline membrane is completed (1). Although some dye is evident in the follicular epithelium, its localization is not clear. Ultrastructural studies of ovaries from females injected with ferritin are now in progress and should clarify the route of entry. It therefore appears that the Drosophila oocyte is able to incorporate blood proteins into yolk spheres via micropinocytosis, but conclusive proof requires further investigation. However, this does not rule out the possibility that the alpha yolk spheres also contain proteins synthesized within the ovary.

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Sperlich, D. University of Vienna, Austria. Data on the genetic load in D. subobscura.

A marker strain for the chromosome 0 of D. subobscura was built up by the following way: Va cu ch/+ cu ch - males (Va=Varicose, dominant, homozygous lethal, cu-curly, ch-cherry) were irradiated and crossed to + + + standard females. The heterozygous Va cu ch/ + + + daughters were backcrossed to + cu ch/ + cu ch males and the offspring examined. Those cultures which yielded no recombinants were cytologically analysed. Unfortunately there was no culture with an inversion long enough to prevent crossing-over over the entire chromosome, but a strain (Va cu ch 33) could be found with an X-0 translocation combined with a long inversion on the 0-chromosome (from region 81-98 of the cytological map). Although further irradiation experiments will be carried out for finding a better marker strain the translocation strain was used for a preliminary examination of the genetic load in the chromosome 0 of D. subobscura.

Va cu ch 33 males were crossed to females of the mutant strain Ba (Ba=Bare, dominant, homozygous lethal) and a balanced strain Va cu ch 33/Ba established. Males of a wild Vienna population were crossed to this strain in the following way: P-generation: Va cu ch33/Ba ♀ x Wild♂ ; F_1-generation: Va cu ch33/Ba ♀ x Ba/+ ♂ ; F_2-generation: Va cu ch33/ + ♀ x Ba/+ ♂; F_3-generation: Va cu ch33/Ba : Va cu ch33/+ : Ba/+ : +/+ (1 : 1 : 1 : 1); If there were no wild types in the F_3-offspring (among a total number of 100 flies) the wild chromosome was considered lethal. Among 38 wild chromosomes 12 (32%) proved to be lethal and 2 (5%) to be semi-lethal (less than 5 wild type flies). From the ratio +/+ : Va cu ch33/Ba in the F_3-generation the relative viability of the viable +/+ -homozygotes can be calculated. In a similar way the relative viability of +/+ - or +/1 -heterozygotes can be obtained by intercrossing Va cu ch33/+ females of the F_3-generation of one culture to Ba/+ males of another culture. 24 different interstrain (lethals excluded) and 35 different interstrain crosses were carried out. The relative viabilities of the different genotypes are listed in the table below:

Genotype	$+^A/+^A$ homozygotes	$+^A/+^B$ heterozygotes	+/1 heterozygotes
Rel. viability (+/+ : Va cu ch 33/Ba)	1,1	1,9	1,6

As expected, the viability of +/+ homozygotes is lower than both, the +/+ -and the +/1 -viability. The difference between the latter two classes is not significant (X^2 = 3,4039, p=0,1). There is no evidence that lethal bearing heterozygotes are less viable than lethal free heterozygotes. Further it was found that +/Ba - and 1/Ba - individuals and +/Va cu ch33 - and 1/Va cu ch33 - individuals do not differ very much with respect to viability. The mean number of +/Ba -individuals in the F_3 cultures was 40/per bottle and that of 1/Ba -individuals 36/per bottle. The corresponding number of +/Va cu ch33 and 1/Va cu ch33 was 72/per bottle and 88/per bottle respectively, indicating rather a superiority of lethal heterozygotes than the opposite.

Denell, R. E. and B. H. Judd. University of Texas, Austin, Texas. Segregation distortion in D. melanogaster; the location of stabilizer of SD.

The SD-72 second chromosome in D. melanogaster carries two inversions and at least three genetic elements important to the expression of segregation distortion. This chromosome has a small pericentric inversion and a distal inversion in the right arm which is apparently identical with In(2R)NS. The genetic elements include SD, which is located close to the centromere; activator of SD (Ac(SD)), located 0.5-1.0 units to the right of SD, and stabilizer of SD (St(SD)). In an examination of recombination in Sd-72/cn bw females, Sandler and Hiraizumi (1960, Gen. 45: 1269) found only products of an exchange between the locus of cn and the proximal breakpoint of In(2R)NS. Recombinant chromosomes carrying SD Ac(SD) had invariably lost St(SD), and it was concluded that St(SD) was probably located distal to the bw locus, at the tip of IIR.

In an experiment involving matings of single SD-72/cn bw males to cn bw females, 4 of the 78 males tested gave exceptional offspring with brown eye color. All four of these males gave a high proportion of SD-72/cn bw to cn bw progeny; three of them in addition gave a single brown offspring. The fourth male gave the following progeny: 477 SD-72/cn bw ♂♂, 408 SD-72/cn bw ♀♀, 1 cn bw ♂, 1 cn bw ♀, 34 brown (presumed bw/cn bw) ♂♂, and 32 brown ♀♀. From these latter brown offspring, four lines were established and maintained for about a year in mass culture.

It was noted that the origin and behavior of these presumably bw-bearing chromosomes were consistent with the possibility that they arose by a recombinational event in the SD-72/cn bw parental male, and thus carried the SD locus. Therefore, males from each line were made heterozygous with cn bw and test crossed to cn bw females. The results are given in Table I, with the addition of data characterizing the SD-72 line from which these exceptions originated. k is given as the proportion SD-72 or brown offspring and sex ratio is expressed as the proportion males.

Table I

SD-72/cn bw or bw/cn bw ♂♂ mated singly to cn bw ♀♀

Parental ♂♂	No. ♂♂ Tested	k	Progeny Sex Ratio SD* or bw*	Progeny Sex Ratio cn bw*	No. Progeny
SD-72/cn bw	51	.992	.510	.238	25,807
bw 29-1/cn bw	15	.989	.537	.130	2,026
bw 29-12/cn bw	18	.960	.526	.306	2,771
bw 29-14/cn bw	21	.986	.534	.302	3,029
bw 29-15/cn bw	21	.992	.538	.240	2,983

*These progeny classes are represented by their paternal second chromosome.

The high k value associated with each of the brown lines leaves no doubt that each does indeed carry SD. Further, however, these high k values are indicative of the presence of St(SD), as well as the other two elements of the segregation distortion system. This conclusion is strengthened by an examination of the sex ratios of the progeny classes representing recovery of bw- and cn bw-bearing second chromosomes. With respect to the work of Hiraizumi and Nakazima (1967, Gen. 55:681), these sex ratios are more characteristic of original-SD than of recombinant-SD lines. The presence of St(SD) is further indicated by the distributions of k values for males in each line, given in Figure I. Finally, a cytological examination of the salivary gland chromosomes of bw/cn bw larvae from each line was made, and each bw-bearing chromosome still retains In(2R)NS.

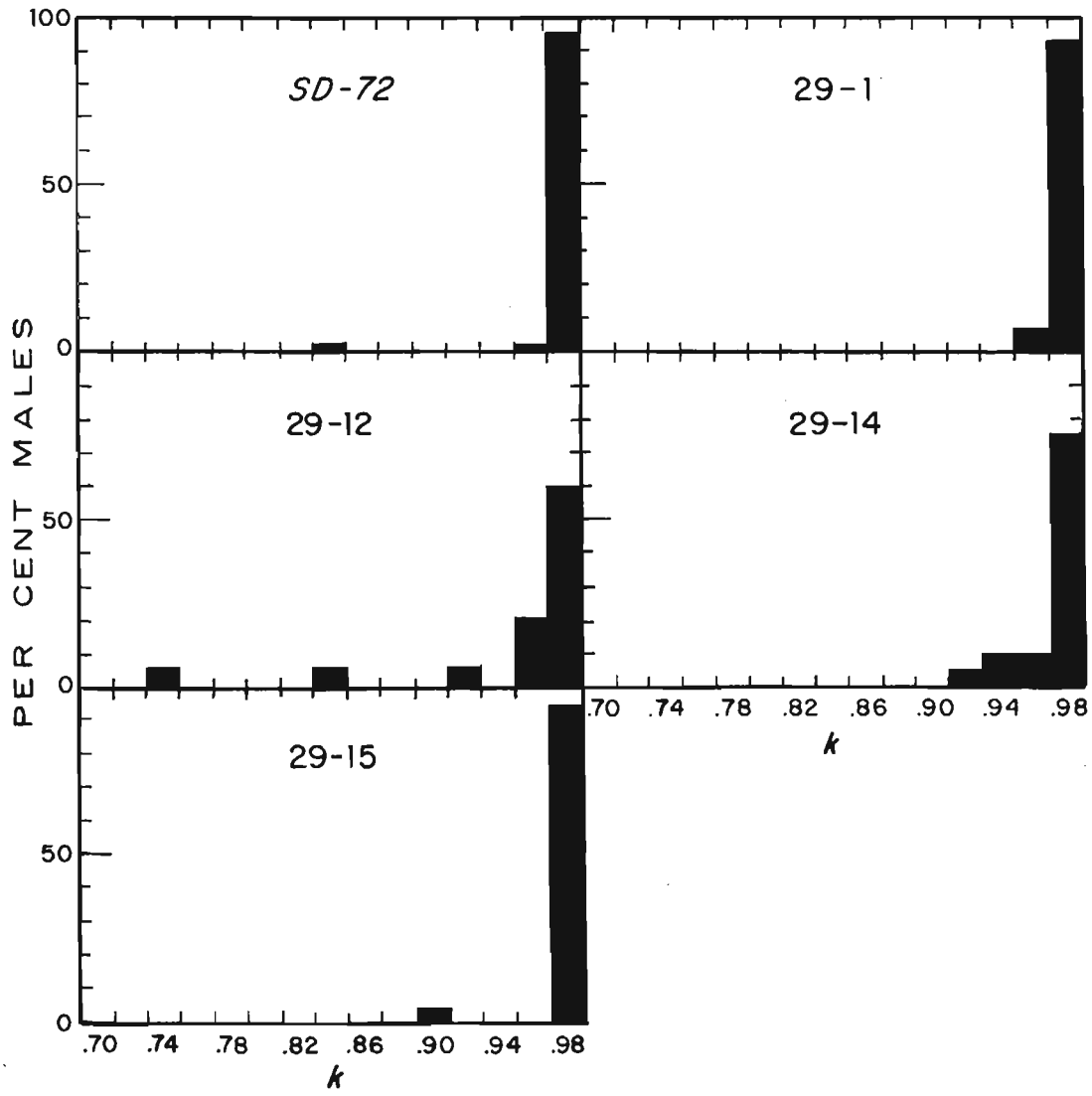
These observations can most satisfactorily be explained by a pre-meiotic recombinational event in the four SD-72/cn bw parental males, yielding brown exceptions either as a cluster or singly. This rate of spontaneous crossing over seems rather high, especially since In(2R)NS greatly reduces the rate of recombination in the right arm of II. However, the four events can most easily be attributed to individual events, and an explanation in terms of recombination seems preferable to one in terms of mutation at the bw locus.

The occurrence of a recombinational event implies, in the case of the exceptions tested, that the exchange occurred distal to In(2R)NS and to St(SD), but proximal to the bw locus. This limits the position of St(SD) to the region on a salivary map from 56F, the

distal breakpoint of the inversion, to 59E, the position of bw. It should be noted that although all four of the established brown lines would be expected to be equivalent, line 29-12 shows somewhat less distortion than the other three. This deviation is probably due to a further modification of SD action during the time in which the four lines were maintained separately in mass culture.

Figure I

The frequency distribution of k values from males heterozygous for SD-72, or a bw-bearing derivative of SD-72, and cn bw by cn bw females.



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Sharma, R. P. and M. S. Swaminathan.
Indian Agricultural Research Institute, New Delhi, India. Induced crossing over in Drosophila males by Ethylmethanesulphonate.

Ethylmethanesulphonate (EMS) a mono functional alkylating agent has been shown to be a potent mutagen in animal as well as plant systems. Besides testing its mutagenicity under different treatment conditions, we were interested in seeing whether it has any effect on meiotic crossing over. A significant increase in crossing over frequency restricted only to the centromeric region in IInd chromosome of Drosophila melanogaster females was noticed. The results will be published elsewhere. The present report deals with the effect of EMS on Drosophila males.

Approximately 24 hr old males heterozygous for IInd chromosome markers (dp b cn bw/ ++++) were injected intraabdominally with 0.3% EMS solution prepared in saline. Injected males were individually mated with four dp b cn bw/dp b cn bw homozygous virgin females. After every 6 days these males were provided with fresh virgin females. Three broods of 6 days each were sampled. The brood scheme is based on the findings of Olivieri and Olivieri (1965)[1]. These authors with the help of autoradiography, have studied time of DNA synthesis in spermatogenesis and fixed about 6 days time for the cells to reach to sperm stage from DNA synthesis time. By following this scheme the sperms sampled in Ist brood comes from post synthetic period whereas the 2nd and 3rd brood covers synthetic and presynthetic periods. Of course, if the chemical delays synthetic period, 2nd and 3rd brood can show a mixture of both the stages. The results obtained are given in Table 1.

Brood	No. of males tested	No. of offsprings scored	True crossovers: involving region I (dp-b)	involving region II (b-cn)	Longitudinally 1/2 normal & 1/2 dp b cn bw	dp	dp one wing	bw	brown One Eye	cn One Eye	dp-b	dp-b-cn	cn bw	b-cn-bw
ist Brood	47	2672	-	-	1	4	4	4	-	1	1	2	-	-
iind Brood	28	1814	1	2	-	8*	3	3	1	-	-	-	1	1
iiird Brood	27	1322	2	6	-	1	4	-	-	-	-	-	1	2

*One ♂ was showing clusters.

It is evident from the data that Ethylmethanesulphonate besides producing mosaics in all the three broods for approximately all the markers involved, induced some true cross-overs only in 2nd and 3rd broods. The frequency is always more in region II (b-cn) involving centromere. EMS has been shown to produce chromosome breaks and exchanges more frequently at the centric region or adjacent to it. It has been further shown to produce all types of symmetrical asymmetrical and incomplete exchanges in plant chromosomes[2], [3]. Thus, the crossovers obtained in this experiment involving the centromeric region may be the product of symmetrical centric exchanges involving homologous chromosomes. The cross-overs obtained in the dp-b region of the left arm of 2nd chromosome may be attributed to the iso-locus breaks in homologous chromosomes followed by the exchanges. The other classes showing more than one marker gene may result either from the deletion of some of the genes or by incomplete exchange. The reciprocal classes in these cases may be lost by either deficiency or duplication. The offsprings having single marker gene may be produced either by the deletion or by mutation of that gene. It is difficult to say which of these two processes is responsible. Since the crossovers were not produced in clusters it seems unlikely that they originated during early stages of spermatogenesis. On the other hand the high crossover frequency in 3rd brood, which covers the spermatogonial stages of development, suggest their occurrence during early stages of spermatogenesis.

References:

1. Olivieri, G. & Olivieri, A. Mutation Res. 2 (1965) 366-380.
2. Swaminathan, M.S; V.L. Chopra, & S. Bhaskaran. Ind. Jour. Gent. Plant Breeding. 22 (1962) 192-207.
3. Sharma, R.P. (Unpublished)

David, J. and J. Merle. University of Lyon, France. A reevaluation of the duration of egg chamber stages in oogenesis of Drosophila melanogaster.

The growth of egg chambers during oogenesis of Drosophila has been divided into 14 successive stages by King, Rubinson and Smith (1), and by King (2 ; 3). Studying the frequency of each stage, these authors were able to calculate their duration. But this estimation was necessarily a very indirect one because it was based on the number of egg chambers produced daily by an ovariole, and the whole duration of egg chamber growth.

Moreover, this calculation is only possible with flies of a high vitality in an optimum physiological state, where all ovarioles are functional and without any retention of mature eggs. This is not generally the case in inbred lines kept for a long time in laboratories, although it is usually the case for highly heterozygous F_1 flies, obtained by crossing inbred lines. For physiological experiments, we generally use such F_1 flies, where chance variability is at a minimum. Studying their oogenesis, it appeared that the frequencies of egg chamber stages were not in agreement with King's data, so new studies were undertaken for a better analysis of the problem.

The daily rate of egg chamber production by an ovariole was accurately estimated in a previous paper (David and Clavel, 4). It was observed to vary from 2.0 to 2.3 per day, according to the food received by the female.

The stage frequency was estimated on mated, 4 day old females, kept at 25° in total darkness. These flies were F_1 heterozygotes from a cross between a vestigial strain and a wild strain : Champetières. Four females and five males were kept together in a glass tube where the food (axenic medium : David, 5) was renewed daily. Whole ovaries were stained by Feulgen method, then the ovarioles were separated and mounted in Canadian balsam. The mean ovariole number for both ovaries was approximately 45 but some ovarioles were lost during mounting. A total number of 1321 egg chambers, corresponding to 175 ovarioles, was examined. As the identification of certain stages is sometimes difficult, the observations were made separately by both authors of the present paper, and the data averaged.

The mean number of egg chambers per ovariole is 7.55, close to the value of 7 found by King. The growth duration of an egg chamber, from stage 1 to stage 14, is obtained by dividing the mean number of these chambers in one ovariole by their daily rate of production. According as the rate is assumed to be 2.0 or 2.3, the calculated duration varies from 3.78 to 3.28 days.

The results concerning stage frequencies are pooled in the Table. The duration of each stage was estimated by multiplying the whole growth period by each frequency. In order to facilitate the comparison with the results of King et al., the closer whole duration, that is 3.28 days, was used for the calculation.

	New data		data of King (2)	
Stages	frequency (%)	duration (hours)	frequency (%)	duration (hours)
1	12.1	9.56	13.3	9.58
2	12.1	9.56	13.3	9.58
3	12.6	9.95	13.3	9.58
4	11.6	9.16	10.6	7.62
5	3.3	2.61	2.8	2.01
6	10.7	8.45	13.3	9.58
7	11.0	8.69	13.3	9.58
8	6.6	5.21	5.2	3.74
9	7.1	5.61	1.0	0.74
10	6.5	5.13	0.3	0.22
11	0.5	0.40	0.1	0.07
12	2.4	1.90	0.1	0.07
13	1.0	0.79	0.1	0.07
14	2.5	1.98	13.3	9.58
total	100%	79h	100%	72h

From the Table it can be concluded that the main discrepancy between King's and our data concerns the stages 9 to 14 : we found that the length of vitellogenesis (stage 9 to 13) is longer, while on the other hand, the stage of mature egg (stage 14) is much shorter.

As our estimation was made on flies with high fecundity, where the relations between egg production and ovariole number were accurately analysed, it may be concluded that the new results describe the process of normal oogenesis in Drosophila melanogaster more precisely. It may be assumed that, in the flies studied by King, a partial retention of stage 14 ovocytes took place, which, as a consequence, partly inhibited the growth of the following egg chambers. Such retention is frequent in most inbred lines (David, 6) and particularly among virgin females. So, the stage distribution given by King is to be taken into consideration by workers utilizing such flies.

(1) King, R.C., Rubinson, A.C., Smith, R.F. - 1956 - Growth 20 121-157
(2) King, R.C. - 1957 - Growth 21 95-102
(3) King, R.C. - 1964 - Symp. Roy. Ent. Soc. London 2 13-25
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(5) David, J. - 1962 - D.I.S. 36 128
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This paper was submitted to Dr. R. C. King and we thank him very much for many helpful suggestions.

Tokunaga, C. Lawrence Radiation Lab., University of California, Berkeley, California. A test for functional allelism between Multiple sex comb (Msc) and the mutants Polycolmb(Pc) and Extra-sexcomb(Scx).

According to Hannah-Alava (1958, '64), the 'extra sex comb' locus consists of two sub-loci each represented by a semi-dominant mutant, Polycomb(Pc) and Extra-sexcomb(Scx) separated by 0.2-0.3 of a crossover unit. Pc and Scx are located in the third chromosome between st(44.0) and p(48.0) probably just to the left of p. They are not associated with any chromosomal aberration.

Multiple sex comb(Msc) (Tokunaga, 1966) is also located very close to p between ri(47.1) and p. It is associated with a small inversion extending from 84B to 84F.

The following crosses were made to determine whether Msc and Pc, and Msc and Scx are functionally allelic.

1. Test between Msc and Pc.

Crosses: A. Pc/TM1,Mé ri spd[1] ♀♀ x pr en; Msc/Sb ♂♂
B. Pc/T(2;3)Mé ♀♀ x pr en; Msc/Sb ♂♂

F_1 segregation (A+B)

	Msc/Pc	Msc/Mé	Pc/Sb	Mé/Sb	Total
♂♂	318	305	288	1	912
♀♀	280	290	325	0	895

The fact that Msc/Pc is viable in contrast to the lethality of Msc/Msc and Pc/Pc indicates that Msc and Pc are not functionally allelic.

2. Test between Msc and Scx.

Cross: th st Pc Scx pP ss/TM1,Mé ri ♀♀ x pr en; Msc/Sb ♂♂

F_1 segregation

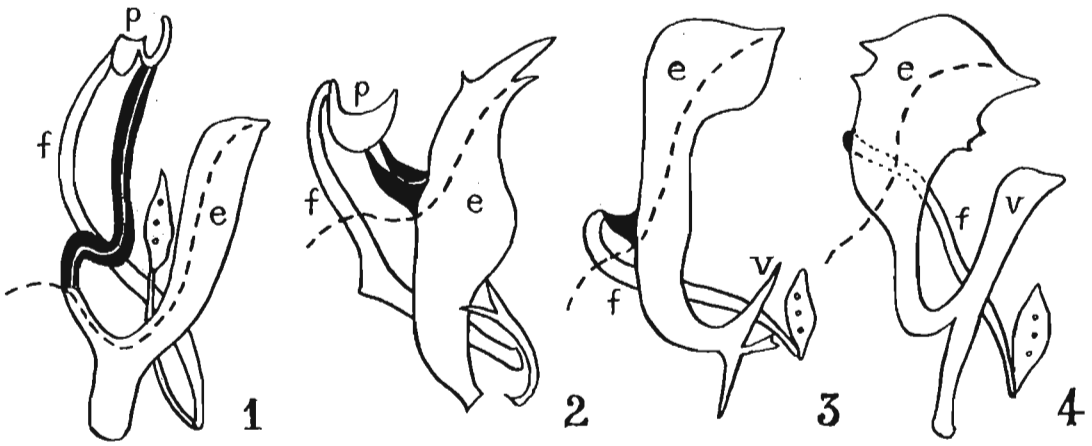
	Msc/Pc Scx	Msc/Mé	Pc Scx/Sb	Mé/Sb	Total
♂♂	790	1026	1063	0	2879
♀♀	662	1099	1176	1	2938

The fact that Msc/Pc Scx is viable in contrast to the lethality of Msc/Msc and Scx/Scx indicates that Msc and Scx are not functionally allelic. It is noted however, that the viability of Msc/Pc Scx is very much reduced.

Furthermore, it is seen that the combinations Tm1, Mé/Sb and T(2;3)Mé/Sb are nearly always lethal, as reported by E. B. Lewis (1949).

Okada, Toyohi. Tokyo Metropolitan University, Tokyo, Japan. Homology in the components of the phallic organs of Leucophenga and Microdrosophila.

The genera Leucophenga and Microdrosophila show close resemblance to each other in their U-shaped elements of phallic organs (compare figs. 1 and 4), which is found, however, to be merely superficial. Misinterpretation (Okada, 1966. Bul. Brit. Mus. NH, Suppl. 6:121) was caused by the treatment of cleared material. The true homology can be established basing on the innervation of ejaculatory duct (shown by broken lines in figures) into aedeagus (e), which is traceable in fresh and non-cleared material. The aedeagus corresponds to the ventral arm of the U-shaped elements in Leucophenga (e.g., nigroscutellata Duda, fig. 1), while it corresponds to the dorsal arm in Microdrosophila (figs. 3, 4). The ventral arm in Leucophenga (shaded black in figures), which had been mistaken as aedeagus, is paired, elongated and connected to ventral fragma (f: left half obliterated) by means of unpaired posterior parameres (p). This paired structure shows gradual reduction in size in Microdrosophila species. It is still paired and large in M. maculata Okada (fig. 2), unpaired and smaller in M. purpurata Okada (fig. 3), while it becomes vestigial in M. nigrohalterata Okada (fig. 4) with the aedeagus attached nearly directly to the ventral fragma. Furthermore, it is observed that in Microdrosophila the ventral process (v = ventral arm) tends to develop in compensation to the reduction of the paired structure in question and parallel to the development of aedeagus (compare figs. 2, 3, and 4).

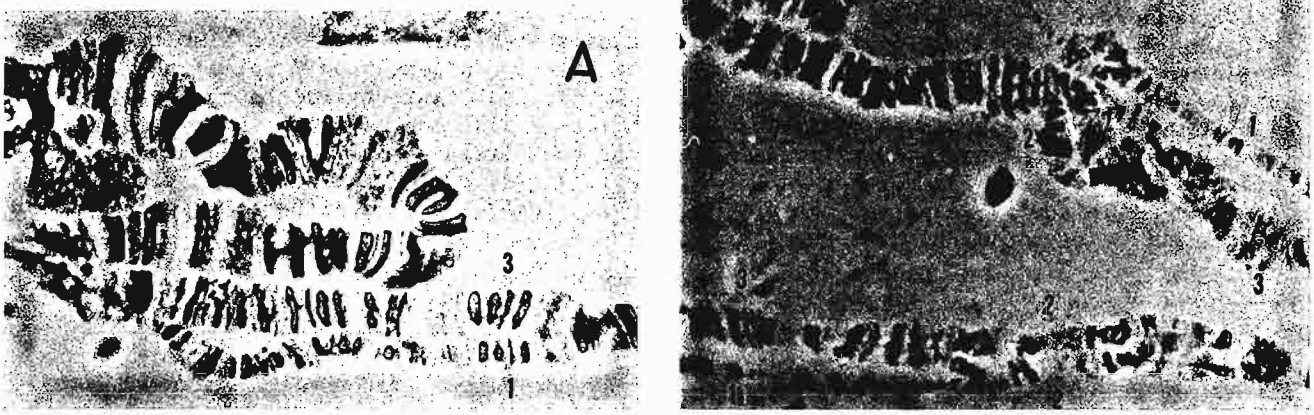


Figs. 1 ~ 4. Phallic organs of Leucophenga (1) and Microdrosophila (2 ~ 4), sinistral aspects. For explanation see the text.

Gersh, Eileen S. University of Pennsylvania, Philadelphia, Pennsylvania. Chromatid asynapsis in salivary gland nuclei.

The accompanying photos were made from a single slide from a rst^2/yw larva. While Figure A suggests a triploid condition with partial asynapsis of one of the chromosomes, Figure B clearly illustrates quadripartite structure in both the regions shown. It is probable, therefore, that these are all examples of chromatid asynapsis, with Figure A showing a 3-1 separation of strands.

Similar conditions were found in a few other nuclei. Such nuclei appeared to have in common a higher degree of polyteny than others (in the same pair of glands) in which pairing was complete or in which, if asynapsis occurred, it was as usual, between two chromosomes.



Schalet, A. University of Connecticut, Storrs, Connecticut. Evidence for a different function associated with the locus of suppressor of forked.

Females of the genotype f su-f/f su-f have bristles that are nearly wild type. Females of a genotype that includes f su-f/f su-f$^-$ have bristles that are nearly wild type with respect to forked but also exhibit a distinct phenotype: Bristles - small, minute-like; some head and thoracic bristles may be absent. Eyes - invariably roughened to some extent, anterior indentation. Wings - in some flies any or all of the following effects - blistery, broader, extra veins, held somewhat upward and outward. This phenotype is also shown by females of a genotype that includes f su-f/f$^+$ su-f$^-$. It is not shown by f su-f$^+$/f su-f$^-$ or f su-f$^+$/f$^+$ su-f$^-$ females. The genotype f$^+$ su-f/f$^+$ su-f$^-$ has not been tested. Males that are f su-f/Y, f su-f/0, f su-f/Ysu-f$^+$ or f$^+$ su-f$^-$/Ysu-f$^+$ do not show this phenotype.

Most of the 13 radiation treated chromosomes which show the new phenotype were of a genotype that included f$^+$ su-f$^-$/f su-f. Nine were picked up in sc^8 inversion chromosomes as induced y or ma-1 mutants. Four others were picked up in non-inverted chromosomes, 1 as a ma-1 mutant and 3 as proximally located lethals. In addition to su-f, all chromosomes were tested for a number of other markers in the region (see my note this D.I.S.). These included 120, close to but not immediately to the left, and bb to the right in the normal order. Lethal 20 as well as su-f is covered by B^{S}Y. The results were: 4) 120$^-$ su-f$^-$ bb$^-$; 1) 120$^-$ su-f$^-$ bb; 6) 120$^-$ su-f$^-$ bb$^+$; 1) 120$^+$ su-f$^-$ bb$^-$; 1) 120$^+$ su-f$^-$ bb$^+$. (Here bb$^-$ means lethal over a bb lethal and bb means a viable bb phenotype over a bb lethal.) In the chromosome that was 120$^+$ su-f$^-$ bb$^-$, there was evidence for another lethal locus between 120 and su-f. The chromosome that was 120$^+$ su-f$^-$ bb$^+$ behaved as sex-linked lethal with a regular Y but was covered by a B^{S}Y. Unfortunately, it has been lost. One of the cases that was 120$^-$ su-f$^-$ bb$^+$ was tested and found to be normal over the bb deficiency chromosome y sc^4 B InS w^a sc^8 chromosome. In addition to the above 13 chromosomes there were a number of cases of deficiencies to the left and to the right of su-f which were su-f$^+$. There were no cases of 120$^-$ su-f$^+$ bb$^-$ or 120$^-$ su-f$^+$ bb.

Taylor, William C. University of Chicago, Chicago, Illinois. Somatic crossing over in $sc^{4L}sc^{8R}$ heterozygotes.

Experiments were undertaken to study mosaic spots produced by somatic crossing over in y $sc^{4L}sc^{8R}/sn^3$ females. Several stocks were maintained as separate lines, each arising from one pair of parents. Stock males were tested for the presence of extra Y-chromosomes by pair-mating a sample to attached-X, no free Y females. Samples of females scored for mosaics were also tested for the presence of extra Y-chromosomes by mating to attached XY, no free Y males; no evidence of extra Y-chromosomes was found in either case. Cytological tests failed to show any extra Y-chromosomes, but did show X-chromosomes which looked like the $sc^{4L}sc^{8R}$ chromosome.

Forty-five abdomens from females which had been irradiated with 1200 R at various stages in development (36-90 hours after egg laying) were scored for single y, single sn^3, and y-sn^3 twin spots. A second experiment used 104 females from a different line not irradiated during development. The results were:

$ysc^{4L}sc^{8R}/sn^3$--frequency of spots/abdomen

	twin spots	single y	single sn^3
45 irradiated females	.49	2.8	1.2
104 non-irradiated females	.05	1.4	.53

In both experiments there were more than twice as many single y spots as single sn^3 spots. One would certainly not expect such a result, as the y $sc^{4L}sc^{8R}$ chromosomes lacks all of the centric heterochromatin with the nucleolus-organizer, thus being unable to carry out ribosomal-RNA synthesis (see Ritossa and Spiegelman, Proc. Nat. Acad. Sci. 53:737-745, 1965, for the localization of the r-RNA loci in D. melanogaster). Simple proximal heterochromatic exchange cannot alone explain these results. Part of the excess of y over sn^3 can be due to the fact that sn^3 is not completely expressed; Brown (unpublished, in Brosseau, J. Exp. Zool. 136:585, 1957) found phenotypically normal bristles on homozygous sn^3 abdomens. When only single spots with more than one bristle in each were counted, there were 24 y and 25 sn^3 in the irradiated group, with 17 y and 9 sn^3 in the non-irradiated group. Thus, the entire excess of y in the irradiated group and some of the excess in the non-irradiated group was due to single spots of only one bristle. Another possibility which might account for the excess of single bristle y spots is the fact that a single exchange within the inversion loop to the right of sn would produce a dicentric chromosome deficient for the y locus. If this exchange occurred in the division just before bristle synthesis, there is a 50% chance that both centromeres of the dicentric would go to the same pole and produce a y bristle.

The next logical step would have been to switch the y and sn^3 markers. However, it is not possible to separate y from the sc^{4L} break. The sn^3 marker was introduced into the inversion and 100 y $sc^{4L}sc^{8R}sn^3/$ + + + females (non-irradiated) were scored for single y sn^3, single y and single sn^3 spots.

y sc^{4L} sc^{8R} sn^3/+++-- frequency of spots/abdomen

	single ysn^3	single y	single sn^3
100 non-irradiated females	.35	.26	.23

A simple exchange in proximal heterochromatin would result in single y sn^3 spots, at a frequency roughly comparable to single y or single sn^3 spots in the previous experiments. A single sn^3 spot requires that a two-strand double euchromatic exchange take place. However, if the distal sections of heterochromatin pair, one of the exchanges could take place here, leaving only one exchange which must take place in euchromatin between y and sn^3. The most logical explanation for single y spots is that they are the reciprocal product of the double exchange which produced the single sn^3 spots. This hypothesis is further substantiated by the fact that the frequencies of single y and single sn^3 spots are almost identical, .26 and .23 respectively.

The y-sn^3 twin spots in the two earlier experiments were most likely the result of a single exchange in proximal heterochromatin. If such is the case, the y bristles were formed from cells lacking the ribosomal RNA loci on both X-chromosomes and thus unable to carry out r-RNA synthesis. These data thus allow one to speculate that hypodermal cells may survive for some generations without a capacity for r-RNA synthesis. Many of the y bristles, both in single and twin spots, were of an abnormally small size.

Weisbrot, David R. and Howard Gross.
Tufts University, Medford, Massachusetts.
Strain-specific aggregation of adults of competing strains of D. melanogaster.

Fifty virgin females and an equal number of males of one strain of D. melanogaster (homozygous for the mutant, ebony) were introduced into a 4"x4"x2" plastic population cage with a like number of adults from either of two wild type strains. (A2- a strain with a history of inbreeding and A6- a strain maintained by mass mating.) Eight equally spaced, removable vials protruded from the sides and opened into the central part of the cage. These vials contained 5cc. of standard Cornmeal-Molasses-Agar media that had been seeded with a 10% live yeast suspension. Twice a day, the vials were removed from the cage, the flies within each vial were etherized and counted, the vial repositioned in the cage, and the flies returned to the center of the cage. After seven days, the vials were removed to allow emergence of the F1 within the vials. The flies of this generation were then introduced into cages and fresh vials inserted. Of the eight new vials, two had been pre-conditioned by placing 60 adults of the ebony strain in the vials for 24 hours, and two had been similarly pre-conditioned by the other strain represented in the cage. Counts were made twice daily for six days.

Using a 2 x 8 contingency table (the two strains with eight vials to choose from) on the daily counts of flies per vial, X^2 values were obtained that indicated an increasing non-random distribution of adults in the eight vials over the 7 day period of counting for the competing ebony and A2 parents. On the other hand, no discernible pattern could be seen for the competing A6 and ebony adults. The data are reported in Table 1.

Table 1

	day 1	2	3	4	5	6	7	
X^2 values	7.32	7.80	6.97	8.74	17.13	14.73	32.73	A2-ebony competition
	4.89	19.12	11.24	4.35	7.70	8.30	12.56	A6-ebony competition

For 7 degrees of freedom, the X^2 value is equal to 14:07 at the 5% level of significance.

Table 2 is a summary of the data for the second generation. Heterozygotes between ebony and A2 were recognized by the trident pattern and it was confirmed by progeny testing. In this case, the data are reported in a 3 x 3 contingency table since there were three classes of flies (ebony, A2 and the heterozygotes) and they had three choices of media (non-conditioned and preconditioned by either A2 or ebony adults for 24 hours.) The numbers in the table are the total of that class on that medium for the 6-day period of counting. The numbers in parentheses represent the X^2 values, and in cases of significant differences, the plus or minus sign indicates whether the observed number was greater or less than the expected. It is apparent that all three classes of flies distribute themselves randomly with regard to the ebony pre-conditioned medium. On the A2 preconditioned medium, however, there were significantly fewer ebony and a significant excess of A2 flies. There is an indication of preference by ebony flies for unused media, while heterozygotes show no preference at all. The suggestion from this data is that ebony and A2 flies seem to establish biotic or ethological niches for themselves within a physically uniform environment.

Table 2

		Class of Flies: Ebony	A2	Heterozygotes
Media pre-conditioned by	Ebony	25 (.11)	104 (.11)	139 (.48)
	A2	7 -(15.56)	166 +(12.41)	139 (1.50)
	Neither	107 +(4.40)	366 (2.62)	489 (.02)

Schalet, A. and V. Finnerty. University of Connecticut, Storrs, Connecticut.

The arrangement of genes in the proximal region of the X chromosome of Drosophila melanogaster.

We have determined the relative positions of a number of genes in the proximal region of the X chromosome. Most of these have been supplied by other workers who had localized them to the right of carnation by conventional crossover tests. We have utilized duplications of the proximal X carried on the Y to test mutants of independent origin for allelism. Other major tools, deficiencies of the maroon-like locus on the X chromosome and deficiencies of ma-l induced in a Y chromosome carrying a duplication for ma-l^+, were used to position the various mutants. The extent of some of these chromosome aberrations are indicated in the new mutants section of this issue. The relevant breakage point in the Y with the duplication of the proximal X, Y^S ma-l^+.Y^L y^+ (reported as y^+Yma-l^+#2 in DIS-38), as well as the pertinent breakage points in the most useful rearrangements are shown in the accompanying figure.

Additional points of interest and amplification:

1) Dp(1;f)3 has been reported by Cooper to have a proximal break in the distal portion of heterochromatic segment hD. On this basis all of the loci to the right of A7 may be considered to be located in the proximal heterochromatin.

1) The relative positions of:
A7 and N30
gluful-2, DCA3-19 and DCB2-19
t2-14a and DCB2-35a, 151
have not been determined.

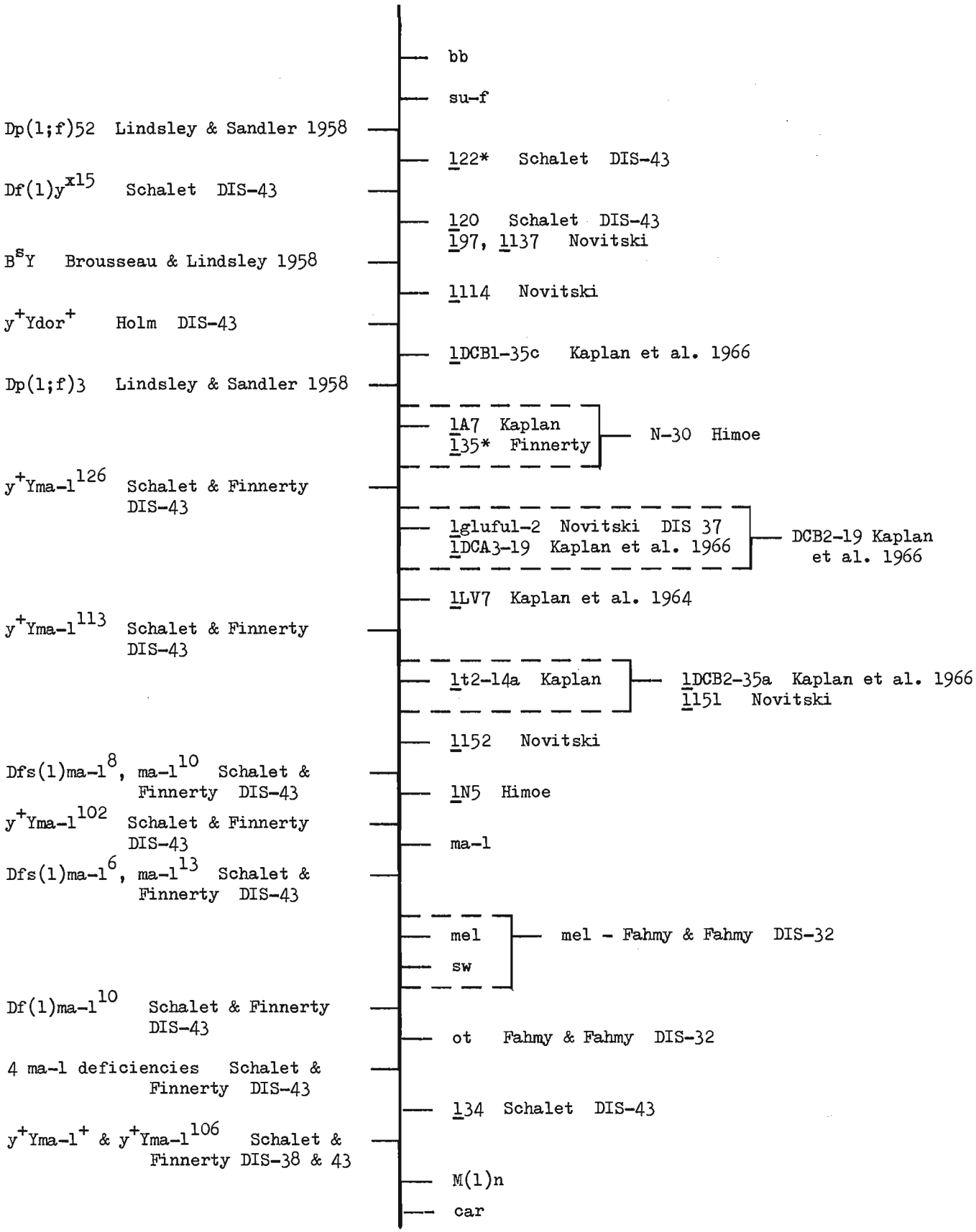
2) Placement of 152 to the left of t2-14a and DCB2-35a, 151 is uncertain and based on the following considerations. A lethal now lost, 133 was found to be lethal with 152 and viable with t2-14a. But 33 was lethal with y^+Yma-l^{102} while 152 was covered by y^+Yma-l^{102}. If 33 was a deficiency that included 152, rather than two close but separable lethals, then 152 should be to the left of t2-14a.

3) Placement of mel to the right of sw is uncertain. However, the viable ma-l^{14}, when heterozygous with either of two ma-l deficiencies, ma-l^6 and ma-l^{13}, with breaks just to the left of ma-l, shows a phenotype that mimics the body color and turned up wing effect of mel. But ma-l^6, ma-l^{13} and ma-l^{14} are all wild type with mel.

4) We have at least 3 additional aberrations, not involving ma-l, that are within the region between 134 and bb.
   a. 1DCB1-35b, Kaplan et al. 1966, is a deficiency with a distal break to the right of LV7, a proximal break to the left of bb and lethal with all the extant lethals in between.
   b. 1D43L1 Himoe is a deficiency with a distal break to the right of t2-14a and 151 and a proximal break between su-f and bb.
   c. 1t2-4a Kaplan is lethal with sw and mutant with mel.

5) Crossing over between some of the loci in the region has been measured under standard conditions.
   a. 120 and su-f - 1/3,507(0.03%) in experiments which gave 2/3,507(0.06%) between su-f and Dp(1)$sc^{V1}y^+$ and 188/3,507(5.4%) between car and su-f.
   b. sw and ma-l - 4/747(0.5%) in an experiment which gave 63/747(8.5%) between f and sw.
   c. ma-l and su-f - 19/865(2.2%) and 15 and su-f - 13/370(3.5%) in the same experiment.
   d. 15 and 120 - 95/2637(3.6%)
   e. 134 and 120 - 63/2632(2.4%)

6) Mutants localized to the right of car but not covered by y^+Yma-l^+.
Lethals - A11, A12, B4, DCB2-35b of Kaplan
Lethals - 1, 77, 106, 412, glufulproless of Novitski
Lethal - 5 of Schalet
Lethal - D43L3 of Himoe
Visible - wa^2 of Fahmy and Fahmy



The arrangement of genes in the proximal region of the X chromosome of Drosophila melanogaster.

* Lost

Spiess, Luretta D. and Eliot B. Spiess. University of Illinois at Chicago Circle, Chicago, Illinois. Mating frequency and conditions of yeast and water in culture of D. persimilis.

In attempting to stabilize some of the conditions important to mating frequency in this species a three-dimensional classification experiment was run (before the authors moved from Pittsburgh to Chicago). Some possible external conditions leading to variation in mating speed were suspected as follows: 1) tap water (Pittsburgh), 2) type of yeast with various contaminants (Fleischman's Baker yeast cake vs. dry yeast), and 3) presence or absence of live yeast in storage at least 24 hrs. before mating tests. Culture medium was corn-meal-molasses-yeast-agar-propionic acid-tegosept.

Flies from the 27th generation of our fast development selected population (DF population in Spiess and Spiess, Genetics 50: 863-877, 1964) were isolated, determined to be Whitney (WT) homokaryotypes, and mass mated in bottles provided with plastic spoons containing food and yeast according to four combinations: using either tap water or distilled and using either dry yeast or yeast cake. (See Spiess and Langer, Evolution 18: 430-444, 1964, for previous methods.) Spoons with 100 - 200 eggs were inserted into the same type of food in bottles and cultured at 15°C. On emergence, adults were sexed and isolated in vials containing the same type of food for 7 days, at which time they were transferred to fresh vials in lots of 10; these vials were then either provided with the same type of yeast or not given any yeast for 24 hrs. Mating frequencies (60 minutes' observations) are given in the following table together with an analysis of variance. One hundred pairs were tested at each combination.

Percentage Matings (DF Population Progeny)

	Storage Conditions	
Culture Conditions	With live yeast	Without yeast
Tap water		
+Dry yeast:	82%	59%
+Cake yeast:	69%	41%
Distilled water		
+Dry yeast:	85%	41%
+Cake yeast:	80%	50%

Analysis of Variance for Mating Data
(80 chambers of 10 pairs per chamber)

	d.f.	Sum Sq's.	M.S.	F
Between treatments:	7	234.3	----	(n.s. = not significant) (** = p<.01 * = p<.05)
Between:				
Tap-distilled	1	0.6	----	n.s.
Dry-cake:	1	7.8	7.8	2.11 n.s.
Yeast in storage (+) - (-):	1	201.6	201.6	54.34**
Interactions	4			
Water x Yeast	1	13.6	13.6	3.67*
Yeast x Storage	1	1.5	----	n.s.
Storage x Water	1	5.5	----	n.s.
Storage x Water x Yeast	1	3.7	----	n.s.
Within treatments:	72	266.9	3.7	n.s.
Total:	79	501.2		

It can be seen that live yeast at least 24 hrs. before mating is critical to the outcome. Probably the live yeast grows better in distilled water than in the tap so that the water x yeast interaction has borderline significance. It is likely that conditioned by

live yeast in nature at the food source to mate, and it is essential to provide them with live yeast in studying mating behavior. This fact has been mentioned by Manning and others in the past, but no data have been published heretofore on this point.
(This work was supported by Contract No. AT(30-1)-1775, U.S. Atomic Energy Commission at University of Pittsburgh.)

Lifschytz, E., and R. Falk. The Hebrew University, Jerusalem, Israel. A new system for fine structure analysis of genes in Drosophila.

In recent years a number of systems for high power resolution of recombination and back-mutation have been described for Drosophila. We constructed a system for high power resolution analysis based on the lethal interaction between pn and K-pn and on lethals in the X-chromosome, covered by segments of the X-chromosome translocated to the Y-chromosome. The stages that were involved in the construction of the system will be described elsewhere. Only the final system is given here:

a. $\frac{y^2\ pn^i\ cv\ l^{B57}}{F\ M\ 6\ (1)} \times \frac{pn^j\ l^{3des}}{Y\cdot\ pn^-w^+}$

b. $\frac{y^2\ pn^i\ cv\ l^{B57} + \quad +}{pn^j \quad + l^{3des}};\frac{}{+} \times \frac{v\ g\ l^{B57}\ l^{3des}}{Y\cdot\ ma\text{-}l^+};\frac{ca\ K\text{-}pn}{ca\ K\text{-}pn}$

The system includes two stages:
a. The stage for large scale "automatic" collection of virgin females.
b. The stage for the detection of recombinants in heterozygotes for different pn-alleles or of back-mutations in homozygotes for a single pn-allele.

It is easy to see that all progeny should die, excluding males that are recombinants (or back-mutants) in the pn-locus and females that are recombinants between the lethals or products of non-disjunction in the fathers.

For the "automatic virgin" system the lethals were chosen so that one was proximal enough to be covered by both $w^+\cdot Y$ and by $ma\text{-}l^+\cdot Y$, while the other was somewhat more distal, thus covered only by the $ma\text{-}l^+\cdot Y$-chromosome. We verified that the frequency of non-disjunction was low and that the lethals did not produce "Durchbrenners" even under the uncrowded conditions that prevailed when most larvae died. The lethals were chosen so that there was about 0.5% crossing-over between them, so that there were enough viable progeny per culture (3-10 females) to facilitate the eventual single rare recombinant between pn-alleles. Furthermore, since the frequency of recombination between the lethals was predetermined, the number of females per culture served to estimate the total number of zygotes that had been produced.

In order to minimize the work involved, we found it best to use 20 pairs of flies per mating in 1/4 litre culture bottles (somewhat above the optimum) and to transfer the parents twice to fresh cultures, after they stayed for 4 days in the old ones. Only a sample of the bottles was etherized and from them the mean number of females per culture was determined. In the remaining bottles the presence of males was checked by inspection without etherization. Analyses of 1.2×10^6 zygotes may be carried out routinely by a single technician.

Preliminary analyses resolved the pn-locus into three sites: $pn^1\text{-}pn^2, pn^{59j}\text{-}pn^{AA1}$.

A somewhat modified system in which the recombinants between pn-alleles were the female progeny and the recombinants between the lethals were the male progeny was also constructed. With proper selection of lethals these systems may be utilized also for analyses of other events in the chromosome, such as unequal crossing-over or negative interference.

Mange, A. P. and A. R. Alexander. University of Massachusetts, Amherst, Mass.

Fecundity of virgin versus non-virgin D. melanogaster males given varying numbers of females.

This experiment was designed to determine the number of females needed to exhaust a male's 24-hour supply of sperm, and to compare previously unmated with previously mated males. All flies were Canton-S raised at 25° C on cornmeal-molasses-yeast-agar food. At the time of the experimental matings (day 0) all flies were four days old. Virgin fathers were stored singly in vials from age 0 to age 4; non-virgin fathers were stored 10♂♂ to 300♀♀ in half-pint bottles from age 0 to age 4; virgin females were stored 200 per bottle from age 0 to age 3 at which time they were distributed into groups of 1, 3, 9, or 27♀♀ per vial. On day 0 one male was added to each of the female-containing vials (etherizing the male only), left for 24 hours, and then removed (without etherization). The females were not separated but were subcultured on days 4, 7, 10, 13, 16, 21, 26, and 33. A group of females mated to a single male, together with the subcultures of these females, will be designated a harem. The results are indicated below.

type of father	♀♀ in harem	replicates	fertile replicates	total offspring	offspring of all replicates per harem	offspring of all replicates per ♀	offspring of fertile replicates per harem	offspring of fertile replicates per ♀
virgin	1	12	6	1,605	134	134	268	268
virgin	3	12	10	5,185	432	144	519	173
virgin	9	12	12	9,073	756	84	756	84
virgin	27	11	11	11,788	1,072	40	1,072	40
		47	39	27,651				
non-virgin	1	12	2	513	43	43	256	256
non-virgin	3	12	8	2,232	186	62	279	93
non-virgin	9	12	12	4,532	378	42	378	42
non-virgin	27	12	12	5,549	462	17	462	17
		48	34	12,826				

It is seen that virgin males were more successful fathers than experienced males during the 24-hour test period: more harems were fertile and more offspring were produced on either a per harem basis or on a per fertile harem basis. Whether this advantage was gained by an increased likelihood of copulation, or by more sperm being transferred per copulation, or both, is unclear, although differences in the frequency of copulation, by itself, could account for the observations. This is supported by Mossige (Am. Nat. 1955) who found that 2-day-old virgin Canton-S males, when provided 10 females for 24 hours, could fertilize 8.5 ♀♀ on the average; under similar conditions, 2-day-old non-virgin males fertilized 4.6 ♀♀.

How many females are needed to exhaust a male's supply of sperm? The results of this experiment are equivocal. The total number of offspring increases throughout the range of females used, although it might seem that 9 females, and certainly 27, could exhaust a male's sperm even if any one female mated only once during the 24-hour period. The experiments of Lefevre and Parker (D.I.S. 1963), also based on total progeny counts, suggest that a male can be "saturated" by fewer than six females when the male is transferred daily over a five day period. Mossige (Am.Nat. 1955) showed that a virgin male of any age (up to 21 days), when provided with ten females, would fertilize about 8 of them during the first 24 hours after mating. With continued daily transferring to 10 new females, however, this number decreased to about 2 fertilized females per day. McSheehy (D.I.S. 1963) reported a male mating frequency of about 1.5 copulations per day when males (virgins?) were provided two females each for 70 hours, and 8.7 copulations per day when provided eight females each.

However, neither total progeny counts, nor number of females successfully inseminated, nor copulation frequency indicates directly the parameter of interest, namely, sperm depletion. If, say, five females can deplete a male's sperm supply, then providing more females may, nevertheless, increase the frequency of copulation (as McSheehy's experiment suggests), and therefore the number of fertilized females and the total progeny count. One need only suppose that, with an increased supply of females, each of the more frequently occurring copulations involves fewer sperm, but not fewer than the female can store and utilize. (A female generally stores far fewer sperm--less than 700--than the 3-4 thousand that a male can transfer

(Kaplan, et al, D.I.S. 1962; Kaufmann and Demerec, Am. Nat. 1942).) This view is supported by the data of Stromnaes and Kvelland (Hereditas, 1962) who showed that females inseminated by highly active males (those mating with all ten of the females provided by 12 hours) produce fewer progeny, on the average, than females inseminated by less active males. However, on a per male basis, the more active ones produced the greatest total number of progeny.

Maroni, Gustavo P. Atomic Energy Commission, Buenos Aires, Argentina.

Mutations induced at specific loci in motile sperm.

Adult virgin "Binsc" males 7-9 days old were X-rayed (3000r) and immediately mated to females bearing the "maple" chromosome (y ac sc pn w rb cm ct^6 ras^2 v g^2 f car) heterozygous with Ins(1)sc^{S1}, dl-49, y sc^{S1} v B. Mating was allowed for only 2-3 hours in order to sample fully mature (motile) sperm (Lefevre and Jonsson, Mut. Res. 1, 1964). F_1 B females were observed for mutations at the marked loci. Altogether, 3163 males were irradiated, 10,132 F_1 females were examined and 69 mutants were found.

The yellow mutants were considered apart from the rest because in the Binsc chromosome this locus is near a portion of heterochromatin and has been shown to be very frequently involved in rearrangements (especially small deletions) with one break in this heterochromatin. The mutation frequency of yellow in this condition is a good estimate of heterochromatic rearrangement frequency. The overall mutation frequency of the y locus was 8.67 x 10^{-7}/r, which is significantly higher than the mean overall mutation frequency found for the remaining loci (ac and sc excluded), which was 1.43 x 10^{-7}/1/r. (The overall frequency includes cases of mutant F_1 females which were inviable or sterile.)

In the table, the results are compared with those obtained by R. Valencia (unpublished) in a similar experiment where a mixture of spermatozoa was studied. In this case, sperm was collected during about 3 days after treatment of 3-5 day old males. Point mutations have been separated from chromosome rearrangements according to the viability and fertility of the mutants which were kept in stocks, the classifications being based upon past experience of Muller and the Valencias with these same loci. This classification will be checked by cytological analyses now in progress.

The results for the 10 "maple" loci observed (other than y) are in agreement with those of Lefevre (Genetics, 1967). That is, the increase in X-ray induced mutation frequency in motile sperm is due to an increase in chromosome rearrangements and not point mutations.

Mutations	Sperm	Av.mut.freq./locus/r x 10^7 (2)	Freq.(motile) / Freq.(mixture)
Point mut. 10 loci(1)	Motile	.4	.8
	Mixture	.5	
Chr. rearr. 10 loci(1)	Motile	.7	1.4
	Mixture	.5	
Chr. rearr. (yellows)	Motile	3.7	1.5
	Mixture	2.4	

(1) pn w rb cm ct ras v g f car

(2) Frequencies include only those mutations of which stocks could be made.

Chromosome rearrangements involving the y locus show a relative increase in frequency in motile sperm which is quite similar to that observed for the other loci. This suggests that the X-ray sensitivity (either increased breakage or impaired ability to repair) of heterochromatic regions and of euchromatic regions is equally enhanced in fully mature sperm. This situation is quite different from that found in female germ cells, in which the increase in yellows due to heterochromatic rearrangements in stage 14 as compared with stage 7 oocytes was about 88 times greater than the increase in other loci (Valencia and Valencia, Rad. Res. 14, 1961, and R. Valencia, Genetics 52, 1965).

Moriwaki, D. and Y. N. Tobari. Tokyo Metropolitan University, Japan. Unusual segregation of the four phenotypes in the dihybrid F_2 generation caused by male crossing over in D. ananassae.

In almost all species of Drosophila male crossing over does not occur spontaneously. Accordingly, an appearance of some flies of double recessive in the F_2 generation of the ordinary dihybrid experiment is liable to lead to the conclusion that the two loci are located on different chromosomes. However, if there exists a spontaneous crossing over in males, the appearance of double recessives does not necessarily mean their independence. In D. ananassae, as reported previously, male crossing over has been known to occur; thereupon it became necessary to reexamine some of the linkage data, concerning several mutant genes which were previously reported to be independent.

Let us take b (black) and se (sepia) for instance. Beginning with a cross of b x se, in the F_2 there appeared a considerable number of b se besides +, b, and se. The frequency of b se was different from the value of 1/16 expected under the condition of independent assortment. Nevertheless, it was concluded that b belonged to the 2nd chromosome and se to the 3rd (the previous conclusion to be corrected in this note). However, further tests by usual back cross technique using the double recessive flies, have shown that these two genes, b and se, must be linked in the same chromosome, although the loci are located fairly apart from each other. The data leading to the conclusion are as follows:

Region	Recombination value ♀♀	♂♂
	48.2% (1781 flies)	4.0% (1624 flies)
b ~ se	49.6% (2404 flies)	12.5% (319 flies)
	48.5% (610 flies)	12.2% (566 flies)

Concerning the estimation of recombination value between two genes, Mather (1938) has discussed the case where the recombination fraction differs in the male and female gametogenesis, representing the former by p_1 and the latter by p_2. The gamete series from the coupling case were expected as:

	AB	Ab	aB	ab
♂	$(1/2)(1 - p_1)$	$(1/2)p_1$	$(1/2)p_1$	$(1/2)(1 - p_1)$
♀	$(1/2)(1 - p_2)$	$(1/2)p_2$	$(1/2)p_2$	$(1/2)(1 - p_2)$

Writing P for $(1 - p_1)(1 - p_2)$, he maximized the logarithum likelihood expression obtained from the expectations of the four classes in the F_2, and arrived at the quadratic equation, $P^2(AB + Ab + aB + ab) - P(AB - ab - 2Ab - 2aB) - 2ab = 0$, where AB, Ab, aB, and ab represented the observed frequencies of the four classes indicated by the letters. Applying this formula to the present case, we reexamined almost every one of the unusual segregation cases. Two of them are sampled below.

(Example 1: repulsion) b^{65} (black-65, allelic to) x se > ♀ x ♂ >

AB(+)	Ab(se)	aB(b^{65})	ab(b^{65}se)	Total
400	126	156	7	689

$689P^2 + 171P - 14 = 0$ $P = (1 - p_1)(1 - p_2) = 0.065 \pm 0.018$

If we assume that $1 - p_2 = 0.5$, approximating to 48 ~ 50% (♀, R.V), we have $p_1 = 0.87$. As this is the repulsion case, the male recombination fraction is $1 - p_1 = 0.130$ (0.094~0.163).

(Example 2: repulsion) b x ma(maroon) > ♀ x ♂ >

AB(+)	Ab(ma)	aB(b)	ab(b ma)	Total
565	230	214	10	1019

$1019P^2 + 333P - 20 = 0$ $P = (1 - p_1)(1 - p_2) = 0.052 \pm 0.013$

If we assume that $1 - p_2 = 0.45$, approximating to 45~ 46% (♀ R.V.), we have $1 - p_1 = 0.115$ (0.086 ~ 0.140) as the male recombination fraction.

These results are at large consistent with the recombination values in the female and male obtained by the back cross technique: hence our revised conclusion seems to be verified. Thus, the mutant genes, st(→ cd, cardinal), se, and ba^{65}, which were once assigned

to the 3rd chromosome should be reassigned to the 2nd. Also the chromosome which bears M, px, and others must be the 3rd, not the 4th. At present no mutant gene is identified in the 4th chromosome. Here we correct our report in DIS 42:81 as follows:

The 16th line: "2-chromosome" for "3-chromosome"
The last line: "3-chromosome" for "2- and 4-chromosomes"
Several places: "cd(cardinal)" for "st(scarlet)".

Merriam, John R. California Institute of Technology, Pasadena, California.

Control of chromosome pairing and the directed segregation of sex chromosomes in XYY males.

The existence of special sites (= collochores) in Xh for regular conjunction of the X and Y chromosomes in males has been demonstrated in spermatogenesis by Cooper (1964). The intercalation of Xe between collochores in In(1)Xh chromosomes with right break distal to NO apparently allows both collochores to pair simultaneously, which Cooper interprets to mean the loss of normally occurring intrachromosomal control over chromosome pairing. Does such behavior influence genetic segregation of the sex chromosomes in XYY males? From crosses of $In(1)w^{m4}/sc^{8}Y/sc^{8}Y$ males (single male/vial) to w^{m4} females we obtained 41 $w^{m4}w^{m4}$ (white eyes) daughters, 427 $w^{m4}w^{m4}Y$ (red eyes) daughters, 351 $w^{m4}Y$ (white eyes) sons and 24 $w^{m4}YY$ (red eyes) sons. Thus for XYY males carrying $In(1)w^{m4}$ segregation is nonrandom in that the Y's separate more often than the 67% expected for nonpreferential segregation (Grell, 1958, X International Congress of Genetics, Proceedings, Vol. II). Comparable crosses of $In(1)m^{m4}/B^{S}Y/y^{+}Y$ males and their $y/B^{S}Y/y^{+}Y$ brothers to y cv v f females yield results, given in Table 1, that each indicate directed segregation but the two sets of results are not significantly different from each other. Any role of the non-functional pole in spermatogenesis to account for the apparent non-randomness of segregation is difficult to evaluate and cannot entirely be ruled out. However, it may be noted for the three crosses that each of the three elements is recovered in 50% of the gametes and that complementary segregation classes are approximately equal, which suggests that the observed classes adequately reflect disjunction patterns in MI.

Table 1

Results of crosses of $y/B^{S}Y/sc^{8}Y$ ♂♂ and their $In(1)w^{m4}/B^{S}Y/sc^{8}Y$ brothers to y cv v f ♀♀

Fathers	Progeny Classes						
	y^{+}♀♀	y^{+} B^{S}♀♀	y ♀♀	y B^{S}♀♀	y^{+} cv v f♂♂	y cv v f B^{S}♂♂	y^{+} cv v f B^{S}♂♂
y	238	--	142	217	239	286	77
w^{m4}	294	158	--	--	215	232	41

Since $In(1)w^{m4}$ males present a different configuration in the first meiotic metaphase from males with a normal X but yield almost identical segregation classes, it may be asked whether the inferred intrachromosomal control over conjunction of collochores in spermatogenesis has any function in determining disjunctional patterns of conjoined chromosomes. Nonrandom assortment is consistent with the three sex chromosomes being associated at MI, as suggested by the trivalent formation observed cytologically. In this sense the role of collochores in chromosome conjunction is not questioned, although the simplest view of the cytological pictures predicts predominantly X - YY segregation, which is not observed. In(1)Xh chromosomes are also of interest because of their behavior when homozygous in oogenesis: X-chromosome exchange is reduced in XXY females relative to their XX sisters. Possibly a relation exists between such behavior in females and the loss of control hypothesized from cytological observations in spermatogenesis.

Yoshikawa, I. and T. Shiomi. Nagasaki University, Japan. Effects of X-rays on polygenes controlling the sternopleural bristle number in D. melanogaster.

To obtain fundamental information about the genetic effects of radiation on the quantitative characters, the increase rates of variance resulted from radiation-induced mutations for the sternopleural bristle number have been estimated.

An isogenic line extracted from a wild population of Erie, Pa., U.S.A., was used. The males were irradiated with X-rays at 250, 500, 1000, 2000, 3000, and 4000 R. Immediately after irradiation, the males were crossed to the virgin females of the same line in half pint milk bottles. The number of sternopleural bristle in F_1 females and males which had hatched on or before the 13th day after the mating were scored, and the means and the variances were calculated to test the heterozygous effects of radiation-induced mutations. The results are presented in the table.

Table. Means and variances of sternopleural bristle numbers in F_1 flies.

Doses		Control	250R	500R	1000R	2000R	3000R	4000R
Means	females	17.83	17.82	17.88	17.96**	18,21**	18.27**	18.53**
	males	16.63	--	16.76**	16.71*	16.84**	--	--
Variances	females	2.4374	2.5314	2.6971**	2.5860*	2.7734**	2.9395**	3.4099**
	males	1.8673	--	2.1083**	2.0878**	2.1657**	--	--
No. of tested genomes	females	5618	5916	4992	4608	3035	1853	847
	males	2617	--	2332	2464	1612	--	--

*Significantly different from the control at the 5% level.
**Significantly different from the control at the 1% level.

The variances and the means in sternopleural bristle number of the irradiated groups have approximately linearly increased with doses in both sexes. The increase rates of variance were 2.04×10^{-4}/ R in females and 1.25×10^{-4}/ R in males, and those of means were 1.77×10^{-4}/ R in females and 0.90×10^{-4}/ R in males.

Van Delden, W. Genetisch Instituut, Rijksuniversiteit te Groningen, Haren (Gr.), The Netherlands. Kurtosis as an indication of fitness.

King (1963) found that the frequency distribution of egg hatching time in heterogeneous randomly breeding populations of D. melanogaster was leptokurtic and that of an inbred strain platykurtic. He suggested that leptokurtosis should indicate a well integrated genetic system, platykurtosis a badly integrated one.

A number of populations, originally started from an inbred strain of D. melanogaster (Van Delden, D.I.S. 42:62) provided the opportunity to see whether kurtosis is useful as a fitness indicator. The 12 populations tested for kurtosis of egg hatching distribution, differed greatly in fitness (using measures as productivity, larval survival, etc.) and genetic variance. The experimental procedure was the same as used by King. During one month each population was tested four times with weekly intervals. The calculated kurtosis (g_2) values were all positive, even the original inbred strain, which was tested simultaneously, showed considerable leptokurtosis. Though all g_2 values were positive, nevertheless a correlation could still exist between fitness and the positive g_2 values. To test this possibility populations were ranked for productivity and for g_2 values and Spearman's rank correlation coefficient was calculated: $r_s = -0.172$ ($t = 0.55$, d.f. = 10, $P = 0.30$). So no correlation is found. Also for the distribution of weights of individual flies no relation was found between kurtosis and fitness. It seems that, at least in these populations, kurtosis is not very useful as an indication of fitness.

Scharloo, W., K. A. Schuitema, J. G. Wijnstra and A. Zweep. Genetisch Instituut, Rijksuniversiteit te Groningen, Haren (Gr.), The Netherlands. Selection on temperature sensitivity of the expression of a cubitus interruptus mutant.

Waddington and Robertson (1966) showed that the sensitivity of the expression of the Bar mutant can be changed by suitable selection. We applied their selection scheme on the expression of ci^{D-G}. The progeny of the selected flies was reared at two temperatures (22.5° and 27.5° Celsius). The length of the fourth vein (expressed as a percentage of winglength) is larger at the lower temperature.

The following selection lines were started.

1. Two lines in which from the cultures grown at 27.5° the flies with the longest fourth veins were selected and from 22.5° the flies with the shortest veins.
2. Two lines in which the flies with the shortest veins from 27.5° and the flies with the longest veins from 22.5° were selected.

In both selection types there is, besides the selection against temperature sensitivity in 1. and selection for sensitivity in 2., disruptive selection. In earlier experiments at 25° (Scharloo, Hoogmoed and Ter Kuile, 1967 and D.I.S. 41:96) it was shown that disruptive selection with random mating (D^R) does not affect the environmental variance but that disruptive selection with compulsory mating of opposite extremes (D^-) increases the environmental variance. Both modes of disruptive selection were practised in our experiments. In the D^R lines the selected flies were mated at random, in the D^- lines ♀♀ from 27.5° were mated with ♂♂ from 22.5° and vice versa. After 24 hours the ♂♂ were discarded and the ♀♀ transferred to one culture. In the lines selected for temperature sensitivity the expression difference between the 22.5° and 27.5° cultures increased rapidly to more than two times the original difference after 5-10 generations of selection. Progress was most rapid in the D^- line where selection for sensitivity and the disruptive selection act in the same direction.

In the D^- line selected against temperature sensitivity the difference did not change very much. Here an equilibrium seems to occur between the selection against temperature sensitivity and the disruptive selection. In the D^R line selected against temperature sensitivity the expression difference between the 27.5° and 22.5° cultures decreased rapidly until in generation 7 it was almost 0. But in later generations the difference regressed to half its original value.

MacIntyre, Ross. Cornell University, Ithaca, New York. Hybridization of subunits of Acid phosphatase-1 from D. melanogaster and D. virilis.

If crude homogenates from both species are subjected to electrophoresis and the gels stained for phosphatases, a definite difference in the migration rates of the most prominent soluble acid phosphatase in the two species can be seen. The isozymes controlled by the $Acph\text{-}1^A$ or $Acph\text{-}1^B$ allele of D. melanogaster migrate to the anode at pH 8.7 (about 1.5-3.0 cm. under the conditions specified by MacIntyre, DIS 41:61). The isozyme from D. virilis (only one stock has been examined to date) migrates to the cathode, about 2 cm. from the origin, under those same conditions. MacIntyre and Dean (Nature 214: 274) found that partially purified Acph-1 in D. melanogaster can be reversibly dissociated into polypeptide subunits by exposure to high or low pH. Inactivation of the enzymes from D. melanogaster occurs between pH 3.0 and 2.6 and between pH 10.3 and 10.8. The acid phosphatase from D. virilis is completely inactivated between 3.1 and 2.7 and between 10.4 and 10.9. If a mixture of acid or alkalai inactivated acid phosphatases from the two species is dialyzed against certain buffers at pH 6.5, some activity is regained. Electrophoretic patterns from these reactivated mixtures show a zone of activity midway between the positions of the melanogaster and virilis enzymes. This indicates the formation of an interspecific "hybrid" isozyme in vitro during reassociation of the subunits into active enzymes. Besides being a powerful tool with which to identify homologous genes through analysis of their protein products, the measurement of specific activities of the "hybrid" enzymes from different species should provide information on the divergence of the Acph-1 gene within the genus Drosophila. Work on quantification systems for both enzyme activity and protein in reassociated and electrophoresed mixtures is underway.

DeMarinis, F. and F. Sheibley. Cleveland State University, Cleveland, Ohio. The effects of glutaramic acid and glutaramide on the eye size of Bar series.

It has been shown by Kaji (Annot. Zool. Japan 29:23, 1956) that the amide group $-CONH_2$ was actively responsible for increasing the number of facets in the Bar eye. Further, the present authors showed (Abs. Proc. XVI Inter. Cong. Zool. 2:203, 1963) that double amides of the type $NH_2CO(CH_2)_nCONH_2$ are more effective in increasing the number of facets in Bar. The present research note reports on the comparative action of glutaramide, $NH_2CO(CH_2)_3CONH_2$, and glutaramic acid, $COOH(CH_2)_3CONH_2$, on the Bar series, that is the effect of substituting a $-COOH$ group for a $-CONH_2$ group on one side of the chain.

The experiment was carried out by adding to Pearl's formula proportionate amounts of the amide by weight. Eggs of different Bar stocks, B, BB, B^3, BB^3, BB^{36b}, and B^iB^i were permitted to develop on these experimental food mixtures. The effect on eye size was determined by counting the number of facets in the males. All tests were carried out at 25° C. The results are shown in Table 1.

Table 1

The effect of glutaramide, $NH_2CO(CH_2)_3CONH_2$, and glutaramic acid, $COOH(CH_2)_3CONH_2$ on eye size of the Bar series (♂♂ at 25°C.)

♂♂ genotype	2% glutaramide no. facets	2% glutaramic acid no. facets	control no. facets
B	over 600	112 ± 4.2	110 ± 3.2
B^3	over 600	244 ± 12	128 ± 7.5
BB	65.8 ± 2.6	28.1 ± 0.54	28.6 ± 0.66
BB^3	266 ± 9.6	98.1 ± 7.1	94.9 ± 6.2
BB^{36b}	253 ± 13	--------	70.1 ± 1.9
B^iB^i	388 ± 12	138 ± 4.5	134 ± 4.2

In each case 2% glutaramide significantly increases the number of facets of each genotype as compared to control. It will be noted that B and B^3 respond very much the same to glutaramide, that is both genotypes are restored to wild phenotype eye. When each genotype is doubled on the same chromosome as in BB and BB^3, their response to glutaramide are quite different, BB increasing to only 65.8 ± 2.6 facets while BB^3 increases to 266 ± 9.6 facets. On 2% glutaramic acid these same genotypes give quite a different picture; B^3 is affected (244 ± 12) while B is not (112 ± 4.2 facets). In the double Bar, BB shows no increase (28.1 ± 0.54 facets) while BB^3 definitely shows an increase (98.1 ± 7.1 facets). This type of evidence not only confirms the individual genetics identity of these stocks but also shows a genotypic specificity to individual compounds.

Komma, D. J. and G. Carmody. Columbia University, New York. Interspecific variation in a "sex peptide" in Drosophila.

A molecular variant (M^{Zw}) of glucose-6-phosphate dehydrogenase (G-6-PD) in D. m. binds to a protein found in males only. The protein has the effect of decreasing the mobility of G-6-PD in starch-gel electrophoresis.[1] Several other species of Drosophila have been tested by homogenizing their males together with M^{Zw} D. m. females. Males of D. simulans, D. willistoni, D. tropicalis, D. insularis and D. paulistorum show an effect on G-6-PD migration similar to that of D. m. males. Males of "Girardot", a "bridge strain" capable of making fertile hybrids with both D. paulistorum and D. equinoxialis[2], retard G-6-PD migration more than do D. m. males, and males of D. equinoxialis seem to increase migration slightly. No females were found that had any effect on G-6-PD migration. The data indicate that this "sex peptide" is quite widespread in Drosophila. Whether it has any functional relationship to G-6-PD in normal flies is not known.

References: 1. Komma, D. J. Biochem. Gen. 1 (3) December, 1967.
2. Carmody, G. R. D.I.S. 40: 53. 1965.

Aubele, Audrey M. Zoologisches Institut, University of Zürich, Switzerland. Morphological nature of the "palp-like" structure in erupt mutants.

Extreme manifestations of the mutant erupt have been characterized as having a "palp-like" growth protruding from the area of the eye which is normally composed of ommatidia. Other manifestations of erupt are characterized as having holes or breaks in the ommatidial region or by the presence of an additional bristle at the antero-dorsal margin of the eye. Closer examination of the abnormal growths in erupt mutants by means of whole head preparations and preparations of eye imaginal discs which were implanted in late third instar larvae and subsequently retrieved after metamorphosis of the host larvae has revealed information concerning the nature of these growths.

In all cases examined the so-called "palp-like" growth is in no way related morphologically or developmentally to the true palp of D. melanogaster which arises during development from the antenna portion of the eye-antenna imaginal disc. All abnormal erupt growths observed were composed of material derived solely from the eye portion of the eye-antennal imaginal disc. In the case of well-defined growths in both the Swedish-b erupt and the Su-er$^+$ b bw; er st strains, this material was identified as an additional set or sets of trichomes forming the frontal ridges and the frontal, orbital and fronto-orbital setae normally associated with the frons and antero-dorsal eye margin. The material forming the erupt growths may be located in the ommatidial region adjacent to the normally occurring trichomes and setae of the eye margin or may be spatially removed and separated from these normal areas by surrounding ommatidia. The additional bristle described at the eye margin of some erupt mutants is, in all cases here observed, an additional orbital seta. The erupt growths form a spectrum with many degrees of differentiation. Some erupt growths cover the area of only one or two ommatidia and are composed of poorly developed trichomes and setae. Under the dissecting microscope these poorly developed erupt growths appear as a hole or break in the ommatidia.

Further studies with respect to the behavior during development of the eye imaginal discs from erupt strains are now in progress.

Douglas, William L. Howard University, Washington, D.C. Substrate specificity of octanol dehydrogenase and alcohol dehydrogenase in different species of Drosophila.

Substrate specificity studies of ODH and ADH activity in D. melanogaster, D. busckii, D. metzii, and D. unipunctata are being carried out. Crude homogenates of single females at the height of egg laying have been assayed using agar gel electrophoresis. For the substrates methanol, ethanol, 1-butanol, 2-butanol, isobutanol, t-amyl alcohol, isoamyl alcohol, cyclohexanol, 1-octanol, 2-octanol, strong ADH activity as judged by formazan staining has been observed, confirming the results of Courtright, Imberski, and Ursprung, 1966, Genetics 54: 1251-1260. Present work also confirms these authors in the finding that ODH activity in D. melanogaster is obtained only with primary hexanol, heptanol, and octanol. In D. busckii strong ODH activity has been found using cyclohexanol. The finding by Pipkin (in Courtright, Imberski, and Ursprung, 1966) that little or no ADH activity is detectable in D. metzii with formazan staining using ethanol or primary six, seven, and eight carbon alcohols has been confirmed in the present work. In addition no ADH activity in D. metzii has been observed using methanol, isopropanol, 1-butanol, 2-butanol, t-butanol, isobutanol, t-amyl alcohol, isoamyl alcohol, cyclohexanol, or 2-octanol. However, present studies have shown formazan staining of both ADH and ODH isozymes of D. unipunctata using cyclohexanol, and also in some individuals, with primary octanol. Pipkin (in Courtright, Imberski, and Ursprung, 1966) previously had reported an absence of both ADH and ODH activity as judged by formazan staining in the electrophoresed supernatant of mass homogenates, centrifuged at 12,500 rpm for half an hour, for three different strains of D. unipunctata, using heptanol as substrate. D. unipunctata has also shown strong ODH activity but only a trace of ADH activity using n-propanol as substrate. This work has been supported by PHS grant GM 14937 and a grant from the Dean of the Graduate School, Howard University.

Ramel, C. and J. Valentin. Institute of Genetics, University of Stockholm, Sweden.

The interchromosomal effect of In (2L) Cy and In(2R) Cy on primary nondisjunction.

In a previous investigation, the interchromosomal effect of the separate Curly inversions was studied on crossing over in the X chromosome (Ramel and Valentin, 1965). It was found that the two Curly inversions, when located in the same chromosome (in cis-position), had a stronger effect on crossing over in X as compared to when the inversions were located in different chromosomes (in trans-position). The investigation reported here deals with the interchromosomal effect of the two Cy inversions on primary nondisjunction of X in the females. The experiments include the separate inversions as well as combinations of them in trans- and cis-position. The females used were heterozygous for a Muller-5 chromosome carrying yellow, y sc^{S1} In S w^a sc^8 (M5 r y, Lüning 1952), and y w sn. These females were crossed to y w sn/sc^8Y males. The Curly inversions were identical with the ones used for the crossing over studies, In (2L) Cy, al^2 Cy, In (2R) Cy, lt^3 sp^2, and Ins (2L + 2R) Cy, al^2 Cy lt^3 sp^2. They are propagated in stock only on the male side against al b c sp females, collected each generation from a common stock. For further details concerning the origin of the inversions, see Ramel and Valentin (1965).

The results are shown in table 1. The interchromosomal effect of the separate inversions was studied in exp. 1. All the females used in this experiment were sisters. It is evident from the data in table 1 that the effect of (2L) Cy and (2R) Cy is quite similar. This result corresponds to the crossing over data, obtained previously, which indicated that the two inversions had an almost identical effect on crossing over in X.

		♀♀		♂♂	
	Autosomes	% Exc.	Total	% Exc.	Total
Exp. 1	+/+	0.04	5079	0.43	3456
	Cy(2L)/+	0.21	5283	0.52	3624
	Cy(2R)/+	0.24	7151	0.59	4798
Exp. 2 a.	+/+	0.23	7694	0.47	5565
	Cy(2L)/Cy(2R)	0.89	6511	1.83	5075
b.	+/+	0.18	7048	0.58	5133
	Cy(2L+2R)/+	1.23	7081	2.72	5140

Table 1. Primary nondisjunction in y sc^{S1} In S w^a sc^8/y w sn ♀♀ x y w sn/sc^8 Y ♂♂.

In exp. 2 a comparison was made of the combination of the two inversions in cis- and trans-position. All the female genotypes required could not be obtained from one common parental cross. To be able to still compare the inversion carrying females with sister control females, the two inversion series (a. and b., table 1) had separate controls. There were no significant differences between the two controls, however, which allows a direct comparison of the two inversion crosses. It can be seen that the frequency of exceptions is higher in the series with the Cy inversions in cis-position. The difference is not quite significant for the female exceptions (χ^2_c, 1 d.f.= 3.4), but significant at a one percent level for the male exceptions (χ^2_c, 1 d.f.= 8.7). It can thus be concluded that the stronger interchromosomal effect of the Cy inversions in cis- as compared to trans-position, does not only apply to crossing over, but also to primary nondisjunction.

A tentative explanation of this difference between the two combinations of Cy inversions, would be that the meiotic synapsis for some reason is affected differently. In such a case it might be assumed that the combination of the two inversions in cis- and trans-position should affect crossing over in chromosome 2 differently. An attempt was made to study this point by measuring crossing over between the two inversions in cis- and trans-position. The results are shown in table 2. As can be seen in the table, no difference can be traced between the two series. Thus no evidence was obtained indicating a different effect on synapsis, exerted by the position of the inversions.

TABLE 2

Genotype of mother	Phenotype of offspring al Cy sp	al Cy+	+ + sp	+ + +
al In(2L)Cy +/+ In (2R) Cy sp	9	11248	13507	9
al Ins(2L+2R) Cy sp/ + + +	10901	10	12	14786

Table 2. Crossing over between the two Cy inversions in ♀♀ crossed to al sp/al sp ♂♂.

Further investigations involving combinations of other inversions, are in progress.

References: Lüning, K. B. 1952. Acta Zoologica 33:193-207.
Ramel, C. and Valentin, J. 1965. Hereditas 54:307-313.

Lefevre, G. San Fernando Valley State College, Northridge, California. Tests for deficiencies in the vicinity of the w locus.

Routinely in this laboratory, as well as in others, mutants suspected of being deficient for genetic material in the vicinity of the w locus are tested by crossing to w^{258-45}, a short deficiency extending from 3B3 through 3C2 (not just 3C1, as previously described by Bridges and Brehme in the "Mutants of Drosophila") and to w^{m4L}-rst^{3R} (w-), deficient for 3C2-3. Both of these known deficiencies can be "covered" by appropriate duplications: w^{258-45} by Dp w^{Vco} and Dp w^{m49a7} ; w^{m4L}-rst^{3R} by Dp w^{+51b7}, Dp w^{m49a7}, Dp $N^{264-58a}$ and Dp(w-ec)^{64d}, among others. The failure of nonduplication-bearing w^{258-45} / mutant females to survive implies that a deficiency exists in the mutant chromosome just to the left of the w locus; similarly, the failure of w^{m4L}-rst^{3R} / mutant females to survive points to a deficiency located just to the right of the w locus. If neither type of female survives, a deficiency surrounds the w locus.

It is now apparent that these tests may lead to erroneous conclusions regarding the extent of deficiencies near w, especially with regard to the use of w^{m4L}-rst^{3R}. In point of fact, males deficient for 3C2-3C6, phenotypically white, roughest, and "vertical" (absence of one or more vertical bristles, and attributed by Gersh (Genetics 51:477-480) to bands 3C5-6) occasionally survive. Males deficient for 3C2-4 survive more readily, expressing white and roughest phenotypes. Males deficient only for 3C2-3 survive in appreciable numbers, though late hatching, and appear white-eyed. (Mutants with the characteristics just described were obtained by M. M. Green from his mutable w^{c} stock and cytologically analyzed in this laboratory.) One may also recall that rst^{2} males, deficient for 3C4-6, are far from lethal and can be obtained readily without duplication.

In light of the apparent nonlethality of these deficiencies, the problem of interest becomes to explain the lethality of w^{m4L}-rst^{3R}, deficient only for 3C2-3. The answer must lie in the fact that w^{m4L}-rst^{3R} is inverted, with the right breakpoint in the proximal heterochromatin. The consequent position effect on rst, together with the deficiency for 3C2-3 (perhaps augmented by a position effect on 3C1), combine to produce lethality (synthetic lethal?) even though each of the components alone, i.e., In(1)w^{m4}, In(1)rst^{3}, and Df 3C2-3, is viable. Evidence that the typical lethality of w^{m4L}-rst^{3R} involves position effect stems from the observation that, when raised at elevated temperatures (29°C), some males survive, white-eyed and roughest. High temperature, like an extra Y chromosome, suppresses variegation. In any event, it is now apparent that deficiencies lethal in combination with w^{m4L}-rst^{3R} must possess a more extensive loss than just 3C2-3. The system will not detect loss of 3C3 alone, for example; nor as yet can a phenotype be ascribed to the loss of 3C3. This band can not contain a lethal locus, as supposed by Lefevre and Wilkins (Genetics 53: 175-187); but its absence in the recombinant chromosome w^{mJL}-rst^{3R}, in conjunction with the position effect on 3C4, is sufficient to produce lethality in XY males raised at normal temperatures.

It should also be pointed out that lethality of a deficiency in combination with w^{258-45} does not require that the lethal effect be attributed to the loss of 3C1. The lethality could be in the right portion of the 3B region (3B3 or 4); deficiency for 3C1 may not, by itself, be lethal. However, mutants having precisely the required cytological characteristics are not as yet available to test the viability of deficiency for 3C1 alone.

As a final note on the analysis of deficiencies near w, coverage by a specific duplication does not guarantee that the deficiency is shorter than the duplication. For example, Dp w^{Vco}, extending from 2C1-3C4, permits the ready survival of males deficient for all of the 3C material from 3C1 to 3C6, since deficiency of 3C5-6 alone is not lethal.

Kakpakov, V. T. and V. A. Gvosdev. Kurchatov's Institute of Atomic Energy, Moscow, U.S.S.R.
Maintenance of diploid embryonic cells of Drosophila melanogaster in vitro.

Embryos of wild stock (Oregon R-C) were collected after oviposition during the night (12-14 hours).
Eggs were dechorionizated in 3 per cent sodium hypochlorite for 6 minutes. They were then rinsed, gently homogenized, centrifuged and the sediment was suspended in C-12 medium, the concentration of cells being no less than 1.5 millions per ml.

In H-5 and H-6 media of Horikawa and Fox (1964, 1966) the cells in our experiments quickly died. Therefore we used C-12 medium.

In this medium the cells quickly aggregated and adhered to the glass at 27°C. Cell growth was observed mainly in multilayer clumps of small cells adhered to the glass. Mitosis however were detected also in large flat fibroblast-like and epithelial-like cells outside of these clumps.

The number of chromosomes was counted in colchicinated cells after hypotonic treatment and staining with aceto-orcein. After 40 or more days the cells in our medium maintained diploid chromosome sets while Horikawa and Fox (1964, 1966) as well as Dolfini and Gottardi (1966) observed in H-5 medium only 20-50 per cent of normal mitosis already after 2-10 days.

Days of culture	Number of chromosomes 4	6^x	8	No. metaphases analyzed
18	1	5	138	144
44	-	2	167	169

[x]The fourth pair was not detected

During the period of observation (more than 40 days) the formation of multilayer clumps of small and colonies of large cells was observed as well as intensive incorporation of H^3-thymidine in both types of cells.

References: Horikawa, M., Fox, A.S., 1964, Science, 144: 1437.
Horikawa, M., Ling, L.N., Fox, A.S., 1966, Nature, 210: 183.
Dolfini, S., Gottardi, A., 1966. Experientia, 22: 144.

Rinehart, Robert R., and Frank J. Ratty. San Diego State College, San Diego, California.
Further evidence that exchange occurs between chromatids in X-Y detachments.

The preferential recovery of the shorter element of asymetrical dyads produced by the detachment of attached-X rejoining with Y-chromosomes from irradiated oocytes has suggested that the exchanges involved are between chromatids (Parker and McCrone 1958: Brosseau, 1964). This is based upon the observation that disjunction is not normal at the second meiotic division when there are structurally different homologs in the dyad; the shorter element being recovered more frequently (Novitski, 1951). More direct proof of the involvement of chromatids in detachment events would be the recovery of an attached-XY and a complete-Y from the same gamete which would be a rare simultaneous detachment and non-disjunction. Such a product was recovered in an experiment in which newly eclosed Drosophila females containing a reversed metacentric attached-X (y v) and a doubly marked Y-chromosome ($y^+Y \cdot B^S$) were irradiated with 3000 r and mated with y ct B (Binscty) males containing a normal Y. Subsequent testing demonstrated that the genotype of the non-disjunction and detachment female recovered was y v $\cdot$ y^+ / y^+ Y $\cdot$ B^S and the y ct B paternal-X. This event was recovered in a sample of eggs collected from a single female. No unexpected non-disjunction types were observed among the offspring of the female producing the detachment and non-disjunction, therefore it is probable that no extra Y-chromosome was present in the gamete from which this event was derived. This observation is further proof of the chromatid nature of X-Y detachment in oocytes of Drosophila females.

Doane, W. W. Yale University, New Haven, Connecticut. The Amylase locus in Drosophila hydei.

Recombination experiments were performed to determine to which linkage group the Amy locus belongs in D. hydei. Berendes (1963, Chromosoma 14: 195) showed that linkage group V (referred to as III by Spencer, 1949) belongs to chromosome 5 in hydei. This is homologous to 2R in melanogaster, wherein lies the Amy locus of the latter species (e.g., Kikkawa & Abe, 1960).

Females from a Chile wild strain, homozygous for the electrophoretic variant Amy^8 (Doane, DIS 41: 74), were crossed to males from a strain homozygous for Amy^7 and which contained the recessive markers bb, p and vg in linkage groups I, II and V, respectively. The reciprocal cross was made and F_1 females of both crosses were testcrossed to males of the marker strain. Progeny were aged one week and each recombinant class homogenized en masse in an equivalency of 1 fly/5µl of water. Cell debris was spun down and 10 µl samples of supernatants were analyzed quantitatively by disc electrophoresis (method of Doane, 1967, J. Exp. Zool., 164: 363). In those cases where both bands #7 and #8 were separated from a given class, determination of the relative activity in each band provided estimates of the frequency of the two Amy alleles. Results showed that Amy is linked with vg, i.e., in linkage group V. Similar experiments using st, sca and jv (groups II, V and III, resp.) confirmed this finding.

A 3-point crossover analysis was next made using sca and cn as markers linked with Amy^7 on the fifth chromosome. Flies from the following cross were aged one week and tested electrophoretically: sca cn Amy^7/+ + Amy^8♀ x sca cn Amy^7♂. Recombinants between markers were tested individually; parental types were tested from mass homogenates of 20 flies or less, as above. In a subsequent 4-point analysis of a parallel nature, vg was included among the markers and a reciprocal cross also made. Testcross results are summarized in the table with the maternal genotype for each listed at the left.

Maternal Genotype	No. of Progeny	Number of Crossovers Between Markers					
		sca-cn	sca-Amy	sca-vg	cn-Amy	cn-vg	Amy-vg
sca cn Amy^7	927	186	210		42		
+ + Amy^8		(20.1%)	(22.6%)		(4.5%)		
sca cn Amy^7 vg	715	122	166	182	52	112	76
+ + Amy^8 +		(17.1%)	(23.2%)	(25.4%)	(7.3%)	(15.7%)	(10.6%)
+ + Amy^8 + *	877	150	190	222	48	100	72
sca cn Amy^7 vg		(17.1%)	(21.7%)	(25.3%)	(5.5%)	(11.4%)	(8.2%)

*From reciprocal mating.

The linear sequence of markers with reference to Amy is: sca cn Amy vg, with no stipulation as to the direction it is read. This is in contrast to the sequence in melanogaster which reads cn sca vg Amy. The % c.o. between each marker, when averaged from the data of all three experiments, is: sca-cn, 18.1%; cn-Amy, 5.8%; and Amy-vg, 9.4%. When the data is corrected for the lowered viability shown by the parental type with the multiple markers, the values become: 17.2%, 5.5% and 8.9%, respectively. (Supported by N.S.F. grant GB 1718. The marker strains were gratefully received from Dr. H. D. Berendes.)

Holm, D. G. University of Connecticut, Storrs, Connecticut. Construction of a dor^+Yy^+ chromosome.

X-rayed y^2 su-w^a w^a $Y^{S \cdot}Y^L$ y^+ (no free Y) males were mated to yf:= females. The viable y^+ female progeny each carry a Y chromosome obtained by a gross deletion of the paternal X. Subsequent mating to dor males permitted the recovery of a newly synthesized Y carrying a dor^+ duplication. Additional analysis revealed that this duplication covered the distal tip of the X including y^2 su-w^a dor^+ and covers 1(1)7. The proximal portion of the duplication includes the region from l114 to su-f (see Schalet and Finnerty, this issue). The presence of the dor^+ Y y^+ chromosome in males gives rise to reduced body size and a pronounced Hairy-wing effect; in compound-X females the body size is apparently normal and the Hairy-wing effect is poorly and infrequently expressed.

Courtright, James B. The Johns Hopkins University, Baltimore, Maryland. Alcohol dehydrogenases (ADH) and octanol dehydrogenases (ODH) in D. melanogaster: Immunochemical differences.

Two clusters of alcohol dehydrogenases, ADH and ODH, with different substrate specificities, have been separated by electrophoresis on agar gels (Ursprung and Leone, J. Exp. Zool. 160:147, 1965). Each group was found to be controlled by a single genetic locus. ADH maps at 2-50.1 (Grell et al., Science 149:80, 1964), ODH at 3-49.2 (Courtright et al., Genetics 54: 1966). It should be noted that both loci are within four Morgan units of the centromere on the respective chromosomes. It seemed possible therefore that the two gene-enzyme systems represented a case in which a locus became duplicated and subsequently translocated to a different chromosome. It was decided to investigate whether these two enzymes were immunochemically related.

Crude extracts of Drosophila melanogaster in 0.1 M Tris-HCl, pH 8.0, were adjusted to a protein concentration of 1 mg/ml, and a total of 2.5 mg were injected suprascapularly with 2.5 ml of Freund's complete adjuvant into each of two adult New Zealand white rabbits. Subsequent injections were carried out every 6-7 days with 2.5 mg protein and 2.5 ml of Freund's incomplete adjuvant. The rabbits were bled from the external marginal ear vein one week after the fourth injection. The serum collected was dialyzed against 18% and then 15% sodium sulfate and the final precipitate re-dissolved in H_2O and dialyzed against 0.1 M NaAc pH 5.8. The resulting gamma globulin was dialyzed against 0.1 M Tris HCl pH 8.0.

ADH and ODH were separated from one another as previously described (Courtright et al., l.c.). Agarose (Nutritional Biochem.) was used for electrophoretic separation; on agarose gels, ADH migrates to the cathode, ODH to the anode.

Aliquots containing equal activities of ADH, ODH, or of a 1:1 mixture of ODH and ADH were applied to a circular well with a diameter of 4 mm in an agarose gel and electrophoresed for 10 minutes at 250 volts. Slits parallel to the direction of migration were then cut and filled with gamma globulin prepared from rabbits injected with the Drosophila extract. Double diffusion was allowed to proceed for 48 hours in the cold, at which time the gels were washed with several changes of Tris HCl, pH 9.0 for two days. The precipitin bands were stained directly for ADH and ODH activity with a solution of 5 mg Nitro Blue Tetrazolium (Dajac), 0.4 mg phenazine methosulfate, 50 mg NAD, 20 ml 0.5 M Tris-HCl, pH 9.0, and 0.5 ml of either ethanol or octanol. Bands of formazan deposition which coincided with the precipitin lines were found on the cathode side of the well but never on the anode side. No precipitin lines were detected when the well contained only ODH. The precipitin line patterns of both the ADH and the ADH-ODH mixture were identical. It is concluded that anti ADH antibodies do not precipitate ODH molecules under these conditions.

Supported by NIH training grant GM-5708 and NSF grant GB-4451.

Valencia, R. M. Atomic Energy Commission, Buenos Aires, Argentina. X;autosome and Y;autosome translocations.

In a study of total genetic damage induced in entire genomes of mature spermatozoa by 4000r of X-rays (Abstr. Eb-5, p. 85, Rad. Res. Soc., May 1967), translocations were collected in both X-bearing and Y-bearing genomes. Since the genomes were collected in the daughters of the irradiated males (regular daughters for X-bearing genomes and exceptional daughters for Y-bearing genomes), and both X and Y were kept in the female until fertility could be determined, no translocations were lost due to male lethality or sterility. In 181 X-bearing genomes there were 6 T(X;2)s, 18 T(2;3)s and 1 T(2;4). In 129 Y-bearing genomes there were 6 T(Y;2)s, 9 T(Y;3)s, 1 T(Y;4), 11 T(2;3)s, 1 T(2;4) and 1 T(3;4). The frequency of T(A;A)s is 10.5% in X-bearing and 10.1% in Y-bearing genomes, but the frequency of T(X;A)s is 3.3% and that of T(Y;A)s is 12.4%. Thus T(Y;A)s were much more frequently recovered than T(X;A)s under exactly comparable circumstances. The lack of T(X;3)s is to be noted, however, and is unexplained, but even if there had been an equal number of T(X;3)s as of T(X;2)s, the frequency of T(X;A)s would still have been considerably less than that of T(Y;A)s. The greater recoverability of Y translocations might be ascribed to a higher frequency of induction due to the heterochromatic nature of the Y, but most likely it is due to a higher frequency of loss of X translocations.

(Work supported at beginning by NSF grant GB-344 and ORNL)

Berendes, H. D. Max Planck Institut f. Biologie, Abt. Beermann, Tübingen, Germany. The effect of ecdysone analogues on the puffing pattern of D. hydei.

Changes in the puffing pattern of mid-third instar salivary gland chromosomes occurring after injection of two ecdysone analogues have been compared with the changes observed after injection of ecdysone (1). The two compounds tested were: 2β,3β,14α,20ζ,22β$_F$, 25-hexahydroxy-Δ^7-5β-cholestenon-(6), described as ecdysterone, crustecdysone or polypodine A (2), and 2β,3β,dihydroxy-5β-cholestenon-(6). Both compounds were kindly supplied by Prof. Šorm. The ecdysterone isolated from Polypodium vulgare L. differs in its chemical composition from ecdysone (2β,3β,14α,22β$_F$, 25-pentahydroxy-Δ^7-5β-cholestenon-(6); (3)) by one additional hydroxyl group. The second compound contains three hydroxyl groups less than ecdysone and the Δ^7 double bond is absent. This sterol derivate inhibits the post-ecdysial hardening and sclerotization of the cuticle of the bug Pyrrhocoris apterus L. For this reason it was assumed that this substance has an ecdysone-antagonistic action (4).

Injection of 1 µl of a Ringer solution containing 25 µg of ecdysterone into mid-third instar larvae resulted in drastic changes of the puffing pattern of the salivary gland chromosomes. Observations made at 10 min. and at 4 hours after injection demonstrated the presence of the complete series of activity changes of the chromosomes known to occur during a 6 hour period preceding puparium formation (5) and also after experimental administration of ecdysone (1).

Injection of the second compound (15 µg/µl) also produced the activation of ecdysone-specific puffs. The puffs 78B (fig. 1), 87C, 95D and 97A become active or increase their activity within 10 min. after injection, and puffs 77BC and 61B become active at 4-5 hours after injection. In contrast to the observed effect of this substance on the cuticle hardening and sclerotization of Pyrrhocoris, no indication of an ecdysone-antagonistic action could be detected in D. hydei on the basis of the activity changes at the chromosomal level. Moreover, this substance reproduced the changes in gene activity pattern normally produced by ecdysone. It may be concluded from these data that small changes in the molecular composition of ecdysone, i.e., the addition or subtraction of hydroxyl groups and the removal of the double bond, does not alter the specific effect on the genome of D. hydei.

Fig. 1. Activity of puff 4-78B at 10 min. after injection of 15 µg/µl 2β,3β,dihydroxy-5β-cholestenon-(6).

Further analogues have to be tested to determine the part of the molecule that is responsible for the specific activation and inactivation of particular genes.

Lit: (1) Berendes, Chromosoma 22 (1967); (2) Jizba et al., Tetrahed. Letters 18 (1967); (3) Huber and Hoppe, Chem. Ber. 98 (1965); (4) Hora et al., On Steroids CIII, Coll. Czech. Chem. Comm. (1967); (5) Berendes, Chromosoma 17 (1965).

Klingele, Sr. Dorothy. University of Notre Dame, Notre Dame, Indiana. Recombinational analyses of the lz^D and lz^{61f} alleles.

Recombinational studies involving the spectacle mutants lz^D and lz^{61f} of the lozenge pseudoallelic series were carried out utilizing the female-fertile lz^{50e} allele. Four recombinant males were found among the 10,062 individuals scored in the lz^D series, whereas no recombinants were observed in the 24,096 F_2 males scored in the lz^{61f} experiments. It seems reasonable to suppose a homoallelic relationship between lz^{50e} and lz^{61f} with assignment of lz^{61f} to the left-most sub-locus of the complex. The recovery of recombinants in the lz^D/lz^{50e} series, establishes a heteroallelic nature of these alleles. The calculated map distance, 0.08 units, agrees favorably with the 0.083 map units determined as the distance between the left-most sub-locus and supports the preliminary placement of lz^D at the lz^1 sub-site.

Hihara, F. and Kurokawa, H. Tokyo Metropolitan University, Tokyo, Japan.

Relationship between D. funebris and D. multispina.

D. funebris which is known to be a cosmopolitan species has been recorded from both Hokkaido and Honshu in Japan. On the other hand, D. multispina Okada which closely allied to the former species has been collected in Hokkaido only. In some localities in Hokkaido, these two species appear to have close habitats though they discrete microecologically. D. multispina prefers to inhabit forested areas while D. funebris is apt to inhabit domestic environments.

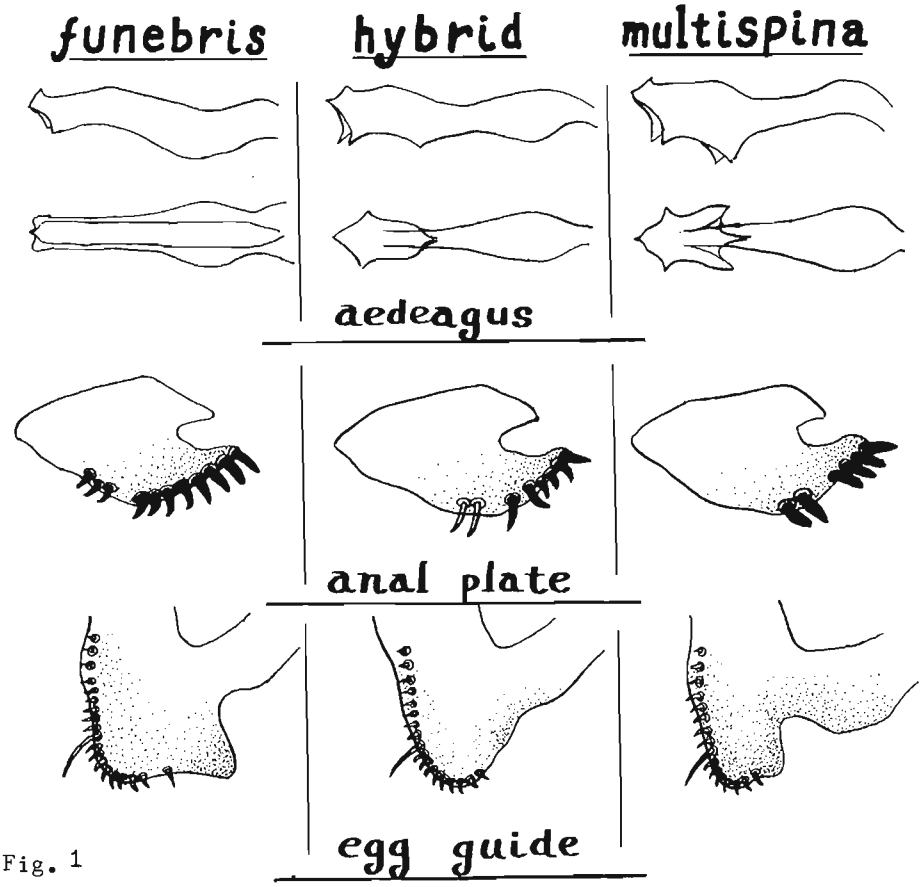


Fig. 1

They are also morphologically similar to each other (Okada, 1956). Only differences in the characters, such as, the shapes of male and female genitalia, and the abdominal sternites were shown (Figure 1). Any difference concerning the ganglionic metaphase chromosomes could not be detected between the two. Sexual isolation experiments were carried out at different temperatures by using male multiple choice technique. The results were summarized in Table 1. In the experiment at 23°C, none of funebris female could be inseminated with multispina male, while a few number of the females carrying alien sperms were seen in the lower temperature at 19°C. It is clear that strong sexual isolation has precluded gene exchange between two species.

Cross experiments between two species were made (Table 2). Hybrid males seemed to be sterile because subsequent backcross experiments using these males were not successful.

Table 1.

Temperatures	Crosses ♀♀ ♂♂	Homogamic N	Homogamic %(+)	Heterogamic N	Heterogamic %(+)	$K_{1,2}$ or $K_{2,1}$	K_1 and K_2	χ^2	P
23°C	f,m x f	100	74.0	98	21.4	0.702	0.851	57.95	<0.001
	f,m x m	98	69.4	100	0.0	1.000		109.79	<0.001
19°C	f,m x f	84	97.6	73	28.8	0.833	0.869	82.09	<0.001
	f,m x m	76	90.8	75	10.7	0.905		95.17	<0.001

K...Isolation Coefficient
f...funebris, m...multispina

Table 2.

Crosses 5♀♀		5♂♂	Pairs tested	No. of offspring ♀♀	♂♂
f	x	f	60	3140	3128
m	x	m	60	1224	1303
f	x	m	200	0	0
m	x	f	200	677	635
$(m \times f)F_1$	x	f	100	2298	1792
$(m \times f)F_1$	x	m	50	365	301
f	x	$(m \times f)F_1$	50	0	0
m	x	$(m \times f)F_1$	50	0	0

Farnsworth, M. W. State University of New York at Buffalo, Buffalo, New York.

Effect of prolonged CO_2 exposure on flight.

In the course of studies of energy metabolism with the Canton S strain of D. melanogaster, we have had occasion to employ thoracic sarcosomes of the adult and have made some observations which may be of interest to any worker employing CO_2 as an anesthetic. Thoraces have been obtained by removing head and abdomen of adults with watchmakers forceps. The procedure can be carried out either with well chilled flies in a cold room or with CO_2 anesthetized flies at room temperature. For the latter method, flies are placed in a dry plugged vial into which is inserted a small glass tube connected to a CO_2 generator consisting of a stoppered sidearm flask containing dry ice. It was noticed that chilled flies returned to room temperature seemed unharmed by the experience, whereas a large number of those exposed to CO_2 intermittently or continuously for periods of 45 minutes or more were unable to fly after recovery from anesthesia. Although the wings could be lifted, flight was not attained even when flies were shaken out of a vial in mid air. Walking and hopping movements, however, were normal. In most such individuals, the condition appeared to be permanent since the inability to fly was still evident the following day and as long as observed thereafter. Microscopic examination showed no abnormalities of thorax or wings. Fertility of males and females was not seemingly affected and the condition was not inherited by subsequent generations. To determine if the effect on flight was induced by anoxia alone, rather than by some more specific effect of CO_2, flies were exposed to a nitrogen atmosphere. The results were similar to those obtained with CO_2 indicating that extended periods of anoxia are a reasonable cause of the deleterious effect on flight. Since walking and body movements were normal, presumably the relevant musculature of the appendages was also normal. In contrast, the more highly specialized flight muscle seemed sensitive to anoxia. One likely site of injury in flight muscle is the sarcosome and thus the possibility that these specialized mitochondria were no longer able to effect coupled oxidation of an appropriate substrate was tested. Sarcosomes were isolated from untreated and CO_2 treated adults and P/O ratios were obtained by conventional Warburg techniques using α-glycerophosphate as substrate. Methodology followed that of Gregg et all (Biochim. Biophys. Acta, 45 (1960) 561). Results of 10 experiments with CO_2 treated flies gave a mean P/O ratio of 1.54 as compared to 1.49 in an equal number of experiments with controls. Oxygen uptake in µatoms/mg protein was 2.42 and phosphate esterified as ATP was 3.73 µmoles/mg protein in the experimental while corresponding values for controls were 2.24 µatoms O/mg and 3.42 µmoles P/mg. Thus, no significant differences in the ability of sarcosomes to effect oxidation and coupled phosphorylation were revealed in treated and untreated groups. It is obvious that the site of injury has not been identified. By analogy with mammalian systems, it is possible that inability to fly after prolonged anoxia may be of neurological origin. In any event, the use of CO_2 or other gases as anesthetic agents may result in physiological side effects requiring assessment in some types of experiments.

(Supported by research grant HD 01240 from NIH).

Kang, Y. S. and K. W. Moon. Seoul National University, Seoul, Korea. Drosophilid fauna in the vicinity of the demilitarized zone in Korea.

The demilitarized zone (4 km wide, 249 km long) and its adjacent area have been kept uninhabited and undisturbed for fifteen years. For the studies of relationship between chromosomal inversion and ecological versalities of Drosophila natural population in the areas adjacent to the demilitarized zone, authors made the preliminary survey on Drosophila during the period of April-October, 1967. According to altitude (sea level), three regions were selected as collecting sites; 1) Eastern region, above 1000 m, 2) Central region, below 800 m, 3) Western region, below 300 m.

The majority of flies were attracted to large containers of fermented fruits, bananas, apples and pears. Net sweeping was not enough for the present work because most of suitable areas for net sweeping were mine field. A total of 3407 specimens were represented by 6 genera, 25 species as shown in table 1. Some remarkable was the fact that D. angularis, D. unispina and D. brachynephros were abundant in all regions, but D. coracina, only in central region, D. sexvittata, in eastern region.

Table 1. Numerical data of drosophilid flies collected in the three regions near the demilitarized zone.

Species	Eastern Region	Central Region	Western Region	Total
* Amiota alboguttata	-	6	3	9
* A. variegata	27	26	8	61
* L. magnipalpis	19	2	-	21
* L. maculata	3	4	-	7
* M. japonica	-	3	-	3
* S. graminum	21	26	72	119
* P. pallida	49	78	94	221
D. sexvittata	35	-	-	35
D. coracina	-	146	-	146
D. suzukii	-	47	-	47
D. melanogaster	-	40	37	77
D. clarofinis	-	6	-	6
D. auraria (A.B.C.)	65	514	141	720
D. brachynephros	75	137	69	281
D. angularis	110	224	110	444
D. unispina	109	95	91	295
D. nigromaculata	17	73	25	115
D. kuntzei	63	74	13	150
D. testacea	-	5	-	5
D. bizonata	11	10	38	59
D. histrio	91	55	44	190
* D. tenuicauda	5	4	-	9
D. virilis	47	63	-	110
D. lacertosa	-	166	16	182
D. sordidula	-	68	27	95
Total	747	1872	788	3407

* Collected with the use of net sweeping

Chovnick, A. University of Connecticut, Storrs, Connecticut. A ma-1^2 marked Y chromosome.

From X-rayed y v ma-1^2 $y^S \cdot y^L$ y^+ (no free Y) males crossed to y^+ y sc^{S1} ma-1^{F3} sc^8 females. A fertile y^+ ma-1^+ male was recovered which was y sc^{S1} ma-1^{F3} sc^8/ma-1^2 $y^S \cdot y^L$ y^+. The ma-1^+ phenotype reflects allele complementation seen in ma-1^2/ma-1^{F3} heterozygotes. The duplication of proximal X covers the region from sw through su-f inclusive.

Tedeschi, Maria Valéria* and Luiz Edmundo de Magalhães. Universidade de São Paulo, São Paulo, Brasil. Analysis on the recombination between chromosomes bearing lethal genes in D. melanogaster.

Chromosomes derived from females bearing two different lethals (l_1/l_2), one in each second chromosome, are put in homozygosity by the usual method, with the aid of Cy/Pu balanced lethals strain. They are also tested for allelism with each one of the lethals present in the parent female. Following this scheme, we analyzed two different pairs of lethals (l_1 = 23, l_2 = 30 and l_1 = 88, l_2 = 53); results are shown in table I. We can observe that the frequency in class 3 for the second pair of lethals analyzed (l_1=88/l_2 = 53) is too high in comparison with the complementary class 4. It could be explained if we assume that the chromosome 88 bears two different lethals instead of only one.

TABLE I

Class number	Homozygosity	Allelism test with l_1	Allelism test with l_2	Observed frequency I l_1 = 23 n	Observed frequency I l_2 = 30 %	Observed frequency II l_1 = 88 n	Observed frequency II l_2 = 53 %
1	+	+	-	28	36.36	43	41.74
2	+	-	+	26	33.76	36	34.95
3	+	+	+	10	12.98	19	18.44
4	-	-	-	10	12.98	0	0.00
5	-	+	-	1	1.29	1	0.97
6	-	-	+	2	2.59	1	0.97
7	+	-	-	0	0.00	3	2.91
TOTAL				77		103	

n = number of chromosomes analyzed.
+ = lethal phenotype; - = non-lethal phenotype.

That groups of genes located in a chromosome may act as lethals is not yet a fact very well established, at least in D. melanogaster. Beside the classes 1 to 4, expected as if each lethal chromosome had normal behavior in the recombination, we got three more classes, 5 to 7, which do not fit in the theory of crossing-over recombination between single lethal loci. They can be interpreted as if some lethals depend on more than one single locus.

Chromosomes in the classes 5 and 6 can be interpreted as recombinants bearing the original lethal plus a recessive suppressor system. Chromosomes in the class 7 behave as if the new lethals are synthetized by recombination once they do not show allelism with any of the original lethals.

Further analysis to clarify exactly the nature of the unexpected recombinant chromosomes are underway.

*Pre-doctoral fellow from FAPESP; contract number 67/248.

Shiomi, T. Nagasaki University, Japan. The mutagenic effectiveness of 14.1 Mev neutrons in post-meiotic germ cells of D. melanogaster.

24 hr old adult males of Canton-S isogenic strain were irradiated with doses of 500, 1280 and 2000 rad. of 14.1 Mev neutrons and germ cells irradiated at post-meiotic stages were tested in five successive one day broods for the presence of sex-linked recessive lethals and autosomal translocations (2;3) crossing with a dual purpose stock females (y sc^{S1} In49 sc^{8} ; bw ; st p^{p}). Each male was crossed to five virgin females.

Differ from the results obtained with X-ray irradiation, present results demonstrated no difference between the mutation rates in 1st and 2nd day broods. However, it became clear that the mutation rates were not constant even during the first 24 hr with the mating sequence after neutron exposure. The RBE of 14.1 Mev neutrons as compared with X-rays was demonstrated to be lower for recessive lethals than for translocations at all stages of spermatogenesis tested.

Jenkins, John B. and J. R. Simmons. University of California, Los Angeles, California. The effect of 5 fluorouracil on Drosophila melanogaster.

The compound 5 fluorouracil (FU) has a marked toxic effect on the larvae of Drosophila. This effect is primarily characterized by the failure of the larvae to develop into the pupal stage. Under normal circumstances a first instar larva will pupate in 4 or 5 days, but when such a larva is fed FU, it will remain in the first or second instar stage for as long as 10 days before death eventually occurs. Experiments were designed to determine if FU is incorporated into RNA and/or if FU, in its deoxynucleotide form, inhibits the methylating enzyme, thymidylate synthetase.

Drosophila larvae were fed or injected with FU and at the same time fed or injected with thymidine in varying concentrations, the objective being to see if a reversal of the toxic effect would occur. There was no reversal of the toxic effect at any FU/TdR ratio, either in feeding or injection experiments.

In other experiments larvae were fed 5-FU-2-C^{14} and their nucleic acids were extracted (Kilgore and Painter, 1964, Biochem. J. 92-353-357). Following alkaline hydrolysis, paper chromatography was used and a highly significant amount of radioactive FURP was identified. Further evidence for the involvement of FU in RNA metabolism was obtained by feeding the radioactive FU to either males or females, allowing mating, then collecting and analyzing the eggs. When the males were fed the labeled compound no significant radioactivity was detected in the eggs. When the females were fed the C^{14} containing compound a significant amount of radioactivity was recovered in the eggs. The results thus parallel the difference in RNA concentration found in the sperm and the egg.

While results of these experiments indicate that part of the toxic effect of FU may be a consequence of its incorporation into RNA, other strong possibilities exist. Metabolic derivatives of FU, such as alpha-fluoro-beta-alanine, alpha-fluoro-beta-ureidoproprionic acid or alpha-fluoro-beta-guanidoproprionic acid, might be responsible for the toxicity. Additional experiments are needed to test this possibility.

Scharloo, W. Genetisch Instituut, Rijksuniversiteit te Groningen, Haren (Gr.), The Netherlands. Polymorphism by disruptive selection.

Scharloo, Hoogmoed and Ter Kuile (1967 and D.I.S. 41:96) showed that disruptive selection practised on fourth veinlength in a cubitus interruptus mutant (ci^{D-G}) causes the appearance of bimodal frequency distributions. Analysis of the phenotypic variability suggested different mechanisms for the bimodality in the disruptive lines with random mating (D^R) and negative assortative mating (D^-). Further analysis revealed that in the D^R line the polymorphism is based on a genetic switch mechanism on the second chromosome. Flies with an extreme long 4th vein are homozygous for a plexus allele and flies with an extreme short vein are homozygous for a factor which seems to be composed of two genes in close proximity and is located 2-4 map units to the left of plexus. This factor causes, in the absence of ci^D, an interruption of the 4th vein and the second crossvein. One of its component genes causes the interruption of the 4th vein, the other component which causes the cross vein interruption seems to reveal its presence only in combination with the first one. Flies without ci^{D-G} but homozygous for both the "low" and "high" factors have a complete fourth vein but lack the cross vein.

Polymorphism in the D^- line, however, is based on a developmental switch mechanism. Substitution of chromosomes of the D^- line in other stocks showed that the genetical differences between the extreme flies are small. Bimodality was still present in lines inbred from the D^- line for 20 generations in which selection for low and high expression did not have any effect. Direct evidence for a threshold mechanism in the development of the fourth vein was obtained by temperature experiments. In contrast with the linear relation between temperature and expression in the base population, this relation is markedly-non-linear in the D^- line and in inbreds obtained from it. There seems to be a marked facilitation of response in the range of expression values between the two modes of the frequency distribution.

Marzluf, G. and E. Mayer. Marquette University, Milwaukee, Wisconsin.
Aldolase of Drosophila melanogaster.

A wealth of biochemical information is available about the enzyme, aldolase, which suggests that it is a tetramer composed of two distinct subunits (cf. Chan, Morse, and Horecker, PNAS 57, 1013, 1967). We have examined this enzyme for genetic variability in D. melanogaster. After acrylamide gel electrophoresis of crude extracts of adult flies in Tris-borate buffer at pH 8.9, the enzyme is demonstrated by incubating the gel in a solution containing fructose-1,6-diphosphate, glyceraldehyde-3-phosphate dehydrogenase, NAD, phenazine methosulfate, and nitro blue tetrazolium. In this coupled system, glyceraldehyde-3-phosphate produced by aldolase in the gel serves as a substrate for the exogenously added dehydrogenase which is revealed by the standard dehydrogenase staining method. The activity "bands" obtained are relatively wide, probably because of the indirect method required for staining. The following strains which have been examined to date do not reveal any difference in electrophoretic mobility of their aldolase: Amherst, Canton S, Cockaponsett, Oregon R, Swedish B, Urbana, cn bw, cn bw e, Gl Sb H/Payne, and pr.
(Work supported by grant 1 R01 GM 15055-01 from the USPHS.)

Valencia, Ruby M. Atomic Energy Commission, Buenos Aires, Argentina.
A cytological study of recessive lethals.

In a study of total genetic damage induced in entire genomes of mature spermatozoa by 4000r of X-rays (Abstr. Eb-5, Rad. Res. Soc., May 1967), a total of 203 recessive lethals was collected in all chromosomes. Of these 21 were in the X, 82 in chromosome 2, 94 in chromosome 3 and 6 in chromosome 4. All were analyzed cytogenetically.

134 (66.0%) were point mutations, apparently agreeing quite well with expectation based upon an earlier cytological analysis (Valencia, Hered. Suppl. Vol., 1954) of 81 lethals induced by 700r (85.1% point mutations) and 122 lethals induced by 2800r (71.3% point mutations). This comparison, however, between the previous experiment and the present one, is not valid, due to the fact that lethals associated with 2-chromosome rearrangements were counted differently in the 2 cases. In the present experiment, since lethals were collected in all chromosomes of entire genomes, two "lethal" chromosomes were collected for each lethal translocation or complex rearrangement involving 2 chromosomes. If the results are corrected in order to be comparable to the previous experiment or any typical recessive lethal study, in which lethals are collected in a single chromosome of any particular genome, the results are as shown in the second column of the table. When treated in this way, the present results show an unexpectedly high proportion of point mutation lethals.

	No. "lethal" chromosomes	No. lethals, counting 1 lethal per transl.
Cytol. normal	134 (66.0%)	134 (75.7%)
Inversions	16	16
Translocations	40	20
Complex, 2-chrom.	12	6
Deletion	1	1
Total	203	177

The paucity of deletions is surprising but might be due to failure to detect very small deletions, since the lethals were not localized (as they were in the previous study). Larger deletions, as well as other deleterious rearrangements, might have been less likely to be recovered in this experiment, in which the flies were already quite heavily loaded with markers and rearrangements. This would explain the higher than expected proportion of point mutations.
(Work supported in beginning by NSF grant GB-344 and ORNL.)

Burnet, B., K. J. Connolly, and J. E. Beck. Departments of Genetics and Psychology, University of Sheffield, England. Restoration of visual acuity in an eye color mutant strain by environmental means.

Kalmus (J. Genet., 45: 206-213, 1943) tested the optomotor responses of a number of mutants of Drosophila affecting eye pigmentation. The optomotor responses were used as a measure of visual acuity. One of the findings was that a deficiency of eye pigment reduced visual acuity. Since Kalmus used different strains of flies it is possible that he was recording strain differences in optomotor responses which were independent of eye pigmentation. If the loss of acuity is referable directly to loss of screening pigment, environmental reversal of the mutant phenotype should lead to restoration of visual acuity. A direct test of this possibility can be made using the double mutant strain vermilion; brown of D. melanogaster which lacks both ommochrome and pterin eye pigments. Ommochrome biosynthesis can be restored to v; bw individuals by supplying the missing substrate, kynurenine, in the larval diet thus bypassing the v mutant block.

Optomotor responses were used as a measure of visual acuity. These were measured by placing flies at the centre of a rotating striped circus.

Preliminary experiments indicate that bw flies, lacking pterin eye pigment only, are indistinguishable from wild type whereas v; bw flies show a considerable reduction in optomotor responses. v; bw flies in which brown pigment synthesis has been restored by kynurenine feeding show almost complete restoration of optomotor response to the level observed in the bw control group, as Kalmus's theory predicts. The results are to be reported in detail elsewhere.

Parsons, P. A. Department of Genetics, La Trobe University, Bundoora, Victoria, Australia. A lack of positive correlation between adult density and oviposition rate in D. melanogaster.

A series of experiments were carried out (Table 1) where 20, 100 and 200 pairs of Canton-S and yellow flies were permitted to oviposit on a watch glass in a 1/2 pint milk bottle for a 4 hour testing period. The flies were 4-5 days old when tested, and had been starved for 2 days prior to oviposition, the sexes remaining together for this time.

Table 1

Mean number of eggs per female laid in 4 hours according to adult density
(The numbers in brackets are the number of replicates)

Genotypes	Canton-S			Yellow		
Adult density (pairs of flies)	20	100	200	20	100	200
Experiment 1	0(3)	-	0.317(3)	0.318(8)	0.700(2)	1.525(2)
Experiment 2	0.242(6)	0.935(2)	0.873(3)	0.317(6)	1.630(1)	1.945(3)

For both genotypes, more eggs per female were laid at the 2 higher adult densities compared with the lowest density. More detailed work is needed to show whether the differences between adult densities of 100 and 200 pairs of flies are real. The observation of fewer eggs per female at a low adult density seems opposite to expectation, which is that aggression between flies at high densities would interfere with oviposition. A possible explanation is that there is a "facilitation" phenomenon, whereby it takes a certain period of time for a fly to commence oviposition, and that once any fly begins, others follow. As adult density increases, the chances of any fly commencing quickly would increase. Another possibility is that the presence of the first eggs triggers off further oviposition. The latter possibility was tested by comparing respectively the mean number of eggs laid over 4 hours on watch glasses on which 20 newly laid eggs less than 1 hour old had been placed, and the mean number of eggs laid on watch glasses initially without eggs. For these two contrasts 0.275 and 0.205 eggs per female were laid respectively for the Canton-S stock, and 1.755 and 1.560 eggs per female respectively for the yellow stock bases on 10 trials in each case. Thus the presence of eggs seems not to induce laying, hence the "facilitation" phenomenon may be a more likely explanation of the results.

Moree, Ray. Washington State University, Pullman, Washington. Selection for and against heterozygous lethals in some American populations of D. melanogaster.

In measuring the heterozygous effects of lethals by the use of estimates of population parameters, one of the estimates employed is the degree of allelism, conventionally calculated as the ratio of the observed number of lethal crosstests to the total number of crosstests, i.e., $I = d/c$. But since in crosstesting a number (k) of lethal-bearing chromosomes, the number of kinds of matings possible ($c = k(k-1)/2$) does not coincide with those occurring in a breeding population (k^2), it is suggested that the allelism estimate may be improved by taking $J = (2d+k)/(2c+k) = (2d+k)/k^2$; this follows from the simple relation $k^2 = 2c+k$. (In an earlier report this distinction between the symbols I and J was not made (Moree 1967)). The elimination rate by homozygosis is then taken as JQ^2 rather than IQ^2, where Q is the frequency of lethal-bearing chromosomes. By this means the experimental estimate of the frequency of lethal deaths is brought into mating correspondnce with the frequency of lethal deaths in the population sampled, and so is a better estimate of the latter parameter.

The effect of an estimate of allelism on the calculated average fitness of a heterozygote can be determined by the method devised by Crow and Temin (1964). These authors have shown that the average chromosomal effect on the total fitness of a heterozygote carrying one lethal-bearing chromosome is given by $H+F = (U-IQ^2)/Q \doteq \overline{U/Q}-\overline{IQ}$, where H is the effect on fitness, F is Wright's inbreeding coefficient, considered negligible in the following examples, and U is the average lethal mutation rate per chromosome per generation, taken as $U = .005$; +H implies a deleterious effect while -H implies a heterotic one. To date, all second chromosome data from large natural populations have yielded + values of H, U being larger, and in some cases considerably larger, than IQ^2, a relation first noted by Sturtevant a number of years ago. But when J is used instead of I, some of the estimates of H are negative, as shown below. Thus in some instances lethal and semilethal genes appear to reduce fitness while in others they appear to increase it.

	Q	k	c	d	IQ^2	JQ^2	$\overline{U/Q}-\overline{IQ}$	$\overline{U/Q}-\overline{JQ}$
Ives: Fla. '40	.670	50	1200	4	.0015	.0106	.0052	-.0084
Ives: Ohio '41	.497	48	1128	7	.0015	.0066	.0070	-.0033
Ives: Mass. '45	.458	37	666	3	.0009	.0066	.0090	-.0035
Band, Ives: Mass. '58	.359	57	1567	10	.0008	.0031	.0116	.0053
Hiraizumi, Crow: Wis.	.288	50	1182	7	.0005	.0022	.0157	.0097
Seto: Ohio '60	.374	54	1418	34	.0034	.0059	.0044	-.0024

These new estimates are by no means intended as final. Further needed adjustments for such variables as synthetic lethals, inbreeding, where relevant, and for the fact that allelism and mutation rates are based on different arrays of lethals (Crow and Temin 1964; Dobzhansky 1964) have yet to be made.

References: Band, H. T. and P. T. Ives, 1963, Canadian J. Genet. and Cytol. 5:351; Crow, J. F. and R. G. Temin, 1964, Amer. Nat. 98:21; Dobzhansky, Th., 1964, Amer. Nat. 98: 151; Hiraizumi, Y. and Crow, J. F., 1960, Genetics 45:1071; Ives, P. T., 1945, Genetics 30:167; Ives, P. T., 1954, P.N.A.S., US 40:87; Moree, R., 1967, 56:578; Seto, F., 1961, Amer. Nat. 95:365. (Aided by funds from the State of Washington Initiative Measure No. 171 for the Support of Biological and Medical Research.)

Charlesworth, B. and R. W. Davies. Cambridge University, England. Secondary non-disjunction in FM6 stocks.

We have evidence that our stocks of FM6 contain XXY females in appreciable frequency. For example, 5 out of 17 single-pair matings of FM6 Ubx^{130} females with Muller-5 males gave exceptional males, at an overall rate of 45%. Exceptional males are fertile, and non-disjunctional cultures give regular and exceptional females capable of transmitting the non-disjunction. The frequency of XXY females appears to vary between sub-lines, and may have been much higher when the FM6 stock was originally obtained from Pasadena 2 years ago. (J.R.S. Whittle, personal communication). It would be advisable for other workers to check their stocks, as erroneous results may occur in using this balancer in chromosome substitutions with unmarked stocks.

Hinton, Claude W. University of Georgia, Athens, Georgia. Meiosis in Drosophila ananassae males.

Reports of crossing over in D. ananassae males have stimulated observations on their meiosis. Most of the material was obtained from a structurally homozygous stock by saline dissection, acetic alcohol fixation, very light acetic orcein staining, polyvinyl pyrolidone mounting and phase contrast observation. Histological organization of the testis is similar to that of D. melanogaster; cell counts in intact cysts as well as cell size aided identification of spermatogenic stages. The large nuclei of early primary spermatocytes contain a prominent nucleolus which gradually diminishes in size as prophase progresses. Chromosomes are first visible as fine dispersed strands not readily equated with standard descriptions of leptonema-zygonema; heterochromatic blocks, probably representing portions of the Y and fourth chromosomes, are intimately associated with the nucleolar surface at this time. The two large autosomes develop into pachytene bivalents (like those seen in Tipula caesia by Bauer and Beerman) which are at least five times their gonial metaphase length. These bivalents then progress, by contraction, through diplonema, diakinesis and metaphase; in many cases it is clear that the arms are not associated through chiasmata and in no case has positive identification of chiasmata been made. Whether or not these cytological features are causally related to genetically detected crossovers remains to be seen. The X, Y and fourth chromosomes are usually observed as a tangled multivalent; this association is not maintained through the nucleolus but rather it appears in occasional figures to be based on pairing at restricted points. The remainder of the meiotic sequence in males of D. ananassae does not differ significantly from that of D. melanogaster as described by Cooper.
(Supported by PHS Research Grant HD01235.)

King, R. C. Northwestern University, Evanston, Ill. Wide spread occurrence of a symbiont in Drosophila.

Cytoplasmic symbionts (A bodies) which presumably are Rickettsias or Mycoplasmas were first observed in our laboratory in electron micrographs of adult ovarian tissues from D. melanogaster females of a variety of genotypes. Photographs of such symbionts may be seen in Growth 22: 323, 26: 241, 28: 320, and J. Morph. 119: 291 and 121: 63. We have subsequently seen the organisms in a variety of other tissues in adult and pre-adult Drosophila. In fact we have never failed to encounter them in any tissue subjected to a thorough electron microscopic investigation. It seems worth-while to provide the following catalogue to document the wide spread nature of the phenomenon. A bodies have been seen in Oregon S, wild type, adult females in the cytoplasm of oogonia, cystocytes, pro-oocytes, pro-nurse cells, oocytes at both pre-yolk and vitellogenic stages, in the ovarian follicle cells, the cells of the esophageal epithelium, in the cells making up the aorta, in pericardial cells, corpus allatum cells, in the cortical cells of the corpus cardiacum, and in hemocytes adhering to the corpus cardiacum. A bodies are seen in the axoplasm of the axons making up the efferent nerves which pass through the corpus cardiacum. Since such axons have their cell bodies in the protocerebrum, the symbionts probably reside in the brain as well. A bodies are also present in the sheath cells covering these nerves. In Oregon S, second and third instar larvae, prepupae and pupae, A bodies occur in cells of the prothoracic gland. In Oregon S pupal females, they are found in nurse cells, oocytes, cystocytes and in the amoeboid cells which populate the extraovariolar and ovariolar cavities. The symbionts have been seen in adult ovarian tissues from females of genotype y g^2 ty (from stock y g^2 ty/ClB), fu (from fu/ClB), su^2 Hw sbd^2 (from su^2 Hw sbd^2/TM1), and from the Chicago + strain. They occur in the corpus allatum of ap^4 adult females (from ap^4/SM5), and in the corpus allatum and prothoracic glands from e larvae and lgl cn bw larvae (from lgl cn bw/SM5). Finally, A bodies are not restricted to D. melanogaster, since they have been observed in the ovaries of adult females from the Barbados 3 strain of Drosophila willistoni. It is is obvious that if "cytoplasmic DNA" is reported for Drosophila, both A bodies and mitochondria should be considered as sources.

Colainne, James J. and A. E. Bell.
Purdue University, Lafayette, Indiana.
The effect of the doublesex (dsx) mutant on the action of daughterless (da) in D. melanogaster.

The gene "daughterless" (da), (Bell, Genetics 39: 958-959, 1954), is a second chromosome, recessive mutant in D. melanogaster, which in the homozygous state in the female parent results in 100% male progeny regardless of the genotype of the male parent. Primary sex ratio appears to be normal, but female progeny die before hatching. The gene "doublesex" (dsx), (Hildreth, Genetics 51: 659-678, 1965), is a third chromosome, recessive mutant in D. melanogaster which in the homozygous condition causes both XX and XY individuals to develop as intersexes. Its time of action seems to be prior to the time of anatomical sexual differentiation.

A genetic study was carried out to determine if the action of the "doublesex" gene would affect the lethality of the female progeny from the da/da female parent. The results of 15 matings are given below. A standard marked X - chromosome system was used to distinguish XY dsx/dsx progeny from XX dsx/dsx progeny.

Mating	Progeny: Male +	Female B/+	Intersex +	Intersex B/+
Bw^a/Y, da/da, Ubx/dsx X +/+, da/da, Ubx/dsx	1113	0	469	0

The lack of female progeny, as well as the lack of the B/+ intersex type, indicates that "doublesex" does not affect the expression of the "daughterless" trait. This observed non-interaction could be interpreted in two different ways. Either the effect of the "daughterless" gene occurs prior to that of "doublesex", and thus lethality of female progeny occurs before "doublesex" has had a chance to operate, or the expression of "daughterless", regardless of its time of action, is independent of the sexual anatomy, and thus independent of the action of "doublesex". Although it is not possible to distinguish between these two hypotheses at the present time, further investigation along this line may give some clue to the mode of action of "daughterless" and to the time of action of "doublesex".

Muñoz, Enzo R. and Beatriz Mazar Barnett.
Atomic Energy Commission, Buenos Aires, Argentina.
Lack of mutagenic effect of Gamma irradiated fructose solution.

An attempt was made to establish the possible mutagenic action of the radiolytic products of a Gamma irradiated (2.5 Mrads) fructose solution. For this purpose a 10% freshly irradiated solution was injected into Oregon R males that were subsequently mated to "Basc" females, in two different experiments. Adequate experimental procedures were followed to ensure that spermatogonia were tested in one case and mature sperm in the other. Standard sex-linked recessive lethal tests were done with the F_1 females.

The results, seen in the table, fail to show either a significant increase in mutation frequency over the untreated controls or significant differences between the two stages tested.

	No. chrom. tested	No. lethals	% lethals
Experiment No. 1 (Spermatogonia)			
Treated	6250	16	0.25
Control	6764	7	0.10
			P > 0.05
Experiment No. 2 (Mature sperm)			
Treated	1829	6	0.32
Control	2439	2	0.08
			P > 0.05

Parker, D. R. University of California, Riverside, California and Oak Ridge National Laboratory, Oak Ridge, Tennessee. On the sequence of elements in compound-XY # 129-16 and in some of its derivative marked Y chromosomes.

A number of detachments resulting from induced exchange between an attached X and the y^+Y were found to be attached-XY, and were considered to be of the sequence $XY^S \cdot Y^L y^+$ if the marker were retained (Parker and McCrone, 1958). One stock, # 129-16, that has been used in making some doubly-marked Y's has been found instead to be of the sequence $Xy^+Y^L \cdot Y^S$, with the break in the y^+Y distal to ac^+ and y^+, but proximal to $1(1)J1^+$. This stock differs from the other compound XY detachments that retain y^+ in having some yellow variegation in combination with yellow or $yellow^2$ in the attached-XY/O. Variegation in the presence of $yellow^2$ is most easily seen as pale spots in the darker sclerites at the tip of the male abdomen.

One derived Y (Y-66d, $Y^S y^+ Y^L \cdot Y^S$) arose spontaneously in a 129-16/O male and was recovered as a single y v bb/Y-66d female. Males of the constitution y sc/Y-66d are fertile and show a more pronounced yellow variegation than do y sc 129-16/O males. Variegation in this case and in the case of the original 129-16 stock suggested a different relationship of the yellow locus in these stocks. Furthermore, were y^+ in a terminal rather than in an interstitial position, the derived Y, having two doses of the y^+ duplication should show the pronounced Hw-like phenotype of extra hairs in the Second Posterior Cell of the wing, branched Posterior Crossveins, etc., that is regularly found in iso-marked y^+Yy^+ chromosomes (Williamson).

A test of the position of the y^+ marker was to reconstitute an attached X from 129-16. Were the marker terminal, it should almost surely be lost in forming a new attached X; if interstitial, it should be retained. One such attached X arose spontaneously in the 129-16/O/y v bb stock; it retained the marker. The interstitial position was further confirmed by finding that the y^+ duplication in 129-16 and in Y-66d did not cover $1(1)J1$ either with or without an extra Y.

Williamson has obtained a number of spontaneous attached X detachments from a doubly-marked Y derived from 129-16, where the detachments carried either both of the markers (y^+ and B^S) or neither, showing this derivative to be $B^S y^+ Y$ rather than $y^+ Y B^S$ as formerly believed.

The interstitial position of y^+ in 129-16 suggested that Y-66d could carry two doses of the Y^S male-fertility complex (KS) as well as one of the Y^L complex (KL), hence should be of the structure, KS y^+ KL·KS. This was verified by testing a series of radiation-induced detachments, using ability to complement FR-2 as a test for the presence of a complete KS. Of 8 detachments that carried y^+, and therefore should in any simple exchange also carry one or the other of the KS complexes, 7 were found to give fertile males in combination with FR-2. The eighth case proved to be a complex one, having all of the markers of 4R as well as y^+ and kl-5 linked with the X, requiring a total of 4 breaks to form the detachment. (ORNL is operated by Union Carbide Corporation for the U.S.A.E.C.)

Mittler, Sidney and Donald D. Grube. Northern Illinois University, DeKalb, and Argonne National Laboratory, Argonne, Illinois. Beta rays and chromosome loss.

Beta rays from Y^{90} source were compared to X-rays (150 Kv 15 ma, 150 r/min) with respect to induced chromosome loss by the XO method, and dominant lethals in spermatogenesis of X^{C2} yB/Y^+ [illegible]^8. The adult males 1 to 4 hours old were positioned inside a lusteroid cylinder so that they only received beta rays 455 r/hr (mean of 0.91 MeV) from 360°. After 1600 r the males were mated daily for 12 days to ywf at ratio of 1 male to three females. The mature spermatozoa were more sensitive to beta rays than X-rays by the XO method; and in the dominant lethals, the induced sterile period as represented by brood day 8 were intensified somewhat by beta rays. There was no difference in genetic effects between beta rays and X-rays in the broods which represent spermatogonia at the time of irradiation.

Williamson, John H. Biology Division, ORNL and University of California, Riverside, California. Identification of Y fragments with two doses of y^+ or B^S.

Parker (1965) has suggested that non-reciprocal exchanges between the arms of the Y chromosome may account for a large proportion of Y fragments induced in irradiated females. When the irradiated Y chromosome is the B^SYy^+ non-reciprocal exchanges between the two arms would produce fragments with two doses of y^+ or two doses of B^S. Flies carrying two sc^8Y's have extra hairs in the second posterior wing cell (Schultz, via Lindsley). This suggested to Parker (personal communication) that Y fragments with two doses of y^+ may cause extra hairs in the wings of flies carrying the fragment. Of 190 y^+ fragments recovered in males, 67 (35.3%) carried 4R and 81 (42.6%) caused extra hairs in the wings of flies carrying the fragments. Not only did the wings possess extra hairs but there were also extra vein cells in the wings, especially associated with the posterior cross vein. These abnormalities are probably due to variegation of the Hw^+ locus in the sc^8 duplication, two doses of which are necessary for expression. In a sample of 56 B^S fragments ments, 29 (51.8%) produced smaller eyes than did one dose of B^S.

The real proof of isomarked Y fragments is to make detachments of attached-X's using these fragments and to analyze all of the detachments for y^+ or B^S and 4R. This has been done and over 90% of the detachments made with presumptive isomarked fragments have been shown to be X-Y or X-4R detachments, while less than 70% of the detachments made with $?Yy^+$ or $B^SY?$ fragments were so identified. Thus approximately 80% of the y^+ fragments and 50% of the B^S fragments have been identified, allowing inferences as to the types of induced exchanges producing Y fragments. (ORNL is operated by Union Carbide Corporation for the U.S.A.E.C.) Parker, D. R. 1965. Mut. Res. 523-529.

Frye, Sara H. P.O. Box 267, Irvine, Kentucky. Pilot experiments involving X-ray induced mutant phenotypes (dumpy and yellow) in Scute-19i chromosomes of mature sperm in Drosophila m.

Young males, heterozygous for scute-19i second chromosomes, were X-rayed at 2-kr (200 r/minute) and immediately mated to virgin-females containing mutant markers, yellow (y); echnoid (ec), dumpy (dp), and clot (cl), i.e., Y / y; sc^{19i}/S InCyL,Cy 2-kr ♂♂'s X y/y; ed dp cl/ed dp cl vv ♀♀'s. Four successive post-treatment 24 hr. broods (i.e., 4 groups of y; ed dp cl vv ♀♀'s) and several transfers of the inseminated females from each brood were made to obtain as many F_1 progeny as possible.

The expected F_1 phenotypes of the above mating were (+) and (y; S Cy), however, only the F_1 (+) progeny were counted and examined for the recovery of exceptional mutant phenotypes. Thirty-four mutant phenotypes (31 dp's; 3 y's) were recovered from approximately 26,000 F_1 + ♂♂'s and + ♀♀'s (representing 2-kr treated scute-19i chromosomes and three mutant phenotypes (3 dp's; 0 y's) were recovered from small scale controls ran six months prior to the treated series. One yellow mutant was progeny-tested and found to be transmissible, but it was not tested for achaete. Another yellow mutant was likely to have been a chromosomal deficiency, because it was observed to be phenotypically clot with a Minute bristle effect.

The proximity of the dumpy and yellow regions in scute-19i chromosomes (see Frye, DIS 42:80) and the absence of chromocentral heterochromatin (CH) made scute-19i chromosomes appear to be favorable for the induction of mutant phenotypes in order to provide an answer to the problem as to whether two or more closely linked markers are ever involved in an X-ray induced yellow mutant phenotype where CH is not adjacent to the y^+ ac^+ region.

The relative frequency of yellow mutants induced by 2-kr in scute-19i chromosomes is less than the frequency of either dp's 2-kr induced in scute-19i chromosomes or y's induced in scute-8 chromosomes (where CH is adjacent to the y^+ ac^+ region) via 2-kr. 38 y^- sc^8 B ♀♀'s were recovered from 19,108 y^+ sc^8 B/y w In49 f ♀♀'s (author's 1964 Texas data). Yellow mutant phenotypes X-ray induced at a variety of X-ray doses often involve closely linked markers (Frye, 1958, 1959, and 1961) in scute-8 chromosomes of different germ cells.

The scute-19i experiment reported here did not solve the problem.

Work supported by NSF, estate of the author's deceased father, and USPHS.

Valencia, Ruby M. Atomic Energy Commission, Buenos Aires, Argentina.
Translocations between SM1 and Ubx^{130}.

For an experiment which was being planned, it was convenient to have the two autosomal balancer chromosomes SM1 and Ubx^{130} linked together. An irradiation was therefore carried out for the purpose of inducing translocations between them. Males of the genotype SM1/B1; Ubx^{130}/Vno were irradiated with 4000r of X-rays and mated to normal females. F_1 Cy Ubx females or males were tested for linkage. 184 fertile cultures yielded 14 translocations, all of which were confirmed by 2 different outcrossings. Most of the translocations are viable (though detrimental) and fertile in heterozygous condition in males and females, but 1 is male sterile, 1 is male lethal and 2 were so inviable in both sexes that the stocks could not be maintained. It has not been possible to determine salivary chromosome break points. All of these stocks will be available for a limited time to anyone who requests them, but eventually all will be discarded except the one chosen for our purpose.

Johnsen, R. C. Brown University, Providence, Rhode Island.
An $X \cdot Y^{S}Y^{L}$ Chromosome.

A new attached XY chromosome has been synthesized which carries a normally arranged X chromosome on one side of the centromere and a complete Y chromosome on the other. This chromosome was derived in the following way. Prepupal males carrying an $X \cdot Y^{S}$ chromosome marked with y and w and FR-2, $y+Y^{L}$. were irradiated with 1000r. Upon eclosion the w males were mated and daily transferred to 3 new y w^{a} bb $\hat{X}\hat{X}$ virgin females without a free Y for 4 broods.

An exchange distal to the fertility factors on Y^{S} and proximal to those on Y^{L} generates a y w $\cdot$ $Y^{S}Y^{L}y^{+}$ chromosome which will be recoverable as a fertile w male. Should such an exchange delete one or more of the fertility factors on Y^{S} or Y^{L} the resulting w male will be sterile. Fertile w males may also arise through nondisjunction.

Of 26 w males recovered, three were fertile and two of these proved to be cases of primary nondisjunction. The remaining fertile male gave only y w^{a} bb females and w males in the subsequent generation. Cytological examination of a few Metaphase II spermatocytes confirms the expectation that the attached XY is a metacentric-like chromosome.

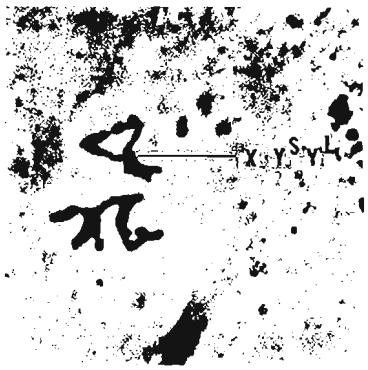
A comparative crossover test was carried out by mating the $X \cdot Y^{S}Y^{L}$ and Ore-R males to females of a stock bearing the markers y(o,o), cv(13.7), ct(20.0), oc(23.1), v(33.0), f(56.7), and car(62.5). The F_1 virgin females of each mating were then back-crossed to males of the marked stock. The crossover values for each region of the $X \cdot Y^{S}Y^{L}$ and Ore-R chromosomes are presented in the table. Crossing over between car and the centromere of the $X \cdot Y^{S}Y^{L}$ was measurable because of the presence of y^{+} on Y^{L}. There appears to be a significant decrease in crossingover beginning at v and progressively increasing toward the centromere. This may be caused by interference of Y heterochromatin on crossingover at the proximal end of $X \cdot Y^{S}Y^{L}$.

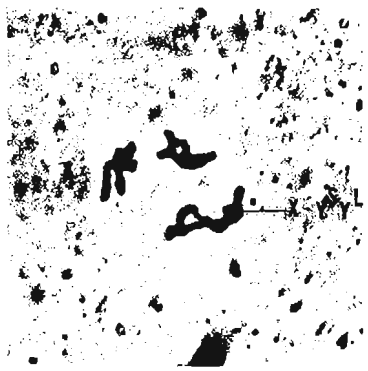
Observe Differences Between Markers

	R_1 y-cv	R_2 cv-ct	R_3 ct-oc	R_4 oc-v	R_5 v-f	R_6 f-car	R_7 car-spa	N
$X.Y^{S}Y^{L}$	11.7	7.8	3.1	9.4	16.6	2.1	1.6	3390
Ore-R	9.6	6.0	3.1	9.1	22.3	4.7	(--)	2784
Bridges & Brehme	13.7	6.3	3.1	9.9	23.7	5.8	3.5	

The chromosome has been carried with $\hat{X}$X/o females for ten generations with no observed breakdown of the newly derived chromosome. However, the frequency of w males was observed to be somewhat lower than expected, and so w males were individually mated with virgin free X y females and y w^{a} bb $\hat{X}$X/o females to test for a semilethal effect. The results of the free X cross were 481 + ♀ : 1618 y♂, and the frequency of $X \cdot Y^{S}Y^{L}$ (481/N) is only 22.9% instead of the expected 50%. In the attached X cross, there were 828♀: 395♂ or a 32.3% recovery of the attached XY.

The results of the free X cross cannot be interpreted in terms of a semilethal effect of the attached XY. Instead, these preliminary results would appear to be compatible with an interpretation of meiotic drive even though a detailed cytological analysis and tests of attached XY females heterozygous for a suitable inversion have not been carried out.





Nash, David, and Effie Woloshyn. University of Alberta, Edmonton, Alberta, Canada.

Instability of Enhancer of Hairless.

Enhancer of Hairless (E-H, II, 50.3) arose spontaneously in a population in which expression of Hairless (H, III, 69.5) was under observation. Its effects and location close to Suppressor of Hairless (Su-H, II, 50.4) were described by Nash (1965). E-H was made homozygous in a background selected for suppression of H effect on head bristles. This background contained a balanced lethal system, H/LVM. The E-H/E-H;H/LVM stock was maintained in mass culture.

On at least three occasions in two years, flies with phenotypes characteristic of E-H/+ and +/+ genotypes appeared in the stock. After the second and third breakdowns the stock was re-established from single-pair cultures with both parents E-H/E-H, judged by the progeny. After the third breakdown the stock segregated a pair of alleles with the same phenotypic effects as E-H and its wild-type counterpart. These alleles segregate on the second chromosome and, although their map position has not been determined, we assume segregation is at the locus of E-H and that the original wild-type locus or the equivalent has somehow been reintroduced to the stock.

We have tested the possibility that the phenomenon results from reversion within the E-H/E-H stock by growing a number of lines and sampling each generation. Starting from twenty homozygous cultures we have observed three reversions in about one hundred twenty generations. Details of their appearance in the lines are shown in table 1.

Table 1

Average numbers of bristles missing and the scores of individual flies in four cultures which originated from E-H/E-H; H/LVM populations.

Generation	A		B		C		D	
1*	10.6	10,11,12,12,12	9.1	9,9,10,11,11	10.3	10,10,11,12,12	11.2	11,11,12,12,12
		9,9,10,10,11		7,7,10,10,11		9,9,10,10,10		10,10,11,12,12
2	11.3	10,11,11,12,12	6.6	8,9,9,10,11	10.6	10,11,11,11,12	10.8	10,11,11,11,12
		11,11,11,12,12		1,1,2,7,8		9,10,10,11,11		10,10,11,11,11
3	11.2	11,11,12,12,12	6.4	2,4,6,9,11	10.4	8,10,11,12,12	8.5	3,10,10,11,12
		9,10,11,12,12		3,5,7,8,9		7,10,10,12,12		2,2,11,12,12
4	10.7	10,10,12,12,12	7.7	3,8,8,9,12	9.3	7,8,11,12,12	8.2	9,9,9,11,12
		9,10,10,11,11		1,8,8,9,11		7,8,9,10,10		2,3,7,9,11
5	11.1	11,11,12,12,12	5.9	0,4,8,8,9	9.2	9,9,9,11,12	7.9	7,9,10,10,12
		10,10,10,11,12		2,4,8,8,8		2,9,9,11,11		2,2,8,9,10
6	11.1	10,11,12,12,12	5.7	2,3,4,6,10	7.7	7,9,9,10,12	5.6	4,5,9,11,11
		10,11,11,11,11		2,5,7,9,9		2,2,6,9,11		0,2,3,4,7
7	10.6	11,11,11,12,12	8.0	5,8,9,11,12	7.9	3,6,8,8,12	7.2	3,9,10,10,12
		**7,9,10,11,12		0,7,8,9,11		6,7,8,9,12		1,3,4,10,10

A is a line in which no reversion has spread in the population. B,C, and D show the three instances of reversion. 5 flies of each sex were scored: The average for all ten flies is shown, followed by scores for females. Males are shown below females.

Prior to generation 1 the cultures had been maintained for two unscored generations. The samples of flies shown were not included in the parents of the next generation.
*Larger samples of flies were checked in this generation. Only B showed possible revertants.
**This fly may have been a single revertant. However, the line has remained stable for several further generations.

The number of bristles missing from the dorsal surface of the head has been used to indicate genotype, since some classes of phenotype are typical of particular genotypes (see Table 2).

Table 2

Cross	Number	Number of head bristles missing												
		0	1	2	3	4	5	6	7	8	9	10	11	12
+/+ x +/+	48	3	2	19	15	8	1							
E-H/E-H x +/+	53							2	12	21	13	5		
E-H/E-H x E-H/E-H	46										2	19	19	6
E-H/+ x E-H/+	48	1	1	8	3	2	2	3	5	11	2	3	4	3

Nos. of flies falling into each class

All crosses carry Hairless balanced against LVM.

Two aspects of the finding require explanation: (a) What causes reversion? (b) Why do the revertants spread relatively rapidly in the cultures?

We prefer to explain the reversions as the result of unequal crossing-over, since their frequency is higher than the expected rate of mutation. This supposes that E-H is a duplication, but we have not been able to demonstrate this cytologically.

If this explanation is correct, then interpretation of recombination data between E-H and Su-H (Nash, 1965) needs revision, because wild type progeny from the cross E-H/Su-H; H/LVM ♀ X +/+; H/LVM ♂, supposed to be recombinants between two point mutations, could have arisen by events which remove the duplication or incorporate the Su-H allele into one of its duplicate parts.

We suggest that revertants spread through the population because of their superior fitness. E-H/E-H; H/LVM flies are less vigorous than +/+; H/LVM flies with otherwise comparable genetic backgrounds. The disadvantage of E-H probably depends on the presence of the H mutant, since E-H/E-H; $+^H/+^H$ mass cultures have been maintained apparently without revertants arising.

References:

Nash, David. 1965 The expression of 'Hairless' in Drosophila and the role of two closely linked modifiers of opposite effect. Genet. Res., Camb. 6: 175-189.

Mazar Barnett, Beatriz. Argentine Atomic Energy Commission, Buenos Aires, Argentina.

Preliminary genetic tests with dimethyl sulphoxide.

Since dimethyl sulphoxide has been shown to act as a whole body protector against ionizing radiation (Ashwood-Smith, Int. J. Rad. Biol. 3, 1961, and Moos and Kim, Experientia 22, 1966), preliminary tests were carried out to investigate if this action could be extended to a chromosomal level as well.

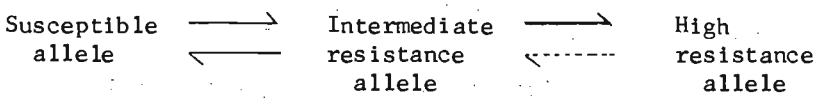
Males of the Oregon R stock were injected with a 10% solution of the compound prior to X-irradiation with 800r at different intervals. At 24 hours after irradiation they were mass mated to "Basc" females, left for one day and then discarded. The females were allowed to oviposit for two additional days. Standard sex-linked recessive lethal tests were done with the F_1 females.

These preliminary results, as can be seen in the table, seem to indicate a certain effect. There is a drop in the frequencies of mutations, as compared with the irradiated controls, which is more pronounced as the intervals before irradiation increase.

	No. tested chrom.	No. lethals	% lethals
800r	2075	40	1.9
DMSO (30 min. int.)	1184	19	1.6
DMSO (5 hrs. int.)	1129	13	1.1
DMSO (20 hrs. int.)	2469	19	0.7

Kikkawa, H. Osaka University, Osaka, Japan. Induction of a parathion-susceptible gene from its resistance allele in D. melanogaster.

As reported in DIS 34: 89, and Botyu-Kagaku (Scientific Pest Control) 29: 37-42, 1964, a parathion-resistance gene (2-64.5) could be induced from its susceptible allele with the treatment of X-ray. In this case, the mutation took place via an unstable intermediate allele as shown in the following process:



However, the step indicated by a dotted line, i.e., the step from a high resistance allele to an intermediate one has not yet been examined. Namely, the question remains whether a high resistance gene mutates to a susceptible one via an intermediate allele, or mutates directly to a susceptible one in one step.

In order to examine this point, males of the Hikone strain which showed the highest resistance to parathion in our laboratory were treated with X-ray (2000 R), and mated to susceptible females carrying two visible markers, cn and bw. The F1 instar larvae were reared in a medium containing 3 mM phenylthiourea (PTU). As shown in DIS 34: 89, flies having a high parathion-resistance gene in homozygous or heterozygous state are unable to survive in such a medium because of the negatively correlating effect between an insecticide like parathion and PTU. However, if the mutation from a high resistance gene involved in the Hikone chromosome to a susceptible allele had been induced by the X-ray treatment, such a fly would survive even in the medium containing PTU.

In this way, we could obtain 7 adult flies from 5510 larvae tested. Two substrains derived from those 7 survivors showed intermediate resistance to parathion. Of interest is the fact that the intermediate resistance gene involved in these substrains is also unstable and often mutates to a complete susceptible allele spontaneously. The reason why the mutation from a high resistance gene to a susceptible one and vice versa takes place via an intermediate one is now being investigated.

Masterson, J. Iowa State University, Ames, Iowa. A method for procuring certain X-Y duplications.

A breeding method was devised in order to facilitate the recovery of duplications of the y-sc region on the Y-chromosome. It was thought that, if a "double lethal female" could be obtained which had in at least one of its X-chromosomes a deficiency for this region, this would make it easier to select for duplications on the Y. The following breeding program was therefore set up:

1,v,car/Muller-5 X y^{3p},In S,sc^{S1}; sc^{19i}/Cy

1,v,car/y^{3p},In S,sc^{S1} ; sc^{19i}/Cy X X•Y(y,v,f)/Y (irradiated 4,000 r)

Score for y,sc+ males

These males were tested against various markers on the distal portion of the X-chromosome and their Y was found to carry the wild-type alleles for su-v, su-w^a(su-apr), ac, sc but not pn. This duplication was also found to be allelic to yellow. Since the irradiated X•Y chromosome carried y,v,f, it is presumed that the resulting duplication carries the mutant allele, yellow.

Utilizing the symbolism of Bridges and Brehme (1944) this X-Y duplication is designated Dp (1;Y) 1E. Another duplication using the same method was found and named Dp (1;Y) 2E. The two duplications are similar but not identical. (See descriptions under New Mutants).

Theoretically any sex-linked lethal could be covered by an insertion of a piece of the X into the Y. It has been found, however, that only areas on the X near the proximal and distal ends can be utilized. A possible explanation would be that in these areas only two breaks in the X•Y chromosome would be needed to give a 1;Y Duplication. To get a 1;Y Duplication covering loci near the vermilion locus, four breaks would be necessary.

Karlik, A. University of Vienna, Austria. Missing recombination in the X-chromosome of D. ambigua between two non-allelic mutant genes determining the eye-color.

Although many members of the obscura-group of Drosophila are genetically well investigated, very little is known about the genetics of the European D. ambigua Pom. Several mutants have been collected during the past years in our laboratory. For the sex-chromosome seven different mutants could be found, of which six have been already described earlier: w,y,or, ct, px, N (Karlik 1958). A new mutant spontaneously arisen in a laboratory strain was isolated and may be described here:

v: vermillion, recessive, sex-linked. Bright red eye color, but normal color of ocelli, testes and Malpighian tubes.

Recombination frequency was checked between v, or and y. The frequency of the different recombination types is given in the following table.

Test cross	n	recombinants
y or/++♀ x y or♂	16,233	4.57%±0.16
y v/++♀ x y v ♂	4,614	6.84%±0.37
+ v/ or + ♀ x or + ♂		0
+ v/ or + ♀ x + v ♂	11,642 (♂ only)	0

From the first two crosses it may be assumed that v and or are mutants of different closely linked genes. This assumption is confirmed by the fact that females heterozygous for + v/or + show the normal phenotype. Yet, there are no recombinants in the crosses between or and v. Since v and or were on different chromosomes in the heterozygous females, only ++ recombinants were looked for among the male offspring. The phenotype of the combination or-v is unknown but no new eye color could be seen in the crosses. Among 11,642 males only v and or males could be observed.

A number of salivary gland preparations were made of female larvae heterozygous + v/or + and the giant chromosomes analysed. Although several autosomal inversions could be observed, none was present in the X-chromosome. There were no striking other abnormalities in the giant chromosomes. So the assumption that the strong linkage may be due to a chromosomal aberration can be excluded.

Karlik, A.: Vererbungsversuche mit D. ambigua Pom. Z.Vererb.-Lehre,89:448-458 (1958).

Kaneko, A., E. Momma, T. Tokumitsu and T. Shima. Hokkaido University, Sapporo, Japan. Frequencies of abundance of the quinaria species group in Hokkaido and Aomori Prefecture.

During the ten years, drosophilid flies have been collected actively from many localities in Hokkaido, and in Aomori Prefecture, northern part of Honshu. Quinaria species is one of the common Drosophila in Japan. Total flies and percentage frequency of each quinaria member in Hokkaido (samples are from 33 localities) and Aomori Prefecture (from 3 localities) are as follows:

	No. of flies of quinaria group	Percentage frequency nigromaculata	brachynephros	unispina	angularis	kuntzei
Hokkaido	19002	79.93	17.90	2.16	0.00	0.01
Aomori Prefecture	436	8.03	16.74	2.75	72.45	0.00

In Hokkaido D. nigromaculata was very frequent in occurrence in all localities. On the contrary, D. angularis was so frequent in Aomori, this has not yet been obtained in Hokkaido in spite of extensive collections. D. kuntzei has been recorded from all over Japan, though it is a rare species in occurrence.

Svirezhev, Yu. M. Institute of Medical Radiology, Obninsk, USSR. Establishment of heterozygotic polymorphism in non-stationary populations.

In (1) the cases of heterozygotic polymorphism in the stationary model populations of D. melanogaster are considered. Dynamics of polymorphism establishment was described by monotonous curve p(t) of gene frequency, which approaches the constant value of polymorphic level $\mathcal{E}^*$ asymptotically. But in the experiments on the polymorphism establishment in populations, subject to temporal size changes (non-stationary populations), the non-monotonous change of p(t) was observed. Naturally, that the coefficients of relative viability are the functions of time, also. The case of a diallelic gene in panmictic population, which increases in size with time (so that $N(t) \to N^*$ for $t \to \infty$), is considered. N(t) is a population size. Let $\beta \to \beta^*$, $\gamma \to \gamma^*$, $\mathcal{E} \to \mathcal{E}^*$ for $t \to \infty$. Here β and γ are the coefficients of relative viability (1) of genotypes Aa (heterozygote) and aa (mutant homozygote) accordingly. And let the sufficient conditions of heterozygotic polymorphism existence be fulfilled. Then: if $p(o) < \mathcal{E}(0)$, $\frac{d\beta}{dt}(\gamma-1) - \frac{d\gamma}{dt}(\beta-1) < 0 \quad [0 < t < \infty]$ then the curve p(t) raises over the level $\mathcal{E}^*$ and then descends, approaching $\mathcal{E}^*$ from above asymptotically; if $p(o) > \mathcal{E}(0)$, $\frac{d\beta}{dt}(\gamma-1) - \frac{d\gamma}{dt}(\beta-1) > 0, \quad [0 < t < \infty]$ then the curve p(t) descends lower than the level $\mathcal{E}^*$ and then raises, approaching $\mathcal{E}^*$ from below asymptotically.

If the population size, and consequently, the coefficients of relative viability, oscillated with the frequency ω about some stationary level, then the amplitude of oscillations of the gene frequency is inversely proportional to ω, and, for high frequency of oscillations, is very small.

Reference: 1. Yu.M.Svirezhev, N.W. Timofeeff-Ressovsky, "On the equilibrium of genotypes in model populations of D. melanogaster," Problemy kibernetiki (Russ.), 16,p.p.123-136, 1966.

Khishin, A. F. and Mokhtar M. Zawahri. Assuit University, Egypt. The effect of yeast concentration on the X-ray induced lethal mutations in D. melanogaster.

A set of experiments was carried out to study the effects of yeast concentration in the media on the X-ray induced recessive sex-linked lethal mutations in the different stages of developing male germ cells of D. melanogaster. Three different yeast percentages were used, and this was applied before, after, or before and after irradiation. Larvae were also fed on different yeast concentration before and after irradiation. The Muller-5 method was used for the determination of mutation rates.

The results given in the Tables below show that starvation of adult males before or before and after irradiation increases the genetic effects of radiation. Irradiation of starved larvae during the actively feeding stage, followed by post-irradiation starvation results in a significant reduction in the frequency of lethals.

Table 1. Recessive sex-linked lethal induced by 2532r units of X-rays in adults males of D. melanogaster fed for 3 days on different yeast concentration.

FEEDING TREATMENT									
	Pre-irradiation			Post-irradiation			Pre-and post-irrad.		
Yeast %	4	0	10	4	0	10	4	0	10
Lethals %	7.06	7.95	6.86	7.27	6.85	5.79	7.01	7.27	6.00

Table 2. Recessive sex linked lethals induced by 1176r units of X-rays in 48 hours-old D. melanogaster larvae raised on different yeast concentration.

Yeast %	4	0.5	10
Lethals %	1.59	0.97	1.82

Srám, R. and M. Ondrej. Institute of Hygiene, Institute of Experimental Botany, Prague, Czechoslovakia. Mutagenic activity of some drugs and pesticides.

Recently there was found that mutagenic properties are not limited only to alkylating agents and analogues of DNA bases. Therefore we tested the mutagenic activity of recently synthesized compounds with which man can get in touch.

We have evaluated the mutation frequency in Drosophila melanogaster using the Muller-5 test. The tested substances were injected into the abdomen of flies with a microsyringe. The substances have been applied immediately after solution in 0.4% NaCl in maximum concentrations, which were low or under the lethality or sterility threshold, in the quantity of 0.2 µl per fly. We used a system of two broods, each of them lasting 7 days.

The differences in comparing compounds with spontaneous mutation rate are not significant.

GROUP	COMPOUND	CONCENTRATION in %	BROOD 1	%	BROOD 2	%
drug	5-azanalidixic acid	0.01	679/1	0.15	104/0	
	6-azauracil riboside	0.01	475/0		675/2	0.30
	cyclohexyl sodium sulphamate	1.0	739/1	0.14	582/0	
	emetine hydrochloride	0.1	437/0		461/0	
	5-fluorouracil	0.01	697/0		347/0	
	2-hydroxy-5-fluorpyrimidine	0.01	664/3	0.45	279/0	
	4-hydroxy-5-fluorpyrimidine	0.1	548/1	0.18	628/3	0.48
	β-4-methaxybenzoylbromacrylic acid	0.01	614/0		539/1	0.19
	ypenyl = 5-di-2-chloroethylamino-methyl uracylhydrochloride	0.2	1409/0		491/0	
pesticide	acrylonitrile = vinyl cyanide	0.1	572/2	0.35	725/4	0.55
	diquat = 1,1-ethylene-2,2 dipyridine	0.02	917/1	0.11	404/0	
	methoxychlor = 1,1,1-trichloro-2, 2-methyloxyfenylethane	0.1	1115/0		363/0	
	spontaneous		41928/58	0.14		

Tupitsina, E. M. Institute of Medical Radiology, Obninsk, USSR. On the influence of homologous and heterologous inversions on the frequency of somatic crossing-over.

In order to check the hypothesis on the role of conjugation in mechanisms of somatic crossing-over, the frequency of mosaic spots was studied in D. melanogaster females of the following genotypic consitution: y +/+ sn; +/+, y +/+ sn; Cy/+, y dl-49 +/+ sn; +/+, y M-5 +/+ sn; +/+, and y M-5 +/+ sn; Cy/+. It was supposed, that the more drastically is the conjugation disturbed, the lower should be the frequency of mosaic spots. It was found, that rather a short inversion dl-49 caused a considerably small decrease in the frequency of mosaic spots (12.7 per 1000 in y dl-49 +/+ sn; +/+ females, as compared to 16.8 in y +/+ sn; +/+ females). While, in experiments with Muller-5 chromosome, in which case a considerably more serious disturbance to conjugation could be expected, a sharp drop in spot frequency was observed (1.2 in y M-5 +/+ sn; +/+ females, as compared to 16.8 in y +/+ sn; +/+ females). Heterozygotic inversion in the II chromosome increased the frequency of mosaicism (27.4 in y +/+ sn; Cy/+ females, and 16.8 in y +/+ sn; +/+ females), possibly, due to more favorable conjugation condition in the I chromosome. Be this supposition correct, then in case of the both I and II chromosome heterozygosity (y M-5 +/+ sn; Cy/+ females) the frequency of spots should be higher than in y M-5 +/+ sn; +/+ females. As a matter of fact, the frequency of mosaicism increased in this case from 1.2 to 4.7 (the difference being significant).

Lefevre, G. San Fernando Valley State College, Northridge, California. Mutant phenotypes immediately to the left of w. Recently, Gersh (Genetics 56:309-319) described a characteristic loss of bristles evidenced in w^{258-45} males covered by the variegated duplication w^{Vco}. Several head bristles are missing, including orbitals, ocellars, verticals, and postverticals; thoracic bristles as well are occasionally absent. This syndrome was ascribed to a locus in salivary chromosome band 3C1, deficiency for which is lethal and which is deleted in w^{258-45}. Cytogenetic studies in this laboratory indicate that bands 3B3 and 3B4, as well as 3C1 and 3C2, are deleted in w^{258-45}; thus the bristle effect cannot be securely attributed to 3C1, rather than to 3B3 or 4.

By use of a new deficiency resembling w^{258-45} and a new duplication resembling w^{Vco}, further information is now available on mutant phenotypes just to the left of w. The mutant $In(1)w^{-64d}$ (see Report of New Mutants) combines a deficiency from 3B3 to 3C2, inclusive, with an inversion whose right breakpoint is in the proximal heterochromatin. This deficiency can be covered by Dp w^{Vco} and expresses a bristle syndrome similar to that described above for w^{258-45}; Dp w^{Vco} males. The effect is more extreme in XO males. Both w^{-64d} and w^{258-45} are also covered by $Dp(1;3)w^{m49a7}$, a section from 3B1-3D6 inserted in the proximal heterochromatin of 3L. Again, the bristle syndrome is evidenced in covered males. $Dp(1;3)N^{264-58a}$, similar to Dp w^{m49a7}, but not including 3B1-2, fails to cover either deficiency. A new duplication, $Dp(1;4)w^{m65g}$, (see Report of New Mutants) extends to the right of w only through the rst locus (3C4), resembling in that respect Dp w^{Vco}; its extent to the left of w is uncertain, but it is quite short. Difficulty in visualizing this short duplication in the heterochromatin of the fourth chromosome has made it impossible so far to determine its exact extent cytologically. However, it produces a low frequency of covered males with both w^{-64d} and w^{258-45}. These males exhibit in exaggerated form the bristle abnormalities mentioned above. The head is virtually devoid of bristles, and the thorax is sparsely bristled. Moreover, the eyes are small and rough, the antennae somewhat abnormal with reduced aristae, and the wings are usually blistered and misshapen. Despite these abnormalities, some males are fertile. The effect is not due to the presence of the duplication, since w^{m4L}-rst^{3R} males, deficient for 3C2-3, show none of the defects when covered by Dp w^{m65g}.

Dp w^{m65g} may not, in fact, fully cover the deficient areas in w^{-64d} and w^{258-45}; the "covered" males may be truly deficient for a short region between their left breakpoints (which follow 3B2) and the left end of Dp w^{m65g}, thereby allowing the extreme phenotype of homozygous deficient to be expressed. Alternatively, Dp w^{m65g} may have its left breakpoint near 3B2 or 3, thus giving rise to extreme variegation for the area just to the right of 3B2. If the left end of w^{m65g} were at 3B1, or further to the left, it should cover w^{-64d} and w^{258-45} as well as Dp w^{m49a7} covers them. The fact that Dp $N^{264-58a}$ covers neither w^{258-45} nor w^{-64d} suggests that Dp $N^{264-58a}$ does not extend as far to the left as its description suggests, i.e., to 3B3. Alternatively, the bristle anomalies described above reside in 3B3 or 3B4 rather than in 3C1.

A final fact to take into consideration in attributing phenotypes to specific bands to the left of the 3C region is that many salivary preparations, in which the 3B region is well stretched and stained, clearly show 3 delicate bands between 3B1-2 and 3C1; Bridges' standard map shows only 2. If this is accepted, the most plausible location for the bristle anomalies is 3B3; Dp $N^{264-58a}$, then, would extend only to 3B4 (on the standard map).

Mglinetz, V.A. Institute of Medical Radiology, Obninsk, USSR. Elimination of radiation induced chromosome aberration in experimental populations of Drosophila melanogaster.

Behaviour of chromosome aberration originating in experimental numerically stable populations of D. melanogaster was studied. The aberrations were induced by (i) single 5000 r gamma-irradiation, (ii) fractionated gamma-irradiation by 1000 r monthly fractions, and (iii) chronic irradiation from P^{32} radioisotope source supplied to the food. It was found, that chromosome translocations induced by ionizing irradiation were eliminated within the first generations, while among 66 induced inversions only 7 remained in populations for 2 to 5 generations. None of the inversions became stable in the populations.

Schouten, S. C. M. and Egberta J. Tuinstra. Genetisch Instituut der Rijksuniversiteit te Utrecht. The influence of live yeast on the incorporation of ^{3}H-Uridine in the testes of Drosophila melanogaster.

Since the observation that male flies of Drosophila melanogaster under starvation conditions yield less recessive lethal mutations than well-fed males (Schouten, 1963) it was found, that the live yeast in the medium is responsible for a high mutation frequency, especially in spermatids and spermatocytes. The observation that Actinomycin D. (R. Mukherjee, 1965) depresses the induced mutation frequency especially in the same stages, has led to the conclusion that RNA synthesis is important in determining radiation-damage in spermatids and spermatocytes. In order to verify the RNA-synthesis in 2 days old males fed with and fed without yeast, the flies were injected with ^{3}H-Uridine in a 0.7% saline solution. Three hours after injection the flies were sacrificed and the testes were sampled. The radioactivity in the testes was determined with a liquid scintillation counter during 50 min.

The results of the experiments are presented in table I. It is obvious that the incorporation in the yeast-fed animals is always higher than in the yeast-less ones with a P 0.01. Thus it seems that the RNA synthesis in yeast-fed males is more intense.

Table I. Incorporation of ^{3}H-Uridine in 2 day old males fed with and fed without live yeast.

Group		Number of testes	"Counts per injected fly" (50')	Difference
A	(no yeast fed -	20	81.63	
				+ 25.27
	(yeast fed +	17	106.90	
B	(-	20	84.41	
				+ 49.76
	(+	18	134.17	
C	(-	30	36.36	
				+ 37.52
	(+	22	73.88	
D	(-	16	85.47	
				+ 12.51
	(+	20	97.98	
E	(-	21	69.74	
				+ 55.44
	(+	24	125.18	
F	(-	20	114.57	
				+ 20.60
	(+	23	135.17	
G	(-	28	89.18	
				+ 56.87
	(+	22	146.05	

Probably it is this RNA synthesis that is at least partly responsible for the higher induced mutation frequency in spermatids and spermatocytes. Further experiments to elucidate this are in progress now. The helpful discussions with and suggestions of Prof. Dr. F. H. Sobels are gratefully acknowledged. Also Prof. Dr. W. A. de Voogd van der Straaten for his help with the scintillation counter.

Lee, Taek Jun. Chungang University, Seoul, Korea. On the polymorphism of color pattern in Scaptomyza pallida in natural populations of Korea.

Three types of color patterns on mesonotum and abdominal tergites can be distinguished in Scaptomyza pallida, referred to as the dark, the light, and the intermediate types. The color patterns are less variable in the light and dark types than in the intermediate one. The intermediate types are more variable in coloration in natural populations of Korea.

Population samples have been obtained in three localities, Mt. Sokli, Mt. Sori and Mt. Sulak. The samples collected simultaneously on May 1965 from three localities show the fact that the intermediate type appear in the higher frequency than the dark and light types, with no clear geographic trends discernible. The relative frequencies, however, vary with season. The data show the percentage of the increase light type in summer. This suggests that environmental factors as well as hereditary factors affect the color pattern in natural populations. The results of these obtained data from three localities are consistent with the assumption that color patterns in S. pallida is controlled by one pair of alleles (D-d) without dominance. The dark (DD) and the light (dd) types are the homozygotes, and the intermediate (Dd) is the heterozygote. These three genotypes are always presented in accordance with the Hardy-Weinberg formula. It suggests that S. pallida exhibits a case of balanced polymorphism in color patterns in natural populations.

Table 1. Observed number of the dark, intermediate and light types in natural populations, and numbers of expected according to the Hardy-Weinberg formula.

Locality	Date	Dark (DD)	Intermediate (Dd)	Light (dd)	Gene frequencies D	d
Mt. Sokli	May'65-Obs.	33(18.86%)	110(62.86%)	32(18.28%)	0.503	0.479
	Exp.	32.99	109.89	32.72		
		X^2 = 1.092	P = 0.70			
Mt. Sokli	Jun.'65-Obs.	579(13.71%)	2750(65.10%)	895(21.19%)	0.463	0.537
	Exp.	578.71	2749.77	895.51		
		X^2 = 12.672	P < 0.01			
Mt. Sokli	Jul.'65-Obs.	105(10.60%)	507(51.16%)	379(38.24%)	0.36	20.638
	Exp.	104.97	506.94	379.08		
		X^2 = 2.344	P = 0.10			
Mt. Sori	May'65-Obs.	80(21.00%)	223(58.53%)	78(20.47%)	0.508	0.497
	Exp.	80.09	222.96	77.96		
		X^2 = 0.846	P = 0.70			
Mt. Sori	Jun.'65-Obs.	102(18.99%)	337(62.76%)	98(18.25%)	0.504	0.496
	Exp.	102.04	336.90	98.06		
		X^2 = 3.697	P = 0.05			
Mt. Sori	May'66-Obs.	376(20.27%)	1062(57.25%)	417(22.48%)	0.489	0.511
	Exp.	376.05	1061.72	417.23		
		X^2 = 34.503	P < 0.01			
Mt. Sulak	May'65-Obs.	331(23.61%)	816(58.20%)	255(18.19%)	0.527	0.473
	Exp.	331.06	815.87	255.07		
		X^2 = 62.068	P < 0.01			
Mt. Sulak	May'66-Obs.	204(19.25%)	625(58.96%)	231(21.79%)	0.487	0.513
	Exp.	204.01	624.84	231.15		
		X^2 = 11.778	P < 0.01			

Grossfield, J. Purdue University, Lafayette, Indiana. Density dependent mating in D. rufa.

Different densities of 4 to 5 day old virgin flies together (after aging) for 7 days in small vials yielded a noticeable difference in the percentage of inseminated females.

	n	%
Single Pair Matings	119	1.7
Two Pair Matings	104	56.7

Similar but less striking density effects were obtained with a Guianian-B strain of D. paulistorum. It would be interesting if such results were obtained with other of the smaller species of Drosophila. Supported in part by PHS Grant GM-11609 and NIH Grant 2T-GM-337-06.

Momma, Eizi. Hokkaido University, Sapporo, Japan. Domestic Drosophila in Hawaii.

From Jan. 19 to Feb. 11, 1967, Drosophila populations were sampled from six main islands in Hawaii. Collections were made with the use of traps containing usually fermented banana (guava fruit was used at Hana) at one hour intervals from sunrise to sunset for two days in each island excepting in Oahu (for one day) by the help of Prof. E. Elmo Hardy, Univ. of Hawaii. We were interested in distribution of immigrant species, and therefore copses near human habitations limited to the lower altitudes (below 2000 ft.) were chosen as collecting sites. Collection sites in each island were as follows: Kauai - 2 miles northward from Lihue, Oahu - Campus of the Univ. of Hawaii, Molokai - 7 miles northeastward from Kaunakakai, Lauai - 2 miles northeastward from Lanai City, Maui - 2 miles southeastward from Kakawao and 3 miles northward from Hana, Hawaii - 6 miles westward from Hilo.

A total of 67,566 flies represented by 3 genera, 15 species (except 12 specimens of unknown spp.) was obtained as given in Table 1. All specimens excepting only 3 individuals were of immigrant species. D. nasuta was the most abundant species in every island excepting in Molokai. The most predominant species in Molokai was D. simulans. D. hydei was the second dominant species in Lanai, though few or no fly of this species were found in other islands.

Table 1. Number of flies collected by trapping in Hawaii (Jan.-Feb., 1967)*

Island	Kauai	Oahu	Molokai	Lanai	Maui	Hawaii	Total
(Immigrant species)							
Chymomyza							
procnemis	-	-	-	101	1	-	102
Drosophila							
ananassae	36	48	2	-	295	3	384
busckii	1	-	3	-	1	1	6
carinata	1	-	414	1711	858	-	2984
hydei	-	1	-	7063	3	-	7067
immigrans	298	28	388	726	1460	507	3407
kikkawai	41	12	2	-	15	104	174
melanogaster	-	-	-	203	33	2	238
nasuta	13201	2402	3502	17049	2208	3974	42336
polychaeta	690	19	-	1	-	-	710
simulans	1775	458	3935	1344	1424	1237	10153
(Endemic species)							
Scaptomyza							
confusa	-	-	1	-	-	-	1
varifrons	-	-	1	-	-	-	1
palmae?	-	-	1	-	-	-	1
Drosophila							
crassifemur	-	-	-	-	2	-	2
(Unknown species)	3	-	1	-	1	7	12
Total	16026	2968	8250	28198	6301	5835	67578
No. of species collected	11	7	11	8	12	12	25?

* Identification of the flies was confirmed by Hardy's descriptions (1965, 1966).

(Supported by a grant from the Japan Society for the Promotion of Science as a part of the Japan-U.S. Cooperative Science Program.)

Lifschytz, E. and R. Falk. The Hebrew University, Jerusalem, Israel. A system for fine structure analysis of chromosome segments.

In a previous note (DIS 42:89) we presented a complementation map of recessive lethals which were induced in the segment of the X-chromosome that was covered by a w^+•Y-chromosome. It was shown that most lethals were multi-genic and that even those, for which there was no direct evidence that they were aberrations, interfered with recombination in their vicinity.

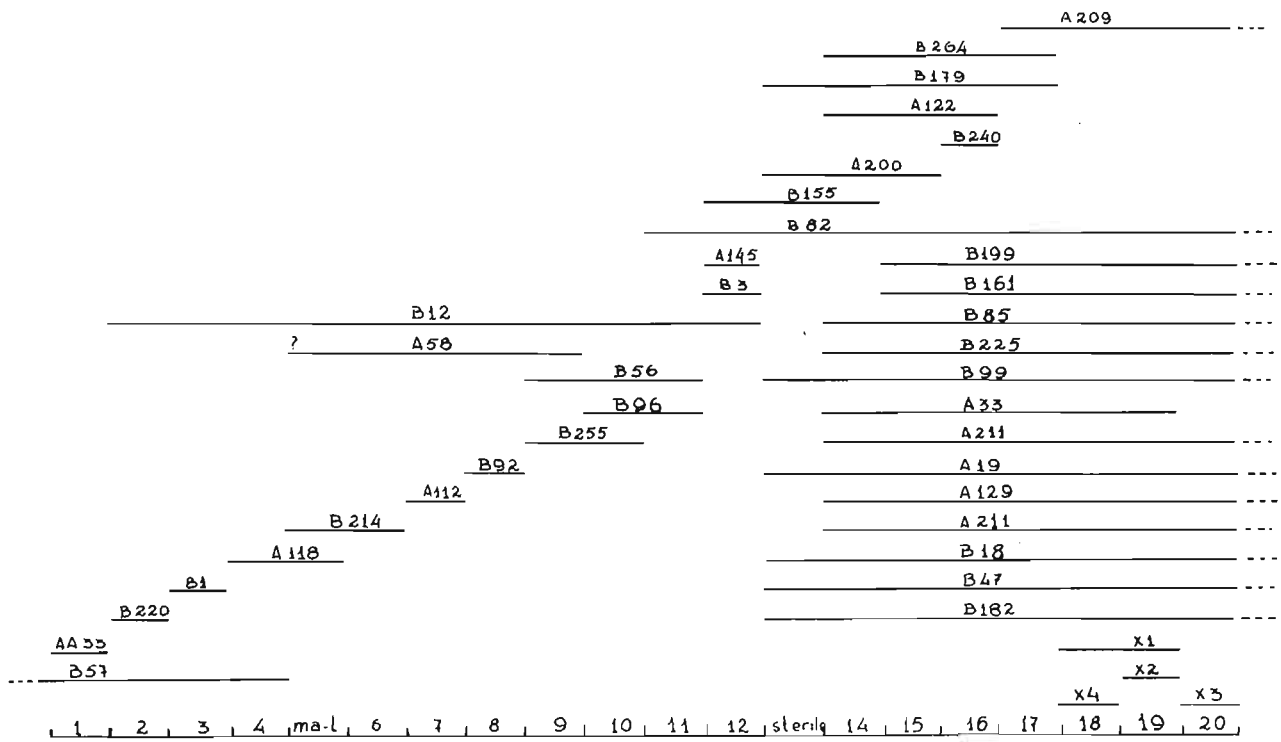
We present here a complementation map of recessive lethals which were induced in the segment of the X-chromosome covered by the ma-l^+•Y (#2) chromosome. Of the 413 lethals induced with an X-ray dose of 3200r 10% (42) were covered by the ma-l^+•Y-chromosome. 34 of these lethals have already been mapped; none was covered by the w^+•Y-chromosome, they extended thus distally to the su-f region. Included in the map were also 4 lethals of the previously mentioned series (X 1, X 2, X 3, X 4) which were covered by w^+•Y, and two lethals (81,AA33) which were induced by alkylating mutagens.

Twenty eight mutations proved to be multi-genic and for only 6 mutations there was no evidence for more than one affected gene. These results indicate to us that practically all the induced lethals were aberrations: even if a couple of the 6 "single gene" mutations will prove to be affected in more than one gene - they probably represent that end of the distribution curve of aberration-sizes which are smaller than one gene (intra genic aberrations).

As expected the map includes also bigger aberrations which extend into the heterochromatin. Some of the aberrations reach as far as the bb-region, as determined by allelism with the sc^4-sc^8 lethal effect, but even they must have at least one other lethal effect in the euchromatin. There must be at least 18 genes in the studied euchromatic segment of the X-chromosome in which lethal mutations may occur.

Some lethals were sterile in males. Even if we exclude those sterile lethals that extend into the heterochromatin (in which the sterile effect could be also in the heterochromatin) it appears as if sterile lethals A200, B155 and B179 all encompass a specific locus (No. 13 in the map) that determines fertility. It is not sure whether flies deficient for the fertility locus alone would be viable. On the other hand it seems that a deficiency of ma-l is not lethal: females heterozygous for lethals A118 and B214 are viable and have an eye-color phenotype akin if not identical to that of ma-l.

With the aid of the more extensive lethal aberrations in the ma-l region we started a modified system for the detection of induced lethals, in which it is possible to recover in F_2 both lethals outside the ma-l region (detected by the absence of one class of males) and lethals within (part of) the ma-l region (detected by the absence of one of the female classes). This technique is now used for studies on the effects of mutagens on the induction of "point" mutations and aberrations as well as for fine structure analysis of a preselected segment of the X-chromosome.



Chovnick, A. University of Connecticut, Storrs, Connecticut. Generation of a series of Y chromosomes carrying the v^+ region of the X.

Following the method described by Brosseau et al (Genetics 46:339), and utilizing T (1;B^S Y^L) 9F #124, a series of v^+ marked Y chromosomes has been produced. X-rayed males of the genetic constitution B^S Y^{LD}, 1(9F-20D) $Y^S \cdot Y^L$ y^+;y, 1(1A-9F)$Y^{LP} \cdot Y^S$ were crossed to C(1)RM, y v bb (no free Y) females, and three (3) y^+ v^+ bb^+ B^S females were recovered. These carried new marked Y chromosomes of the constitution B^S Y^{LD} v^+ $Y^S \cdot Y^L$ y^+. Although indistinguishable from tests conducted, they are maintained in stocks as B^S v^+ Y y^+ #1, 2, and 3.

By X-raying the B^S v^+ Y y^+ #1, two derivatives without the B^S marker have been produced, and these are maintained as v^+ Y y^+ #1 and 2.

All of the above Y chromosomes show the following characteristics: (1) In males, find a low frequency of intersexes, which appears to vary with culture conditions; (2) Lethal with Muller-5 in males; (3) All cover several lethal markers adjacent to, and on both sides of v, but do not cover ras or m.

Seegmiller, R. E. and G. D. Hanks. University of Utah, Salt Lake City, Utah. Mating success of vestigial males.

Two causes for lowering the mating success of vestigial (vg) males have been considered. Experiments using clipped-winged males have placed emphasis on the importance of wings in facilitating courting and successful mating. It is therefore quite possible that the important element in reducing the mating success of vg males is their lack of functional wings. Another possible cause for lowering the mating success of vg males could be that their motivation or vigor is decreased.

In order to test the vg males' motivation or vigor, an experiment was performed using females that were made antennaless by removing their antennae with forceps. Since these females could not receive the courting stimulus of wing fluttering from wild-type (+) males (supposedly received via antennae), they would not discriminate against males with vestigial wings, i.e., males lacking the element of wing fluttering. The difference if any, in the two types of male success (+ vs. vg) with antennaless females would be due only to male vigor or motivation.

The experiment consisted of 25 pair matings of each type: (1) antennaless female X + male, and (2) antennaless female X vg male. As a control 25 pair matings of each of the following were used: (1) normal female X + male, and normal female X vg male. (Both normal and antennaless females were heterozygous.) All flies were etherized more than 24 hours before the experiment began. As soon as the males were admitted to the vials containing the females, they were observed and the number of copulations were recorded during a two-hour period. Those that did not mate were transferred to new vials after 24 hours, and the males were killed after 48 hours so that if mating did occur after the two-hour observation period, it could be detected by the progeny in the first or second vial, depending upon whether the mating took place before or after 24 hours.

Figure 1 depicts the cumulative percentage mating success over a 48-hour period with particular reference to + and vg males mated to either antennaless or normal females. It can be noted that when normal females were used, the + males far exceeded the vg males in successful copulations. For + males the majority of copulations occurred within two hours, whereas vg males were slow throughout the course of the experiment. As a comparison, when antennaless females were used there was no significant difference ($P > .50$) between the two males' success. In fact, the vg males were slightly more successful. It was expected that if vg males were equally motivated and just as vigorous as + males, the percentage success would be similar in the case where antennaless females were used, but if vg males were less vigorous, they would thus be less successful than + males under the same experimental conditions.

From the results shown in Figure 1, it can be assumed that the difference in male vigor is not significant and therefore would not account for the large variation in copulation success which was observed when both types of males were separately mated to normal females.

This difference in copulation success seems to be due only to the absence of normal wings. Given the same type of females (antennaless) and the same type of resistance, the + males are no more successful at copulating than the vg males.

Reference: Details of this study may be found in: Seegmiller, R. E., 1967, "Fitness in Drosophila melanogaster." M.S. Thesis, University of Utah Library, Salt Lake City, Utah.

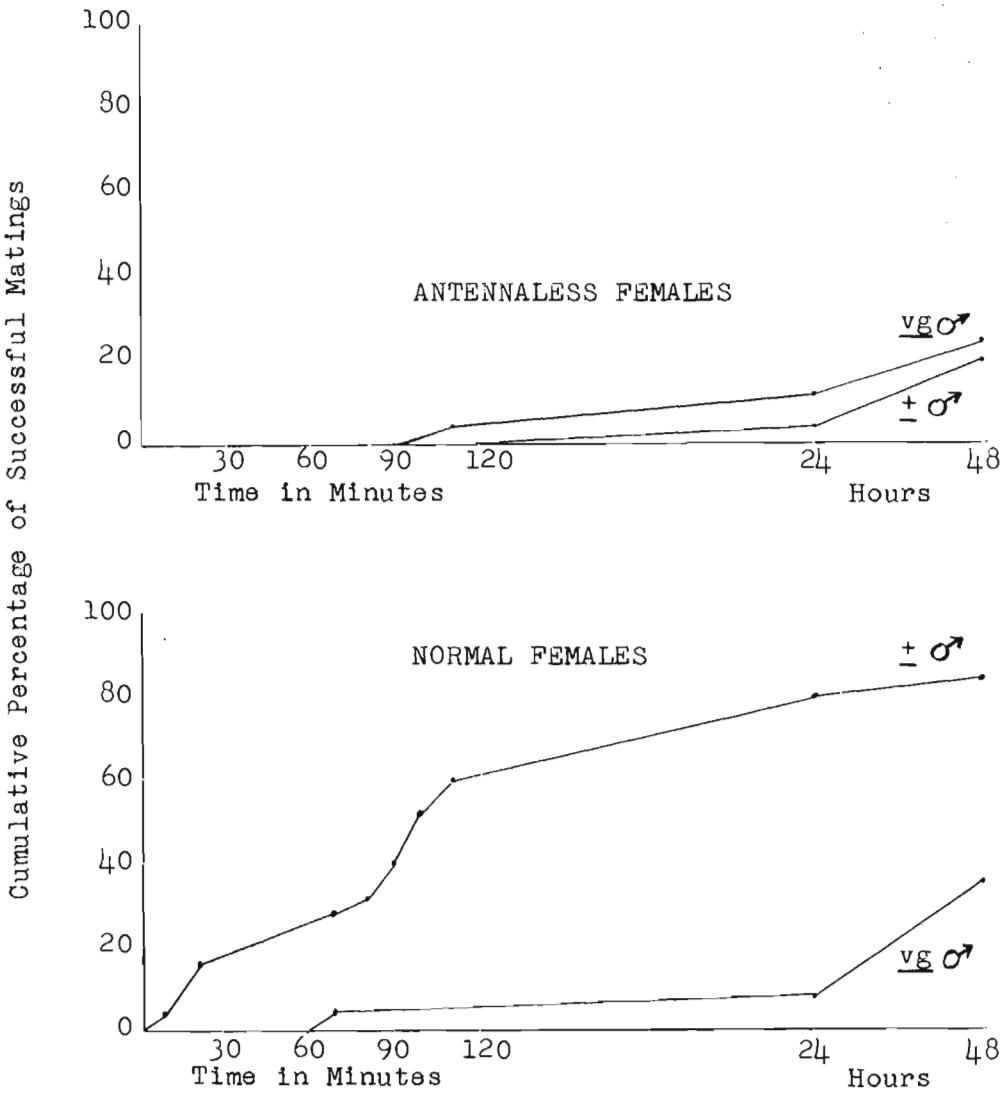


Figure 1.--Frequency of successful copulations of wild-type and vestigial males pair-mated with either normal or antennaless females (25 pair matings for each class).

Gartner, Leslie P. and B. P. Sonnenblick. Rutgers University, Newark, New Jersey.

Drosophila husbandry and extension of lifespan mean and lifespan range.

Some modifications of routine culture conditions and careful operational procedures with small samples of Drosophila adults yield marked effects on mean and individual longevity, demonstrated by % surviving to 100 or more days (Table 1). The populations of the first seven listed groups are the unirradiated samples of seven radiobiological test series, with exposed and unexposed populations cultured similarly during any one investigation. (B. P. Sonnenblick and L. P. Gartner, Radiation Res. 31:612-13, 1967). Test 8 was an independent trial. The data then do not represent a pointed attempt to determine singularly optimal culture conditions, merely an attempt to reduce obvious hazards.

Table 1. Lifespan mean and % surviving to 100 or more days (Canton-S adults, D. melanogaster)

Test No.	$\bar{n}$	Temp. °C. Average	Mean Lifespan of $\bar{n}$ (Days)	No. Surviving to 100 or More Days	% Surviving 100 or More Days
1.	91	21.5 ± .5	44	0	0
2.	160	20.0 ± .5	46	0	0
3.	125	18.5 ± .5	49	2	1.6
4.	66	19 ± .5	64	2	3.0
5.	74	19 ± .5	68	4	5.4
6.	73	19 ± .5	77	15	20.5
7.	50	19 ± .5	68	4	8.0
8.	100	19 ± .5	81	23	23.0

Young Canton-S adults of D. melanogaster, mass bred after earlier pair inbreeding, were used. Temperature fluctuations must be kept minimal and averages are noted in the Table. Standard errors of the mean were prepared for samples by sex and were generally about three and a fraction days; with increasing radiation exposure the S.E. diminished markedly due to a saturation effect of the stress. Tests 4-8, with culture modifications, are compared with Tests 1-3 which were performed some years ago. Most determined efforts at care occurred in Tests 6-8, with 4 and 5 representing our earliest experience with these methods.

Modifications in preparation of standard media include absence of live yeast from surface of vials, no use of benzoyl benzoate, lessened water content of media, placing of cellucotton and preparation of cotton plugs so that adults cannot penetrate either. One etherization only was employed, five pair of flies per vial instead of two, as formerly, were used, and fresh food was supplied weekly with care taken in the passage of organisms to new vials. Vials were examined daily when possible. Relative humidity was low in the first three tests but maintained at 60-65% in later ones.

Whether any, or which, of these factors, acting singly or in concert with deliberately employed, small heterogeneous populations, account for the longevity results evident in the Table we cannot tell. The survivors to 100 or more days were 33 females and 9 males (of 223 individuals) in the last three tests. How would other strains respond to such arbitrarily selected conditions of culture and physical environment? We may note that a strain containing attached-X females and ring-X males, with mutant genotypes, reared simultaneously with Canton + individuals in the same incubator, and as described above for tests 6-8, consistently had 6% of the populations in the three trials (female-male ratio of 2.5:1) living over 100 days, with some of both sexes surviving more than 120 days.

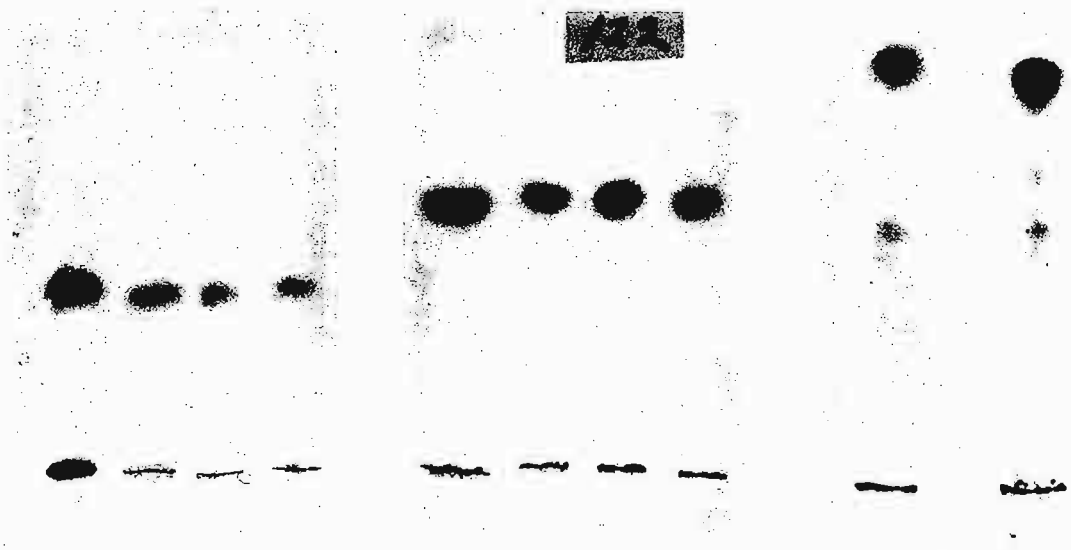
Pipkin, S. B. Howard University, Washington, D.C.

Allelic and non-allelic complementation in Drosophila metzii.

Complex isozyme patterns of octanol dehydrogenase have been observed using 1-octanol as substrate, with agar gel electrophoresis, in strains of D. metzii. True breeding lines 1Tr17e and 58B8, extracted from the Trinidad and Barro Colorado Island, Canal Zone, strains respectively, show a single strongly staining isozyme band at position # 3 with occasional faint bands at positions # 4 and 5 (designated pattern # 3; see Fig. 1a). Hybrids between these lines show the same pattern. In contrast, zymograms of lines 1Tr17h4d and 1T9g, extracted from the Trinidad and Turrialba strains, respectively, show strong staining at position # 6 with faint bands sometimes at positions # 4

and 5 (Fig. 1b). Hybrids of lines 1T9g or 1Tr17h4d and 58B8 show strong staining at positions # 3,4,6, and sometimes # 5 (pattern # 3-6). Also isolated from the Trinidad strain were the almost true breeding lines 1Tr17g17 and 1Tr17g13 with 5,6, or 7 isozymes in positions # 1 to # 6 or # 7 (designated pattern # 1-6; see Fig. 1c). 1Tr17g13 males in pair matings with 58B8 females produced F_1 hybrids usually with 5,6, or 7 isozymes. Fifteen such F_1 males back crossed individually with 58B8 females produced progenies of four patterns: 48 individuals with pattern # 3; 45, with pattern # 1-6; 57, with pattern # 3-6; and 43, with isozymes in positions # 1,2,3 (called pattern # 1-3; see Fig. 2.). This ratio does not differ significantly from that expected from a dihybrid cross (chi square, 2.38; 0.5 P 0.3). Thus 1Tr17g13 males are believed to have transmitted two structural genes for ODH, ODH^A and ODH^B, or more simply, genes A and B, located on nonhomologous chromosomes, to the fifteen F_1 males siring segregating back cross progenies. The F_1 male parents must have had the genotype Aa Bb; the 58B8 females, aa bb. Back cross progeny with the # 1-6 pattern were then Aa Bb; with the # 3-6 pattern, Aa bb; with the # 1-3 pattern, aa Bb; and with the # 3 pattern, aa bb. The same four segregating patterns were seen in similar back cross progenies in which strain 1Tr17e replaced 58B8. Successful prediction of complex ODH ixozyme patterns in crosses between strains of uniform pattern implies these to be the result of subunit composition (see Schwartz, 1962, Proc. Nat. Acad. Sci. U.S. 48: 750-756). Courtright, Imberski, and Ursprung, 1966, Genetics 54: 1251-60) concluded that the ODH molecule in D. melanogaster was a dimer. In D. metzii, a double heterozygote of genotype Aa Bb, is expected to produce ten combinations of dimer ODH molecules: AA, BB, Aa, Ab, aB, Bb, AB, aa, bb, ab, if both allelic and non-allelic complementation occur. If the last three combinations migrate to the same position, # 3, eight distinct mobilities might be expected, and seven have been observed. In 84 singly assayed individuals of the Darien strain of D. pellewae (Fig. 3), a sibling species of D. metzii, 27.4% possessed isozymes migrating to positions # 3-6 and were supposed to have had the genotype Aa bb; 26.2% had isozymes of pattern # 1-3 and were supposed to have been genotypically aa Bb; 35.7% showed a single isozyme at position # 3 and were presumably aa bb; 4.7% had a single isozyme at position # 1 and were apparently aa BB; while 3.5% possessed spaced isozymes at positions # 1, 3, and 6, and were thought to have been genotypically AA BB. The distribution of the dominant three groups of ODH isozymes; i.e., patterns # 3-6, # 1-3, # 3, as alternates in this unselected D. pellewae population implies that these represent a single functional enzyme system in vivo. The low frequency of individuals of presumed genotype AA BB indicates that non-allelic complementation involving A and B subunits may result in a poorly viable individual under crowded culture bottle conditions.

This work has been supported by PHS grant GM 14937.



1a 1b 1c

Fig. 1.

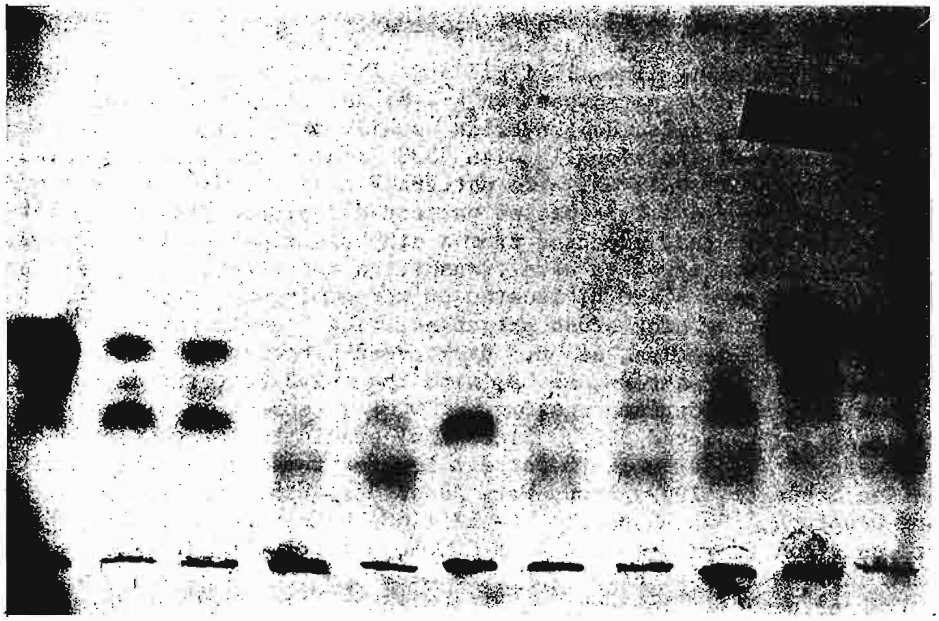
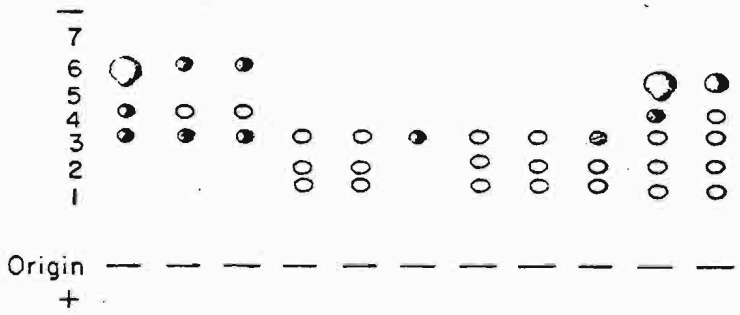


Fig. 2.



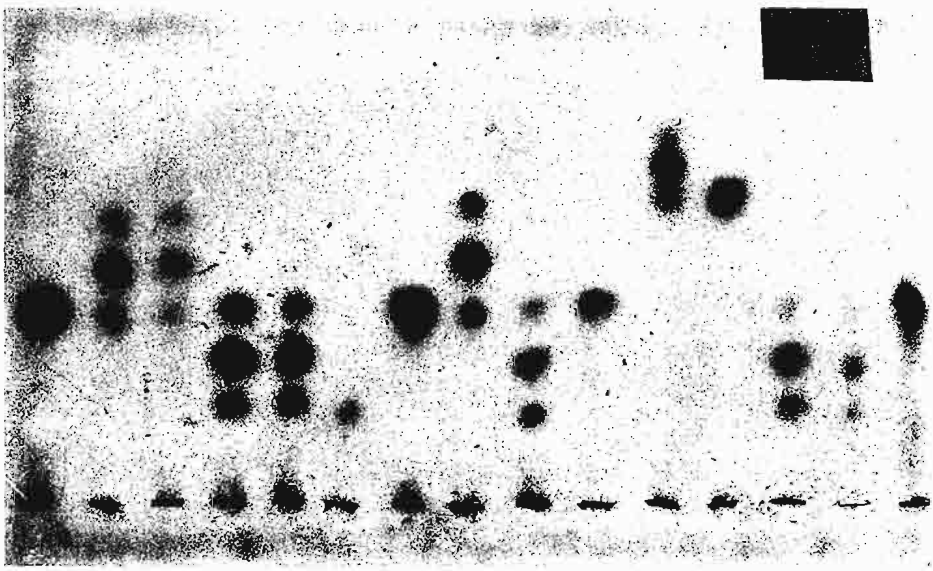


Fig. 3.

Hiraizumi, Y. and D. L. Hartl. The University of Wisconsin, Madison, Wisconsin. Evidence for normal chromosome disjunction in segregation distorter males.

The sex-ratio effect of segregation distorter (SD) (Hiraizumi and Nakazima, Genetics, 55:681-697, 1967) can be formally explained by the disjoining of the SD from the X chromosome in heterozygous males. Such disjunction could result from an "affinity" of SD for X, caused, say, by a small region of homeologous association. It could also result from a large scale nonspecific pairing of SD with X to an extent to exclude both the non-SD and the Y chromosome from the association. The latter possibility would show itself in an increased frequency of second and sex chromosomal non-disjunction in SD males.

The frequency of nullo-II and diplo-II sperms was assessed by mating sets of 15 males (experimental: SD(NH-2)/cn bw, controls: cn/cn bw and Tokyo/cn bw) with 15 attached-II females (dp/dp; cn bw/cn bw) in vials, transferring to fresh medium every 4 days for 16 days, and placing 3-4 pairs of Cy/Pm in the vacated vials to maintain favorable culture conditions. The exceptional progeny are of 3 types: SD or control/cn bw (diplo-II sperm), dp/dp; cn bw/cn bw (nullo-II sperm), SD or control or cn bw/dp cn bw (which arise from detachments of the attached-II, presumably by crossing over). Since the frequency of exceptional types was approximately the same between SD and controls, no progeny tests were performed to determine which were detachment progeny. The frequencies were: SD gave 6 exceptional progeny from 1005 males, or .60%; cn and Tokyo (pooled) gave 6 exceptions from 450 males, 1.3%. One experimental vial gave 10 wild type females and 3 wild type males. This was attributed to pre-meiotic detachment in some female and was therefore not included in the analysis.

A test for the production of XXY females used the fact that an extra Y suppresses the Pm phenotype. R(cn)-14/Tokyo and cn/Tokyo males were brooded at 4 day intervals with Cy bw/Pm females (3 pairs per vial) and the non-Cy progeny were scored for the Pm phenotype. Several parental females were evidently XXY Cy bw/Pm, for they gave a high frequency of secondary exceptions. These were excluded from the following summary: SD gave 6 XXY females out of 15,515 non-Cy progeny, .04%; cn males gave 9/13,954=.06%.

A very small experiment testing for the production of XO males used y w^a/Y·w^+; SD(NH-2) or Tokyo/+ males mated with y w^a/y w^a females. The frequency of exceptions was: SD, 1/243=.41%; Tokyo, 1/221=.45%.

We conclude that there is no significant increase of second or sex chromosomal non-disjunction from SD males and, therefore, that models for the mechanism of SD or for the sex-ratio effect which imply a gross increase in second or sex chromosomal non-disjunction are untenable. While this excludes wholesale X-II pairing, it does not rule out short segment interactions as a workable hypothesis for the sex-ratio effect.

(Supported by NIH Grant GM-07666 and NASA Training Grant NsG(T)-23).

Hijikuro, S. Osaka University, Osaka, Japan. On the binding state of beta-alanine in the pupal sheaths of D. melanogaster.

It has been reported that the binding state of beta-alanine in the pupal sheaths of insects differs from that of other amino acids (Fukushi, Japan. J. Genet. 42, 1967). In the present study, to determine the free amino groups in the pupal sheaths, the pupal sheaths of Oregon-R and ebony^{11} strains of D. melanogaster were reacted with fluorodinitrobenzene by Sanger's procedure. DNP-amino acids in the acid hydrolysate of dinitrophenylated pupal sheaths were, then, extracted with ether and analyzed with IRC-50 column chromatography (Seki, J. Biochem. 47, 1960). In results, beta-alanine in the pupal sheaths of the wild strain was recovered as DNP-derivatives, although no other DNP-amino acids were detected by this method. On the other hand, no DNP-amino acids were detected from the dinitrophenylated pupal sheaths of ebony^{11} strain. The yield of finally recovered DNP-beta-alanine suggested that all amino groups of beta-alanine in the pupal sheaths were in free form. Similar results were obtained with the wild and black pupal strains of Musca domestica and Bombyx mori.

Ogonji, Gilbert O. Howard University, Washington, D.C. A comparison of octanol dehydrogenase isozyme patterns of D. neomorpha, D. simulans, and D. metzii.

A polymorphism of octanol dehydrogenase isozymes has been found in unselected strains of D. neomorpha which closely resembles that which has been genetically analyzed by Pipkin (this issue DIS) in strains of D. metzii. Crude homogenates of single females taken at the height of egg laying have been assayed using agar gel electrophoresis with 1-octanol as substrate for formazan staining. The ODH isozyme patterns of females of the Rio Raposo, Colombia, strain of D. neomorpha (Fig. 1) can be interpreted in accordance with Pipkin's genetic analysis of D. metzii, as being due to two unlinked genes, A and B, and their respective electrophoretic variants, a and b. Assuming the ODH molecule to be a dimer (see Courtright, Imberski, and Ursprung, 1966, Genetics 54: 1251-1260), flies of the first and seventh slits of Fig. 1, going from left to right, are supposed to have had the genotype aa bb. Those of the 4th,5th,6th,8th and 10th slits are supposed to have been aa Bb. The third female from the left is assumed to have been a double heterozygote, Aa Bb; while the next to last female was apparently Aa bb. Females of the unselected strain of D. neomorpha from Barro Colorado Island, Canal Zone (Fig. 2) are thought to have been either aa bb, with a single band in the zymogram; or Aa bb, with four isozyme bands. The slowest migrating isozyme of the group of four is supposed to represent dimers aa, ab, and bb, all of which migrate to the same position. The three faster migrating isozymes in the group of four are supposed to represent dimers AA, Aa, and Ab. Thus both allelic and non-allelic complementation in the formation of ODH dimers is suggested by the presence of four isozymes in certain females of this polymorphic strain. In D. simulans a somewhat different ODH isozyme pattern occurs which can be interpreted as being due to the occurrence of either fewer dimer molecules arising from non-allelic complementation or lower activity of such dimers judged by intensity of formazan staining. Fig. 3 shows the monomorphic strain of an Australian strain of D. simulans. The more cathodally migrating group of isozymes represent alcohol dehydrogenase. The two more slowly migrating groups of isozymes are ODH isozymes. Midway between the latter are faint ODH isozymes. The females are all thought to have the genotype AA BB. The strongly staining two rows of ODH isozymes are believed to be AA and BB dimers respectively, resulting from allelic complementation. The faintly staining intermediate isozymes are thought to be AB dimers arising from non-allelic complementation. A similar pattern of ODH isozymes has been seen in preparations of D. simulans from Lima, Peru; Rarotonga, South Pacific; and Pleasant Hills, Washington, D.C.

This work has been supported by PHS grant GM 14937 and a grant from the Dean of the Graduate School, Howard University.

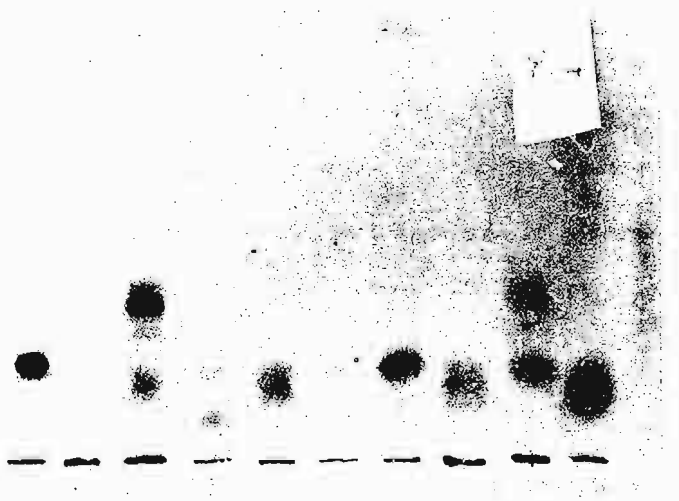
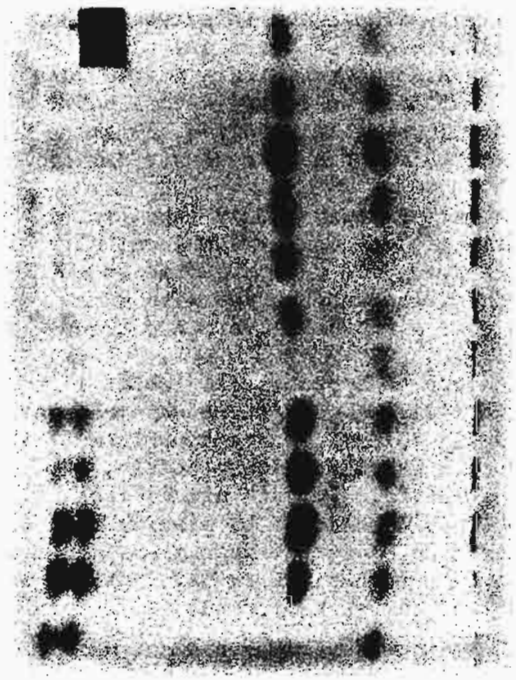


Fig. 1



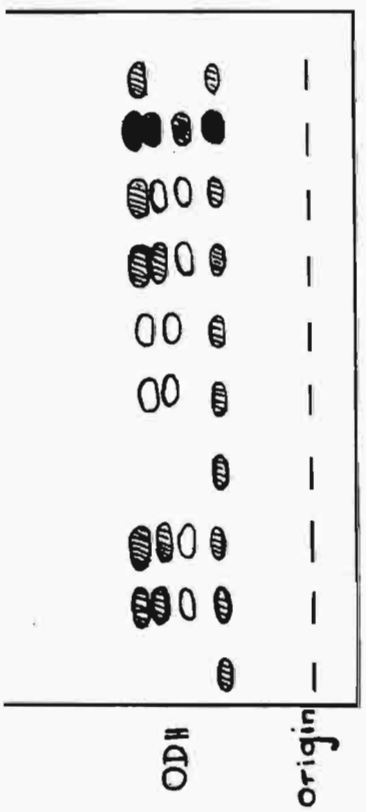
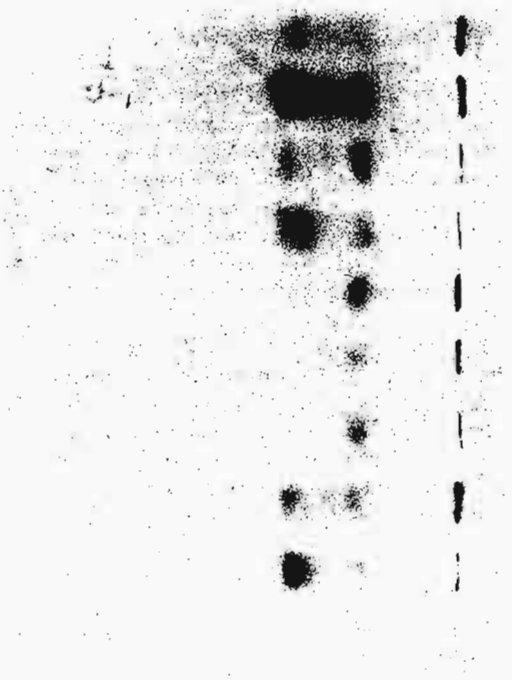


Fig. 2



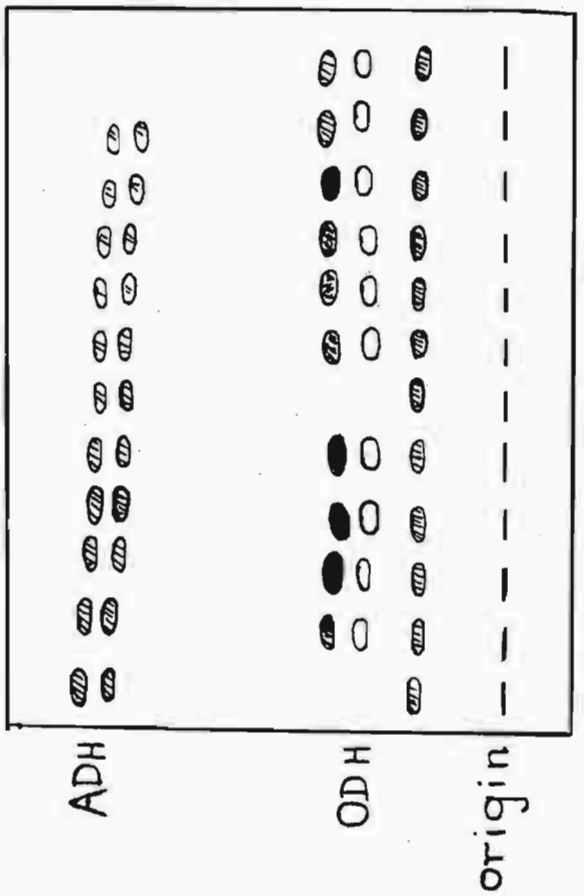


Fig. 3

Williamson, John H. and D. R. Parker. Biology Division, ORNL, and University of California, Riverside, California.
Recovery of multiple-break rearrangements from irradiated oocytes.

Until recently it has been tacitly assumed that only simple two-break rearrangements can be recovered in oocytes of irradiated attached-X females. This report describes four cases of rearrangements involving three or four breaks each. In two cases both a detachment and a Y fragment were recovered in sons of irradiated y v bb/$B^S Yy^+$ females. Both detachments carried B^S and none of the Y fertility factors, while both Y fragments retained the y^+ marker and were capped by 4R. Fertility tests showed that one fragment was a complete Y and that the other was broken betwen kl-2 and kl-3. Thus in one case a minimum of three breaks must have occurred while in the other a minimum of four breaks were necessary to produce both the detachment and the fragment.

The third case was a detachment recovered from irradiated y v bb/$Y^S y^+ Y^L \cdot Y^S$ females that proved to contain kl-5, y^+, and $ci^+ey^+spa^+$. The production of this aberration required a minimum of four breaks. The fourth example came from irradiated y v bb/O females, where the fourth chromosomes were differentially marked (spa^{pol}/Dp(1;4)y^+). Recovered was a detachment capped by 4R (spa^+) along with a free maternally derived recombinant fourth chromosome marked with spa^+ but not y^+. Thus, there were recovered products of two separate two-break events. (ORNL is operated by Union Carbide Corporation for the U.S.A.E.C.)

TECHNICAL NOTES

Keith, A. D. and H. Goldin. University of Oregon, Eugene, Oregon.
A method for rearing Drosophila axenicaly.

In certain instances it is desirable to raise Drosophila under sterile conditions. A relatively simple and effective method has been developed. Seven day old flies, reared on a standard culture medium were removed to pint canning jars (about 1500-2000 flies/jar). The jar was capped with a petri plate containing medium of the following composition: agar, 30 gms; milk solids, 30 gms; tegosept, 1.5 gms; Karo Syrup, 50 ml; fermented apple cider, 45 ml; the mixture was diluted to a total volume of 1 liter.

The jar and petri plate were inverted and the flies were allowed to lay eggs over a 2 to 4-hour period at 25° C. This method produced approximately one egg/hr-female or slightly less. The petri plate was removed and the remainder of the procedure was conducted in a chemical hood which was sprayed with 70% ethanol. The petri plate was flooded with water, the eggs were loosened by gentle brushing and were poured over a 2.5 cm filter in a Buchner Funnel while under suction (about 500 eggs/filter was optimal).

The eggs were dechorionated and surfaced sterilized with 6% hypochlorite (about 1 min.) and washed several times with sterile water. If more rigorous conditions were warranted the eggs were further sterilized with a 10% $CuSO_4$, 0.25% $HgCl_2$ solution and again rinsed several times with sterile water. The filter was removed to a sterile culture bottle, inverted, pressed on the food and removed to the side of the bottle. In this manner most of the eggs adhered to the food. The sterility of this method was determined by placing squashed larvae on a petri plate containing bacterial growth medium. No bacterial growth was noted. Mold or yeast contamination was occasionally noted in the culture bottles (< 5%). These bottles were detected by the characteristic growth and odor of the contaminants.

Johnson, W. W. University of New Mexico, Albuquerque, New Mexico. An improved Drosophila population cage.

The weakest feature of Drosophila population cages has been the means by which food bottles are added to the cages. What is desired is a sturdy cage that not only remains fly-tight when it is handled or moved, but also provides for convenient replacement of food containers. A cage with these properties can be built by permanently mounting screw-type bottle caps in the floor of the cage to receive the food bottles. One advantage of this arrangement over others is that no matter how many times bottles are added and removed, a tight seal between the bottles and the cage is assured. Although the body of the cage can be fashioned from any polystyrene storage box that has a close fitting lid, a box measuring approximately 9-1/2" x 12-1/2" x 4" should be satisfactory for most purposes. A cage of this size will accommodate 15 food bottle sites arranged in three rows of five and will support a population of from 300 to 400 flies. Most adaptable for use as food containers are 1/2 ounce bottles with molded bakelite caps.* The actual construction of the cage is very simple.

For a number of reasons it is more convenient to have the lid of the polystyrene box serve as the floor of the cage. Therefore, the 1-1/4" diameter holes into which the bottle caps are fitted are drilled in the box lid. These holes can be made with an electric drill and a hole saw. Before the caps are inserted, a 1/4" diameter hole is drilled in each to furnish communication between the food bottles and the main chamber of the cage. Then, with the mouths directed outward, the caps are pressed part way through the holes in the lid and epoxy glue or acetone based cement applied to the joints between the caps and the plastic on both sides of the lid. Legs to lift the weight of the population cage from the food bottles can be affixed to the bottom of the cage by drilling a 1/16" diameter hole in each corner of the lid and fastening a 3" length of wooden dowel tightly up against the plastic with a screw. Felt washers should be used to cushion the screws and the legs to prevent the plastic from cracking.

In one end of the cage a 1" diameter sampling hole and a 1-1/2" diameter hole for ventilation are drilled with a hole saw. A sample of the population in the cage can be made by pulling the cork stopper from the sampling hole and inserting a test tube with slanted food medium either to obtain a sample of the population's egg production or for the direct removal of adult flies. Fine mesh organdy glued over the ventilation hole allows adequate air circulation but makes it impossible for flies to escape.

When flies are to be placed in the cage, the seam between the two parts of the box is sealed with masking tape to insure that the floor and the top of the cage remain securely together.

*Bottle #2854 and black molded cap #28400 are obtainable from Twin City Bottle Company, 1227 East Hennepin Avenue, Minneapolis, Minnesota 55414.

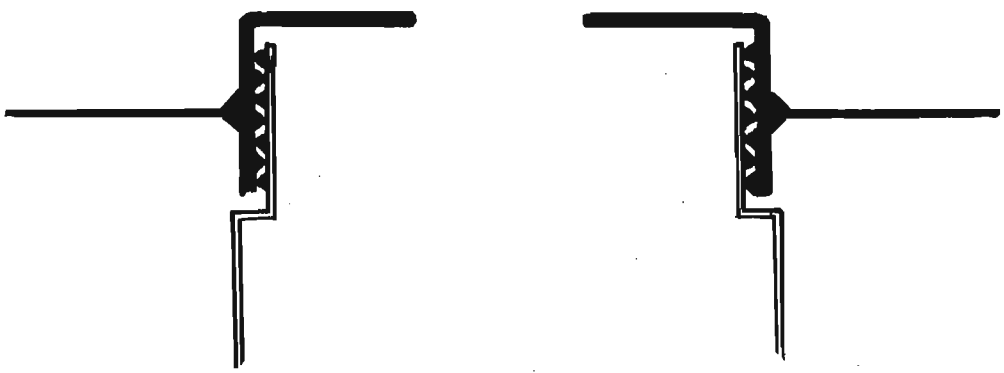
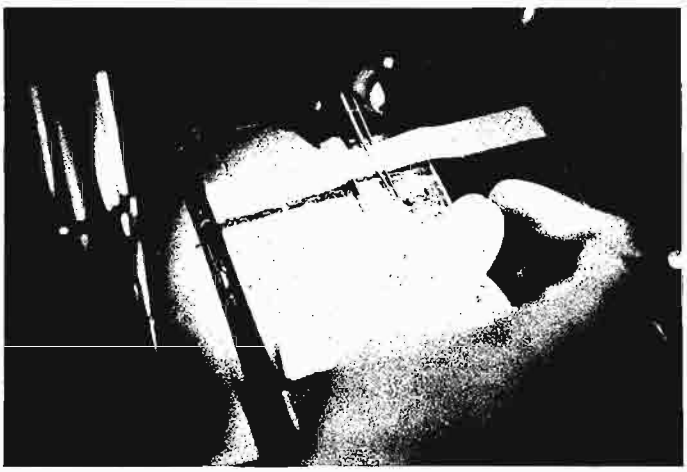


Diagram showing the assembly of a cap and the cage floor.

Félix, E. R., and Rosario Rodríguez.
National Commission of Nuclear Energy, Genetics Program, Mexico City, Mexico.

A microinjection technique for Drosophila.

A modified system for microinjection of larvae and adult Drosophila has been devised from the description of R. Félix and V. M. Salceda (D.I.S. 39: 135). The needle is a 3-5 millimeter long segment of - Santotube Q (Prager, D. J. "Constant volume, self-filling nanoliter - pipette". Science, Vol. 147, N° 3658: 607-608). A broken thermometer calibrated with a syringe of 50 microliters is used to measure the capacity of the needle which may be used several times ensuring the delivery of a constant volume of liquid. The volume contained in each self-filling needle depends on its length and oscillates from 0.5 to 1.0 microliters. The cohesion forces between the solution and the inner surface of the needle is considerably high due to the small amount of liquid to be injected, and requires an air compressor machine connected through plastic tubing to the syringe for such a purpose. A treadle actioned with the foot starts on the compressor delivering a 0.5 kg/cm^2 pressure to the syringe. Working under the microscope the needle is retired from the specimen as soon as the liquid is expelled from the needle. Following the procedure outlined above adults are injected dorsally between the fifth and sixth abdominal tergite with a survival of 80-90%.



Legend: The specimen is fixed with a forceps while the microinjection is applied by means of a compressor machine connected by plastic tubing to the microsyringe.

Hanks, G. D., A. LaVell King and A. Arp.
University of Utah, Salt Lake City, Utah.

Control of a gram negative bacterium in Drosophila cultures.

It was found that a rod shaped bacterium which colors the food brown and is commonly found in cultures of Drosophila melanogaster could be controlled or eliminated in stock cultures by adding Zephiran (a brand of refined benzalkonium chloride) to the nutrient medium at the time the propionic acid is added and rapidly transferring the flies. Usually 7 to 10 daily transfers (2 ♂♂ and 2 ♀♀ per vial) were enough at the concentration used to complete the elimination of the bacterium. Two mls. of the concentrated (17%) solution of Zephiran is used for 1000 mls. of H_2O in the nutrient medium. The nutrient medium in addition consists of agar, yeast, cornmeal, sugar, and propionic acid. A few tests were run on the production of flies allowed to develop in 3 different concentrations and there was no detectable lowering of production with increasing amounts of Zephiran. The cooperation of Dr. Paul Nicholes in the Department of Microbiology is greatly appreciated.

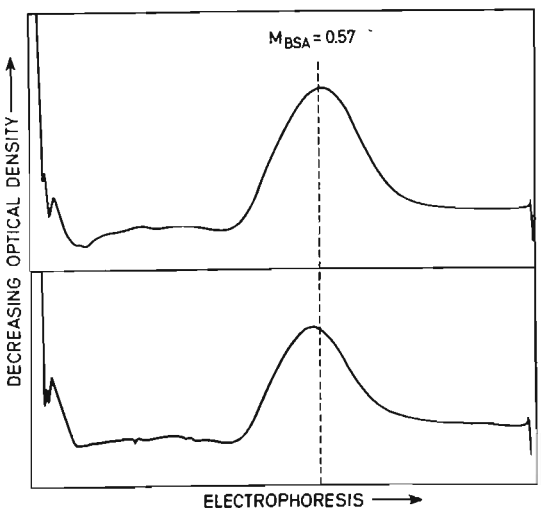
Boyd, J. B. Max-Planck-Institut für Biologie, Tübingen, Germany. Mass preparation of salivary glands from prepupae of Drosophila hydei.

To facilitate biochemical studies of the prepupal salivary glands, a method has been developed for the mass isolation of these organs. The procedure requires very little work with a microscope and permits one person to prepare about 500 glands/hour.

The developmental stage of the animals is determined to within one hour either by collecting white prepupae or by the floating method of Mitchell and Mitchell (DIS 39:135). Several hundred synchronized prepupae are placed in one side of a large petri dish which is tilted at a slight angle in a dish of ice. About 5 ml of Ringer is placed in the lower side of the dish. Each prepupae is held in the middle with blunt forceps while one tip of a pair of sharp forceps is inserted into the posterior tip of the operculum. The contents of the animal are then forced through the hole by firmly squeezing the blunt forceps, and the animal is placed in the cold Ringer.

After the desired number of animals has been so treated, 15 ml of Ringer is added and the suspension is pipetted up and down in a fire-polished 10 ml pipette that has had the tip cut off to a diameter of 6 mm. This step frees the glands from the other organs and removes all fat from glands obtained from prepupae which are eight hours or older. Pipetting is done gently, but considerable air is included to help break up the fat body. The suspension is filtered through a tea strainer into a beaker at 0° C. The cutical is washed in a stream of Ringer, resuspended in Ringer, and the pipetting and straining steps are repeated. The glands, which settle rapidly, are washed twice with 250 ml of cold Ringer.

If fewer than 100 glands are required, they can be picked out quickly under a dissecting microscope. For larger quantities, about 1000 glands are layered on a discontinuous gradient containing 8 ml 75%, 12 ml 62%, and 20 ml 20% sucrose (w/v). The sucrose is buffered at pH 7.0 with Tris. After spinning at 20,000 rpm for 7 min. and at 4° C in the Spinco SW 25,2 rotor, the glands are removed from the top of the 75% sucrose layer and washed in Ringer. The few contaminating testes are removed with forceps.



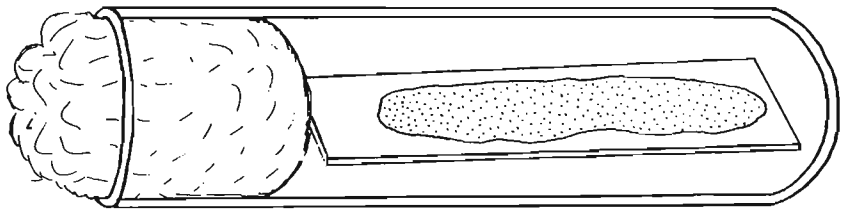
The recovery and quality of the glands depend on the developmental stage used. Yields of 50-75% are obtained from animals 8 - 14 hours after puparium formation. In white prepupae, glands are too firmly attached to the larval mouth parts to be recovered by this method. Yields from slightly older animals are low and the recovered glands have some attached fat body. At 25° C and between 16 and 16.5 hours after puparium formation glands can no longer be recovered.

This method has been used to study DNases in these glands after their separation in acrylamide gel (Boyd and Mitchell, Anal. Biochem. 13, 28, 1965). The enzyme patterns, presented in the top and bottom of the figure, were obtained from hand dissected and mass-prepared glands respectively. The gels were incubated at pH 9 in the presence of Mg^{++}. This enzyme activity, which is not detected in the larval salivary gland, is only slightly reduced by the isolation procedure. The morphology of the glands does change, however, as a result of centrifugation in sucrose. For some purposes it is, therefore, desirable to replace the sucrose with Ficoll as used by Fristrom and Mitchell (J. Cell. Biol. 27, 445, 1965).

David, J. and M. F. Clavel. University of Lyon, France. A new method for measuring egg production without disturbing the flies.

For metabolic studies on Drosophila adults, egg production appears to be the most easily measured character. As a consequence, a large number of references, describing various techniques for determining the daily egg production, are to be found in the literature. All these methods have some common features, particularly the necessity of frequent renewals of the food, which also serves as a site for egg deposition.

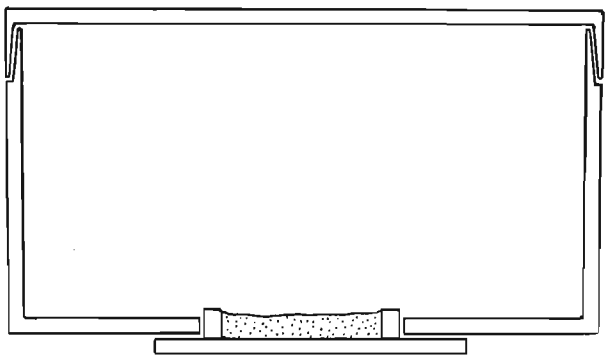
A



For many years we have used a method which is illustrated in Figure A. The flies are kept in a glass tube (11 x 3 cm) closed with a cotton plug. The food is deposited on a glass slide which is introduced in the tube and maintained by the plug. This method has the following inconveniences:

- it is necessary to shake the tube when changing the food, to prevent the flies from flying out
- when introducing a new slide, some flies may be crushed
- the thickness of the food is irregular and its area variable
- the food usually begins to dry near the plug

B



In order to overcome these deficiences, a new method has been worked out. The flies are put into a box of plastic material with a circular hole in the bottom. The food is placed in a small round plastic recipient (4 mm high) exactly fitted to the opening of the cage (see fig. B). The advantages are:

- the possibility of changing the food without disturbing the flies
- the constancy of the food area
- the smaller dessication of the food due to its greater thickness
- the greater volume available to the flies, giving them the possibility of small flights and allowing them a more normal behavior.

Some experiments were performed to compare the results given by the two methods. With the plastic cages, two food areas were studied. The fecundity was always measured on groups of 4 females and 5 males. The results, summarized in the following table, are presented in two ways:

- the maximum daily egg production (average of the daily output from the 4th to the 9th day)
- the total egg production per female during its first 10 days of life.

method	food area (cm^2)	maximum daily egg production	total fecundity to 10 days	number of experiments
glass tube	10	85.7	739.2	4
plastic cage	9	83.2	721.0	4
plastic cage	4.5	84.6	726.6	5
mean		84.5 ± 2.02	728.8 ± 20.1	

Variance analysis shows that there are no significant differences between treatments. So it may be concluded that an area of 4.5 square cm is sufficient for egg deposition of 4 females.

It was also of interest to compare the frequencies of loss of flies with both methods. For this it was possible to pool a larger number of experiments. The data, concerning the proportion of flies still under test on the 10th day, are the following:

	female	male
glass tube	94.5% (n - 92)	87.8 (n = 115)
plastic cage (small area)	97.7% (n = 86)	99.0 (n = 105)

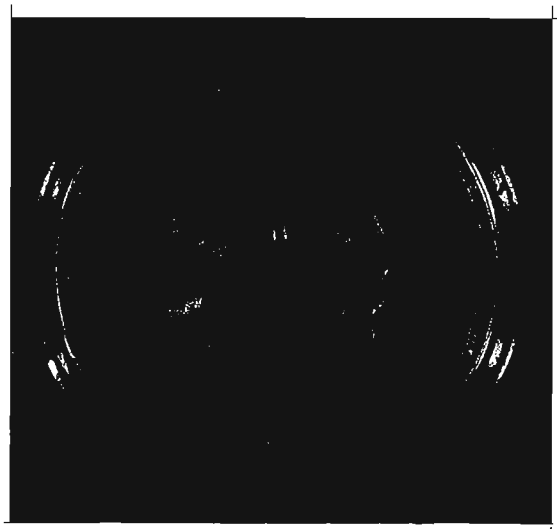
It appears that the new method affords greater safety by preventing the flies from escaping.

Because of its above mentioned advantages, the plastic cage technique (with small area of food) is now preferred. It is also worth emphasizing that this method allows new types of experiments, for example, food preference studies.

Gottlieb, F. J. and B. Langer. University of Pittsburgh, Pittsburgh, Pennsylvania.

A device for holding alcohol preserved specimens during microscopic examination.

Scoring morphological traits, such as bristle numbers, on alcohol preserved Drosophila requires a means of holding and orienting the submerged specimens without damaging them. A simple device has been used successfully in our laboratory. A clean, dry Syracuse watch glass (50 mm ID) is filled with hot dental wax (SHUR Pink Base Plate Wax). When the wax has cooled, a concave depression 25mm in diameter and 7-10 mm deep is excavated. The brush and metal collar of a size 2 or 3 red sable watercolor brush (Grumbacher "Showerproof") is removed from the wooden shaft all but about 12 mm of the metal is removed. This brush is then inserted horizontally metal end first, in a hole, made with a hot wire, in the side wall of the depression and sealed in place (see photograph). The bristles should touch the bottom of the depression so that no animals will fall under the brush.



To use, the depression is filled with alcohol (or other preserving fluid). The specimens are oriented by gently entangling them in the brush hairs and are thus held firm for examination under the dissecting microscope.

Frankel, A. W. K. and G. E. Brosseau, Jr. University of Iowa, Iowa City, Iowa. A Drosophila medium that does not require dried yeast.

In the past, our laboratory has experienced difficulty with the standard cornmeal-molasses medium. Occasionally we had periods in which pair matings and low viability stocks grew poorly. This appeared to be correlated with an aging of the dried, heat killed Brewers yeast. In addition we have experienced difficulty in obtaining fresh supplies of dried yeast. Therefore we decided to find a non-spoiling substitute for the yeast. The substitute finally adopted is a mixture of yeast extract and non-fat dry milk solids. Yields using the recipe given below are as good or better than those we obtained using the yeast formula. We have tried three brands of yeast extract and found no difference between them and no evidence of deterioration with age (the oldest yeast extract was more than three years old). While this recipe is slightly more expensive than media using yeast, we feel its advantages offset the extra cost.

Recipe for cornmeal-molasses-yeast extract-milk solids medium.

Ingredient	Amount for 150 quarter pint bottles[1].	% of total water
Water	4286.6 ml	79.62
Agar	30.3 gm	0.56
Molasses	692.7 ml	12.87
Cornmeal	578.7 gm	10.75
Milk Solids[2].	118.3 gm	2.20
Cold Water	930.3 ml	17.28
Yeast Extract	30.3 gm	0.56
Boiling Water	166.7 ml	3.10
Acid Mix[3].	60.3 ml	1.12

1. About 37,800 cc of medium.
2. Carnation non-fat dried milk solids were used but any brand should be suitable.
3. Propionic-Phosphoric acid mix (from E. B. Lewis DIS 34:117) was used as a mold inhibitor.

Directions for preparing the medium:

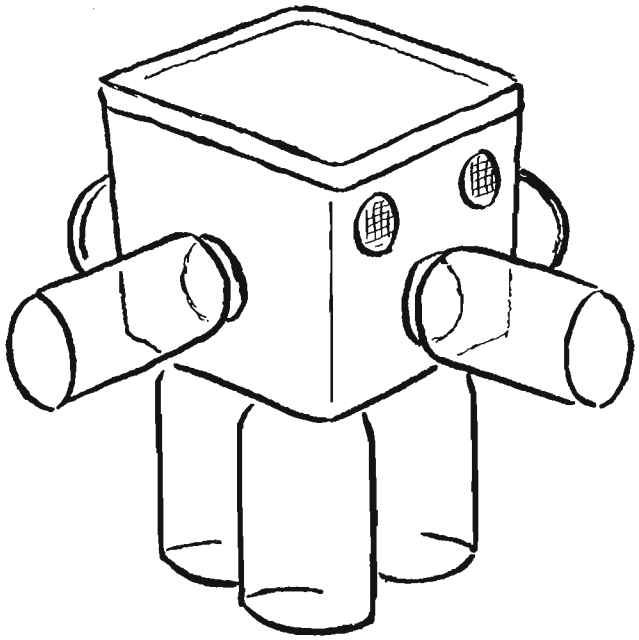
1. Dissolve agar.
2. Reduce heat and add molasses (We use sorghum syrup because of local availability).
3. Dissolve yeast extract in boiling water.
4. Make a slurry of cornmeal and milk solids using the cold water.
5. Stir the cornmeal-milk slurry into the agar-molasses mixture and then add the yeast yeast extract solution (adding the latter earlier makes a deficient medium). Heat to 80-82° C.
6. Turn off heat, stir in acid mix and pour.

Grossfield, J. Purdue University, Lafayette, Indiana. Dental Rolls for pupation sites.

To facilitate the collection of large numbers of virgin adults of species less productive per bottle than D. melanogaster, I have been using dental rolls as pupation sites. Dental rolls (3/8" by 1-1/2") are poked into the food medium when 3rd instar larvae begin to crawl and left until the rolls are full of pupae. The pupae-laden rolls are then poked into the surface of a fresh bottle of food. Generally two bottles of larvae result in one of pupae on rolls (6-7 rolls/bottle). When adults emerge they can be shaken out without the accompaniment of liquified medium. This method works well with a reasonably large number of species, with the trenchant exception of D. virilis. The rolls are available from Johnson and Johnson at 2000 for $6.50 (suggested price as of March 6, 1967). A dispenser is available if one feels the need.

Ayala, Francisco J. Rockefeller University, New York, New York. An inexpensive and versatile population cage.

I have designed a small population cage which is being used in several laboratories for both research and teaching. The cages support populations of 1000-3000 adult flies, which can be easily censused. The cages can be washed and reused. They occupy little shelf space. The cost of materials is less than $1 per cage.



The cages are made of 1-pint flexible plastic containers with lids, available commercially for refrigerator storage. Four holes, 1 1/8" diameter, are cut in the bottom to fit 1-ounce wide-mouth specimen jars with Drosophila medium. Two small ventilation windows, 7/8" diameter, are cut on one side; squares of 40 gauge brass mesh are secured to the plastic by heating them. Opposite the ventilation windows an additional 7/8" hole is cut which remains plugged except when flies are introduced. Etherized flies are placed in 1-cent coin collector plastic tubes, which fit these holes, and allowed to fly into the cages as they recover. To take a census, a plug with a few drops of ether is placed in this window; the cage is inverted and gently shaken until the etherized flies fall on the lid which is then removed.

If more than four food jars are desired, four additional holes can be cut, one on each side of the cage (see figure). If a larger cage is preferred, 1-quart or 2-quart containers may be used which can support more food jars. Using a drill press, I built 40 cages with the help of a student in about three hours.

Schmidt, P. and H. Traut. Institut für Strahlenbiologie, Westfälische Wilhelms-Universität. A method facilitating the counting of Drosophila eggs in experiments on dominant lethals.

In our experiments on dominant lethals we use the following method which facilitates the counting of the eggs.

The food medium blackened by adding powdered charcoal is poured into petri dishes. While the food is still soft small disks (diameter 24 mm) of wire mesh (1.5 x 1.5 mm) are slightly pressed on it. The disks are then tight on the medium surface especially if ordinary meal (yielding a smoother medium surface) is used instead of cornmeal. The wire mesh disks serve as a "raster" facilitating egg-counting considerably. The eggs are viewed at a magnification of 10 times. So far no differences in the number of eggs laid per female have been noted when either an unpainted (but colourlessly lacquered) or a painted copper wire mesh is used. In the former case it is advisable to filter the light of the microscopic lamp by blue glass. The area covered by the disk is then smeared with a baker's yeast suspension. The flies are retained on this area by a glass cylinder (diameter 28 mm, height 80 mm), the upper end of which is closed by cotton. Several cylinders can be pressed into the medium of one petri dish. The use of vials with bottom (instead of glass cylinders) which are inverted on the petri dishes has proved to be unsatisfactory because it results in a depression of the viability of the flies, the number of eggs laid per female, and the hatchability of the eggs.

We prefer to put only one female per cylinder in order to be able to exclude those (probably uninseminated) females laying only non-hatching eggs.

Fristrom, James W. and William Heinze.
University of California, Berkeley, California. The preparative isolation of imaginal discs.

In an earlier paper (Fristrom and Mitchell, 1965) a method was published for the isolation of 2-4,000 discs. It was confidently stated that the limiting factor controlling the yield of discs involved only the number of larvae used. Subsequently, using large numbers of larvae (300-400 gm) it was possible to obtain only 4-6,000 discs indicating that modifications in the method were necessary to obtain a substantial increase in the yield of discs. The following procedure describes a method which normally yields about 40,000 discs, and has given preparations containing as many as 70,000 discs. Only details which differ from the original paper are included. The method starts with ~ 200 gm of larvae.

1. Larval Cultures: The method of Mitchell and Mitchell (1964) is used. Eight boxes of larvae, obtained in one 8 hour and one 16 hour collecting period are used. The larvae are grown at 25° C in 65% relative humidity for five days (after removal of the box from the cage). Only larvae which have crawled to the top of the box or the side of the box are used. Usually some pupae have formed at the time of use.

2. Grinding the Larvae: The larvae are ground in four separate 50 gm batches. The larvae are suspended in cold Ringer's solution (final volume 250 ml) and poured immediately into the running grinder. The grinder is washed with a small volume of Ringer's solution (25 ml).

3. Sieving the Homogenate: The homogenate is poured rapidly through three sieves into beakers; the first two sieves are stainless steel and the last one is made of silk bolting cloth. The sieves have respectively 2 mm, 0.8 mm and 0.2 mm openings. The stainless steel sieves are washed briefly with a stream of Ringer's solution driven through a needle valve at 12 P.S.I. from a 15 liter reservoir. The silk cloth is washed thoroughly until the 1.5 liter glass beaker used to catch the last filtrate is full. The original homogenate and first two washings are caught in 1 liter polyethylene beakers.

4. Reducing the Volume: All steps are carried out in ice baths. The initial 1.5 liter volume is reduced to 50 ml in three steps. The initial suspension is allowed to stand for 10 minutes and the top 1300 ml are removed with an aspirator. The remaining material is transferred with washing to a 250 ml beaker where it is allowed to settle for five minutes. The top 175 ml is removed with an aspirator and the remaining material is transferred with washing to a 100 ml beaker. After settling for three minutes and removing the top 60 ml the material is finally transferred to a 50 ml beaker. After all four concentrations are completed the sediment is combined in two 50 ml beakers, allowed to settle for two minutes and the top 30 ml of fluid is removed.

5. Washing the Material: The material is then run through several washing cycles:

(1) The material is suspended in 50 ml of Ringer's.
(2) The material is allowed to settle 1 to 1.25 min.
(3) The fluid is slowly aspirated from the top down until ~ 15 ml of fluid is left.
(4) The cycle is repeated.

About 20-30 cycles of washing are required. We have designed a machine which will perform all the above washing steps automatically. We will happily provide a description and wiring diagram to anyone desiring to build it.

6. Centifugation: The washed material is then spun on six discontinuous gradients (14 and 20% Ficoll in Ringer's). We have discontinued use of the continuous Ficoll gradient although it may still be used.

7. Manually Removing Debris: Non-disc material can be removed from the preparation with forceps or by using a micro-needle (suitable for disc transplantation) which is attached to an aspirator. Usually about one hour of "picking" is required.

The entire procedure takes about four hours. Because of the increased number of discs, and the decreased efficiency of removing debris the preparations are less free of debris than before, averaging between 90-93% pure. The types of discs are recovered at different frequencies. Leg discs now predominate and increased numbers of genital and eye discs are found. Many variations may be applied to the procedure. The material caught on the first sieve may be reground yielding, at a maximum, an additional 20,000 discs. The material caught on the first sieve may be resuspended and resieved yielding a higher proportion of wing discs.

References:

Fristrom, J. W. and H. K. Mitchell, J. Cell Biol., 1965, v. 27, 445-448.
Mitchell, H. K. and A. Mitchell, D.I.S., 1964, v. 39, 135.

Richardson, R. H. and M. P. Kambysellis. University of Texas, Austin. A cactus-supplemented banana food for cultures of Repleta group Drosophila.

A number of investigators over the years have found that the addition of various plant extracts resulted in improved cultures of certain "problem" species of Drosophila. However, it is frequently a problem to be approached anew for a new series of experiments, since food formulations for routine stock cultures rarely are published.

Recent studies with Drosophila aldrichi and D. mulleri required efficient culturing of large numbers of isofemale lines. When the wild female is placed into a vial, the low density of eggs and frequent high density of mold spores result in failure of flies to develop from most of the vials.

By using a cactus-banana food in 7/8" (ID) vials, we were able to get about 80% fertile isofemale lines from aldrichi and about 95% fertile isofemale lines from mulleri. The food formula used was

5 l. water
110 g. flake agar (S. B. Penick, Jersey City, N.J.)
2 Tbs. malt extract (Blue Ribbon)
14 bananas
200 g. brewer's yeast (Fleishman's, type 2019)
170 ml. white Karo syrup
5 l. prickly pear slurry
56 ml. 95% ethanol
56 ml. propionic acid stock solution

The prickly pear (Optunia lindheimeri) stems were gathered at various times during the year, and the season of gathering had no discernible effect upon its benefit to the flies.

The cactus was diced into about 1" - 2" pieces, autoclaved for 45 minutes at 118° C, and ground thoroughly in a food blendor (Waring Commercial Blendor). To improve circulation during blending, about 200 - 300 ml. of water was added to each batch of cactus (about 1200 ml.) being ground. In order to use the food dispensing pump (Faberge and Cave, D.I.S. 26: 129) this slurry must also be strained through 1/4" ("Hardware cloth") wire mesh to remove the vascular tissue which is not cut by the blendor knives sufficiently to prevent clogging the pump.

The propionic acid stock is made by mixing 5 ml. propionic acid and 1 l. water.

It was found beneficial to periodically add a few drops of dilute baker's yeast (Fleishman's) to the culture vials, after the larva were present. It is questionable if the yeast had any effects, but certainly the water was beneficial. The frequency of adding the yeast suspension varied with the humidity of the season, but the surface of the food was never dry nor "soupy."

Similar food modifications have been tried before. For example, Crow (1942, Univ. Texas Pub. no. 4228) added prickly pear fruit for culturing these species. Also Wagner (1944, Univ. Texas Pub. no. 4445) extensively examined the food components which gave good cultures for these two species. The cactus fruit juice alone was an incomplete medium for either aldrichi or mulleri, while an addition of any of several yeast strains gave good survival. Thus the yeast growing on the food was of great importance, probably from its contribution of sterols to the diet of the fly.

Also Spieth (personal communication) has used pieces of sterile cactus placed on the surface of banana food to culture several species of the Repleta group. We have also tried this method, which works well for larger numbers of flies in a vial, but a single female frequently becomes stuck in the mucilaginous cactus juice. Also the labor of placing pieces of cactus in vials is much greater than mixing it in the food.

This work was supported (in part) by a U.S. Public Health Service research grant (GM-11609 to W. S. Stone and M. R. Wheeler) and training grant (2 T1-GM-337-06 and GM 00337-07 to R. P. Wagner et al.) from the National Institutes of Health.

Zimmerman, W. F. Amherst College, Amherst, Massachusetts. A technique for mass rearing and collection of Drosophila pupae and adults.

Studies of the circadian rhythm of eclosion in Drosophila pseudoobscura necessitated the development of a technique for mass rearing and collection of pupae and adults. The technique may be of interest to researchers faced with similar problems in the same or other insects.

Drosophila pseudoobscura were reared at 20°C in plastic refrigerator boxes (27 cm x 19 cm x 9 cm) whose tops had been cut out with a jig saw (leaving the frame) and replaced with nylon curtain material glued to the edge of the top with methylene chloride. A 1 cm hole was drilled in the top, so that flies could be dropped via a funnel into the box.

Four hundred to six hundred parent flies 1 to 3 days old were placed in such a box containing 13 to 20 mm of fly food medium and patches of live Brewer's yeast in water suspension. Two to three "valleys" were made lengthwise in the food, since crowded females lay eggs more readily in creviced areas.

The flies were allowed to lay eggs 3 to 5 days and were then removed by inverting the box and anaesthetizing them with CO_2. After the unconscious flies fell onto the detachable top of the box, they were either discarded or transferred to fresh food. To prevent the future escape of larvae, masking tape was used to seal the edges of the box.

Seven to nine days later, the boxes were opened and larvae on the sides of the box returned to the moist food below. If the food supply was seriously depleted, live yeast in a suspension of cream consistency were added. Then, 10 to 12 large sheets of plastic were crumpled, twisted, and placed lengthwise in the box. The plastic sheets were obtained by cutting off the 2 edges of large refrigerator bags (Baggies) on a cutting board. Almost all of the larvae pupated on these plastic sheets.

Pupae were collected 8 to 9 days later by submersing and agitating the plastic sheets in a 15-gallon aquarium filled with water at 20°C. The plastic sheets were then removed; pupae floated to the surface, while the food and larvae sank. Pupae were collected from the surface with sieves made of cylindrical refrigerator containers, the bottoms of which had been replaced with nylon curtain material.

The sieves containing pupae were placed for a few minutes on paper towels to remove excess water and were then placed in a stream of air to complete the drying. Once dry, the pupae were knocked out of the sieves into a plastic box where they were brushed and shaken to separate clumps. From a rearing box of the size described, about 10 to 15 thousand pupae can be collected.

Large numbers of freshly eclosed flies can be easily collected by gluing the pupae to the inside of an empty plastic box. The bottom and side of the box is smeared with Elmer's Glue-All, slightly thinned with water. Then the pupae are shaken inside, until all are stuck. In Drosophila pseudoobscura, almost all flies emerge within several hours after dawn of a 12 hours light/12 hours dark cycle. The freshly emerged adults can be collected by inverting the box and anaesthetizing the flies with CO_2. Several such collections can be made on successive days.

The mortality resulting from the washing, drying, and gluing is less than 5%. Death is most frequently due to blockage of the emerging adult because the operculum happens to hit the glue first.

Although the technique was developed for Drosophila pseudoobscura, it could be used and modified for other insects as well.

Supported by research funds granted Princeton University (laboratory of Dr. C. S. Pittendrigh) by the Office of Naval Research, Nonr 1828 (58).
Mr. E. Pauming made several good suggestions during the development of this technique.

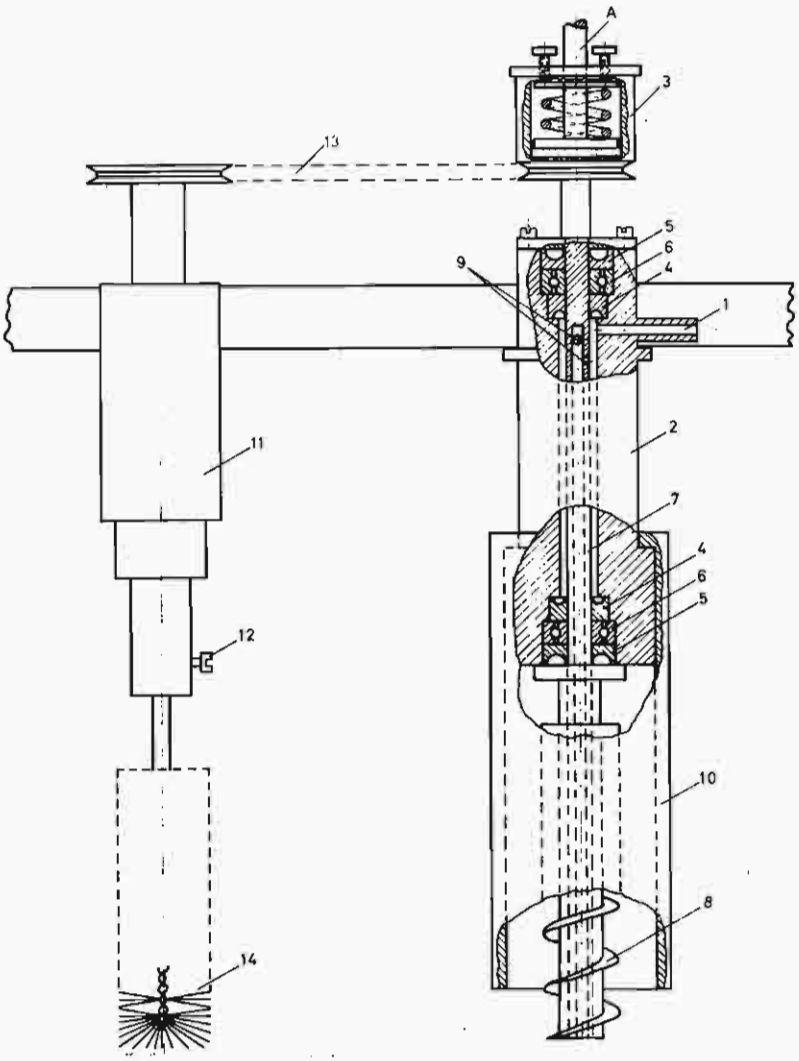
Strömnaes, Öistein. University of Oslo, Blindern, Norway. Insecticides.

We use Drosophila in laboratory class work in genetics and the regular cornmeal molasse agar-agar medium. For two weeks all experiments failed, also the extra cultures made up by the teaching staff. At that time the cornmeal was suspected to be poisoned. The firm delivering the cornmeal told that it was stored in large containers. To avoid hatching of Ephestia they had sprayed the top layer with an insecticide. When our order came they sent us a sack of cornmeal taken from the top layer, believing they made us a great service.

Our conclusion is that from now on we have to order insecticide-free cornmeal.

Wind, Heinz and Heinz Mertens. Institut für Strahlenbiologie, Münster (Westf.) Germany. A device to clean culture tubes.

In order to clean Drosophila culture tubes in relatively short time, we have developed a cleaning device which is in operation in our laboratory since a year and proved to be very helpful.



In principle it consists of a waterflown rotating spiral of polyamide (see Fig., position 8) with the help of which the used culture medium is detached from the glass walls and - based on the Archimedes Principle - spiraled out. The finer particles still attached to the glass walls are removed by a a rotating brush (14). The whole assembly is driven by an ac motor provided with a gear that can be continuously regulated. The number of revolutions is about 250 rpm. To protect the worker from being hit by the glass pieces of a breaking tube, a safetyclutch (3) is built between the motor and the washing assembly. The washing unit is built as follows: Through the inlet (1) the water flows into a chamber contained in a brass cylinder (2). The upper and the bottom ends of the chamber are sealed with Simmer rings (4), thus protecting the ball bearings (6) from the inflowing water. The outer Simmer rings (5) protect the ball bearings from the atmospheric humidity. In the ball bearings (6) a hollow waterflown shaft (7) rotates. To the distal part of this shaft the polyamide spiral (8) is fastened tightly. The proximal part of the shaft is closed and connected with the safetyclutch (3). Two holes (9) are drilled in the proximal part of the hollow shaft. The water enters the shaft through the holes and flows out of the distal end of the spiral under pressure, thus helping to eject the medium out of the tube. The plexiglass tube (10) gliding on the cylinder (2) protects the worker from the spray. The support (11) for the brush (14) is built in the identical way. The brush is not waterflown (normal reagent-glass brush); but if required a waterflown brush can be inserted. The brush is fixed with a screw (12) to the support. The motor-driven spiral is connected by a strap (13) (which is running over pulleys) with the brush and thus both are driven simultaneously. The plan of the safetyclutch is simple as can be seen from figure. The shaft of the safetyclutch is connected to the gear. The whole assembly is mounted on the wall at an angle so that the ejected medium is collected in a vessel placed below without being hit by the water stream coming out of the spiral.

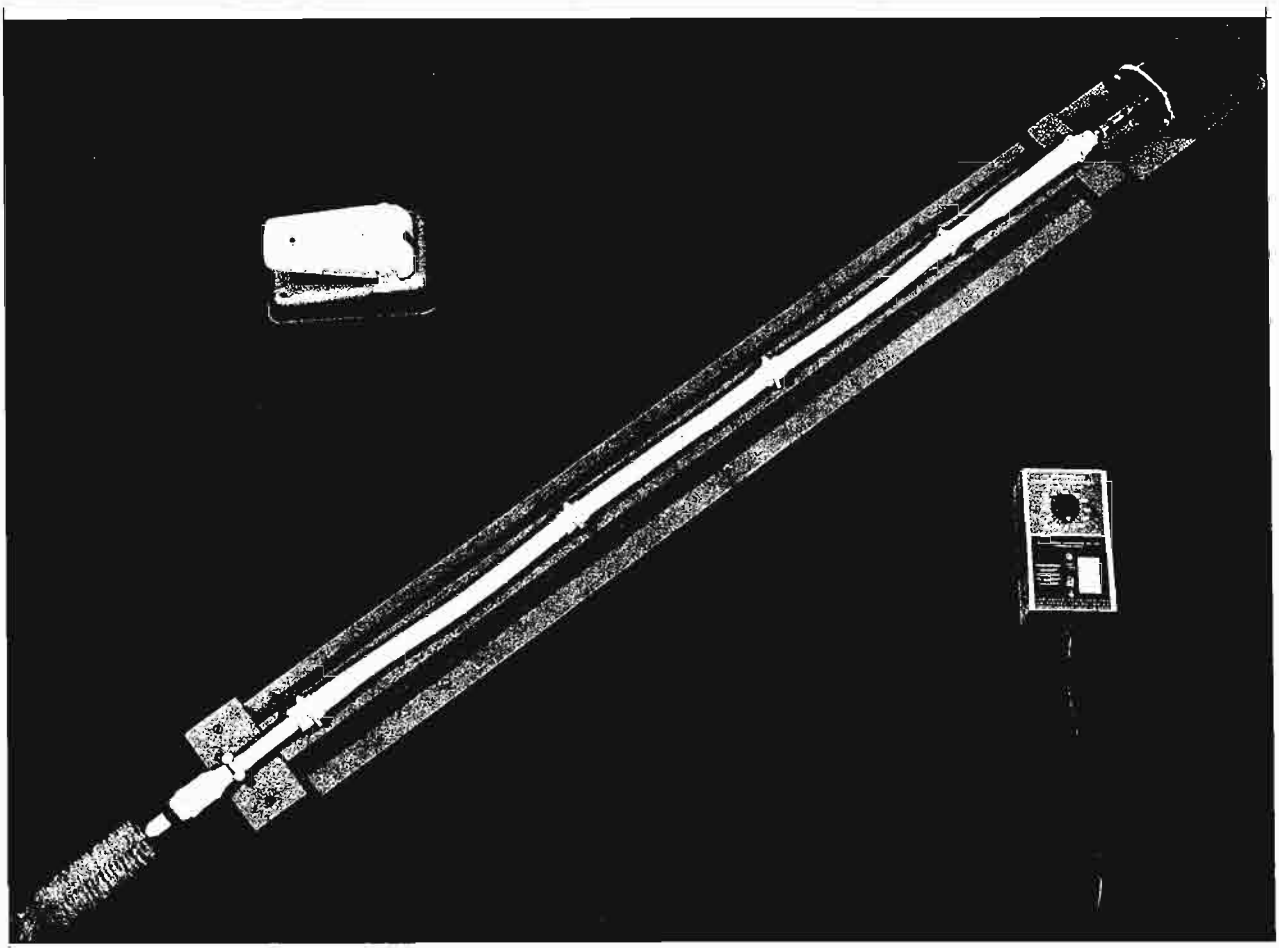
Turoczi, L. J. Rutgers University, Newark, New Jersey. A motorized brush for small cleaning needs.

Cleaning of glassware consumes considerable time in Drosophila laboratories. In order to facilitate the washing of shell vials and other small items, we have designed a motorized brush. The unit can be constructed easily and inexpensively and provides an efficient and rapid means of maintaining a clean glassware supply.

The apparatus (Fig. 1) is composed of a small motor connected by a drive shaft to a brush. The drive shaft consists of a flexible cable, measuring 36 inches long and having an adjustable chuck fitting. The shaft and motor are attached to a wooden base, with the brush overhanging the edge of the base.

Experience has shown that the best procedure for washing dirty vials is to soak them in a cleansing solution, such as "Haemo-Sol" (Meinecke Co., Baltimore, Md.), prior to brushing. The brush is allowed to rotate for a few seconds inside vials partially filled with the solution. Motor speed can be regulated either through a variable foot pedal or electric rheostat. Test-tube brushes of various sizes may be employed to clean vials of different dimensions. To shield the operator from splashing, a plastic deflector can be mounted on the base to partially cover the brush.

In our laboratory, the motorized brush finds an additional application because of considerable use of "dual purpose plastic stoppers" in studies in aging (Sondhi, K. C., 1965. Life Sciences, 4:57-61). These stoppers contain the Drosophila food medium and are fitted to shell vials. Since Drosophila populations are usually provided with fresh food medium daily, the stoppers require cleaning at frequent intervals for re-use. The cleaning procedure involved is readily handled with the motorized brush.



Kircher, Henry W., Kenneth G. Goodnight, Robert W. Jensen. University of Arizona, Tucson, Arizona. A medium for Drosophila that are difficult to rear in the laboratory.

Many species of Drosophila spend their larval stage in leaves that are rotting on the forest floor in the Hawaiian Islands (1). Most of these species cannot be maintained in the laboratory on normal banana media or even the enriched medium recently devised by Wheeler and Clayton (2). We had some success rearing these flies last summer in Hawaii with the following medium. It's presented here as a starting point for further nutritional experiments with species of Drosophila that are difficult to rear in the laboratory.

Ingredients:

1. Brewer's yeast, U.S.P., is stirred overnight three times with several volumes of 2:1 v/v chloroform-methanol to remove the lipid fraction (Note 1). Between extractions the yeast is filtered thoroughly with a vacuum on a Buchner funnel. The final product is dried in air and finally in an oven at 50°C until no trace of solvent remains.
2. Turtox brand agar, General Biological Supply House, Chicago, Ill. (Note 2).
3. Fructose, propionic acid.
4. Enzymatic hydrolysate of soy protein, Nutritional Biochemicals Co., Cleveland, Ohio (Note 3).
5. B-Vitamins, thymine and choline chloride.
6. β-Sitosterol, cholesterol and ergosterol.

Preparation of One Liter of Medium:

1. The extracted yeast (35g.) is ground thoroughly in a mortar with 0.5 g. of the sterol. A single sterol or a mixture of the three above can be used.
2. The following vitamins, thymine and choline chloride are dissolved (riboflavin dispersed) in 100 ml. water. The mixture is stirred magnetically before a 10 ml. aliquot is added to 1 liter of medium. It is conveniently kept in the refrigerator in a small flask containing a magnetic stirring bar.

For 10 liters of medium:

Thiamine	20 mg.
Nicotinic acid	120 mg.
Riboflavin	100 mg.
Calcium pantothenate	160 mg.
Biotin	4 mg.
Pyridoxine	25 mg.
Folic acid	30 mg.
Thymine	20 mg.
Choline chloride	600 mg.

3. One liter of water (Note 4) add 15 g. agar is brought to a boil. To the hot solution is added 10 g. fructose, 20 g. soy protein hydrolysate, 10 ml. of the vitamin suspension, 5 ml. propionic acid and 35 g. of the yeast-sterol mixture.
4. After the yeast has been thoroughly wetted and dispersed, the medium is autoclaved (20 min., 15 psig), and when cool, is poured into autoclaved stoppered shell vials. It can be used for axenic or xenic rearing of Drosophila.

Note. 1. The leaf-breeding Hawaiian Drosophila do not use yeasts in nature. The yeast sterols (mainly ergosterol) may inhibit the utilization of the necessary sterol (cholesterol or β-sitosterol). Other yeast lipids may also be disadvantageous to the flies.

Note 2. Erk and Sang (3) have reported that Difco Bacto-agar is toxic to certain species of Drosophila.

Note 3. Hagen (4) has shown that the oriental fruit fly, when grown axenically, does much better when a partially hydrolyzed protein is furnished the young larvae. We have also observed this in our work with D. pachea. With this species, enzymatically hydrolyzed casein was toxic.

Note 4. The replacement of water with a hot aqueous extract of the plant or the lipid extract of the plant in which the Drosophila larvae are found may be beneficial here to supply feeding or ovipositional stimulants.

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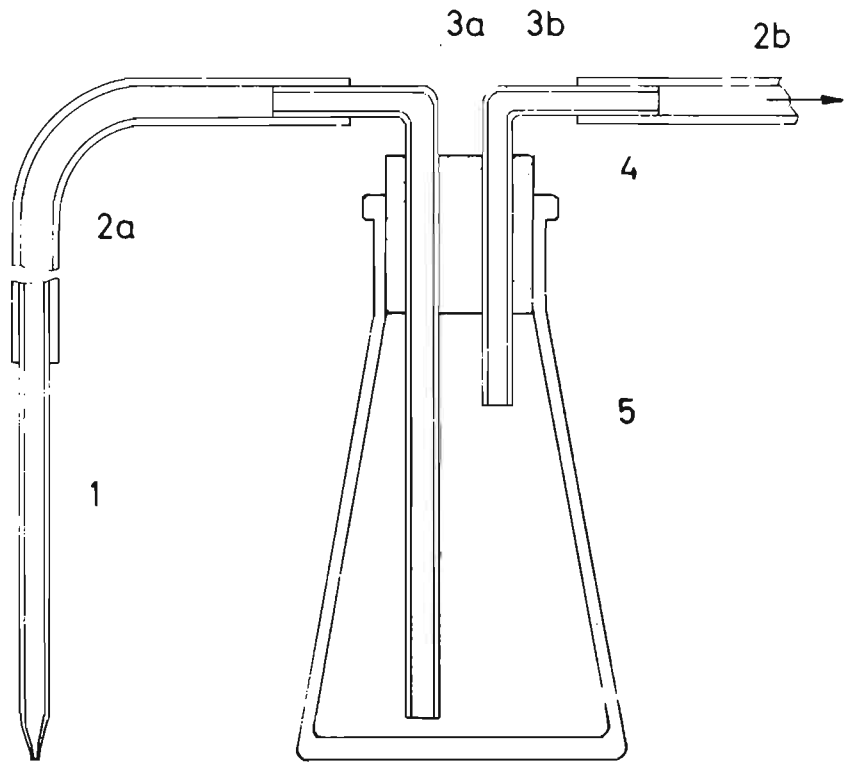
Milkman, Roger D. and Ned Feder. Syracuse University, New York, and N.I.H., Bethesda, Maryland. A water-soluble permanent mounting medium for salivary chromosomes.

To a 60% acetic acid solution, add 30% (w/v) PVP-40 (polyvinyl pyrrolidone, m.w. about 28,000). After fixation in 60% acetic acid for several minutes (for phase microscopy) or after fixation and staining, transfer gland to a drop of the medium on a slide, with forceps or dropper. Add cover slip and press, or hit with reflex hammer. Indeed, orcein and perhaps other stains can be incorporated directly into the medium, so that the gland may be introduced right after fixation. Rotation or sliding of cover slip often straightens out the chromosomes. Plasdone C is similar to PVP-40. Higher m.w. PVP has poor viscosity/shrinkage properties. Lower m.w. PVP, expected to be better, distorts chromosomes badly. We welcome information on use of polyvinyl alcohol (PVA), additional stains, opinions on concentration optima, and any other modifications. Preparations harden quickly, should last indefinitely. We developed this method at Harvard University, Cambridge, Mass.

Wind, Heinz. Institut für Strahlenbiologie, Münster (Westf.) Germany. Device for isolating part of flies out of a culture without disturbing the rest of the population.

A simple method to separate flies belonging to one sex from the other out of a culture bottle has been devised in our lab. The one sex to be discarded or to be isolated was sucked out of the culture bottle (with the help of a water suction pump) into the assembly shown in the Fig. The diameter of the drawnout tip of the glass tube (1) is to be such that only one fly could be sucked in at a time. If the separated flies are to be used again, the flask (5) is not filled with alcohol, instead a nylon net is fastened to the short glass tube (3b) to prevent the flies from getting sucked into the pump. Positions 2a and 2b are silicone tubings, position 4 a rubber stopper. The arrow shows the direction of the air stream.



Kaplan, Martin L. Queens College of C.U.N.Y., Flushing, New York. A simple method for collecting and surface-sterilizing eggs from D. melanogaster.

The collection and subsequent surface-sterilization of D. mel. eggs were facilitated by using black filter paper (S & S #551) and 0.007 inch mesh monel-metal wire cloth (obtained from G. A. Williams & Son Inc., 17 Murray St., N. Y. C.) respectively.

Sterilized, rectangular strips of filter paper (2-1/2" x 5/8") with parallel lines scored at 1/16" intervals along the length of the strip, were moistened with sterile distilled water and placed on the surface of yeasted food media prepared previously in aluminum foil dishes. Eggs were layed along the length of the furrows in the paper thereby permitting rapid counting and collection of relatively yeast-free eggs.

Cleansing and surface-sterilization of eggs was accomplished by placing the harvest into baskets (1/2 in. x 1/2 in. x 1/8 in.) fashioned from monel-metal wire cloth with 0.007 inch spacings. The mesh spaces were small enough to prevent the passage of eggs but large enough to permit newly-hatched larvae to escape onto the substratum. The egg-filled baskets were then transferred through 2 washes of sterile, distilled water, 2 washes of 70% ethanol and finally placed into 70% ethanol for 40 minutes. The baskets were rinsed briefly in sterile distilled water, placed on the surface of petri dishes containing 2% Agar, and incubated for 24 hours at 25$\pm$ 0.5°C.

After incubation the transparent agar plates were examined against a black background using a dissecting scope at 11x and newly-hatched larvae selected for further treatment. The use of agar plates facilitated counts of egg mortality since unhatched eggs in the baskets were clearly visible against the black background. Agar plates showing evidence of contamination were discarded.

In this laboratory, monel-metal baskets also have been used successfully to transport fixed larvae through a dehydration sequence of alcohols as well as paraplast infiltration.

Lewis, E. B. and F. Bacher. California Institute of Technology, Pasadena, California. Method of feeding ethyl methane sulfonate (EMS) to Drosophila males.

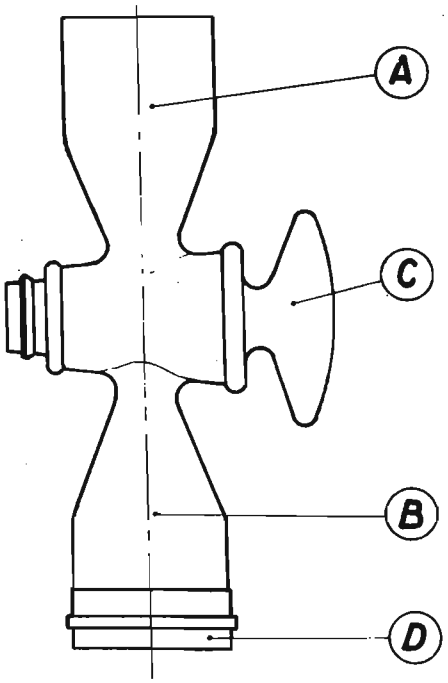
The alkylating agent, EMS, is remarkably mutagenic when fed to adult Drosophila males as first shown by T. Alderson (Nature 207: 164-169). A simple and effective feeding method is described below. In view of the potential hazard of EMS to human beings a description is also given of special precautions which we take in handling this chemical.

Adult males (collected 0 to 48 hours after emergence) are fed on Kleenex saturated with an 0.025 M solution of EMS in sterile 1% sucrose solution. To prepare 100 ml of this solution, 0.24 ml of EMS (Eastman Organic Chemical; or K and K Laboratories) is taken up in an 0.5 cc hypodermic syringe and injected into 100 ml of a sterile 1% solution of sucrose in distilled water. In order to achieve complete miscibility of EMS in the sucrose solution, the mixture is agitated by aspirating with a 10 cc disposable hypodermic syringe. A single Kleenex is crumpled and pressed to the bottom of a 1/2 pint culture bottle. Nine or ten ml of the 0.025 M EMS solution is taken up in the 10 cc syringe and injected directly onto the Kleenex until the paper is just saturated. Adult males (usually 50 to 100) are then added and the bottle is tightly stoppered with a large wad of cotton. It is important that the males have had time to recover completely from etherization (at least one hour). Males are fed on the EMS solution, usually for 24 hours at 25°C, after which they are shaken into a fresh culture bottle containing the standard food medium. They are then mated to females of suitable tester strains.

The above operations are carried out in a chemical hood. Hands are protected with disposable plastic gloves. Bottles and glassware contaminated with EMS are decontaminated by rinsing with a solution ("MA") containing 0.5% mercaptoacetic acid in 1 M NaOH or KOH. Cotton stoppers, Kleenex, syringes and gloves after decontamination with the MA solution, are discarded. (In these decontamination procedures the amount of the MA solution added should always be sufficient to give a higher concentration of mercaptoacetic acid than that of EMS.)

Kirschbaum, W. F. and Beatriz M. de Rey. Atomic Energy Commission, Buenos Aires, Argentina. Drosophila egg treatment chamber.

In order to reduce to a minimum the manipulation of immature eggs obtained with an egg-collecting device patterned after the "ovitron" of Yoon and Fox (Nature 206: 4987, 1965), we have designed a special chamber, which is proving very successful in avoiding egg loss and contamination in collecting embryos for tissue culture.



In the diagram, D is a metal filter that does not allow the passing of the eggs. A and B are open-ended identical chambers, and C is a simple stopcock.
To de-chorionate eggs, they are placed in A and the apparatus with stopcock open is immersed in hypochlorite solution, so that both chambers A and B are filled. An electromagnetic stirrer inside B is used for stirring. Dechorionated eggs remain floating in A while the chorions drop down to B. Stopcock C is then closed and the chamber is taken out of the solution. Filter D is removed and B emptied. A clean filter like D is placed on A. The chamber is turned upside down, placing A into position B of the figure. C is now opened to let air in and the hypochlorite solution out. Eggs are washed several times by immersing chamber A (now in position B) in different solutions. Finally, D is removed and turned upside down over an appropriate funnel. The eggs are washed into the homogenizer by pouring culture solution through the filter. All the elements are previously sterilized and the operations done in a sterile culture chamber.

Cooper, K. W. University of California, Riverside, California. Freeing Drosophila of Mold.

When using ordinary media (as those without propionic acid or special yeasts), there inevitably comes the day when valuable crosses become mold-ridden despite the presence of an inhibitor such as moldex. If flies from such cultures are transferred (preferably as soon as mold is detected) to fresh medium containing roughly 1%-2% of crystal violet (ca 1 gm per 100 cc), in most cases two passages of 12-18 hours each on this medium will effectively free the flies of mold. As yeast is killed or inhibited in the flies' digestive tracts as well, just as are most molds on the crystal violet medium, it is best to seed vials of fresh food just before final transfer of the flies from the crystal violet medium to them, although this is generally not necessary if the new medium contains as much as 3-4 gms of dried yeast per 100 cc.

Flies lay readily on the food containing crystal violet, and the eggs hatch normally. The larvae of course do not develop on the sterilizing medium. It is possible, therefore, to accumulate large numbers of sterile, viable first instar larvae none of which have been nourished. It is interesting, but not surprising, that an occasional vial of crystal violet medium, a week or ten days after removal of the now ordinarily mold-free flies, may itself develop slow growing mycelia which are resistant to the dye. For this reason it is inadvisable to reuse a vial of crystal violet medium after the passage of flies.

Wind, Heinz. Institut für Strahlenbiologie, Münster (Westf.) Germany. Some practical improvements for handling etherized Drosophila flies.

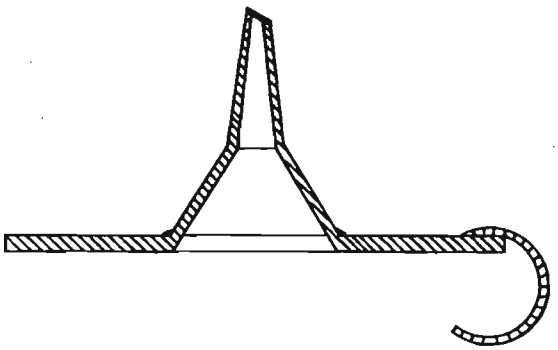


Fig. 1

1) For mating large numbers of flies, we have been using for several years a plexiglas plate, having in its center a hole (diameter about 40 mm), under which a polyethylene funnel is seamlessly fixed (Fig. 1). Two batches of etherized flies of either sex are put beside the hole, and with a brush one male and one female (or whatever combination) are thrown through the funnel into the vial.
2) A special plexiglas re-etherizer which proved to be helpful has been constructed by us (Fig. 2). Its inner diameter is about 35 mm, the height of the inner space being about 15 mm (6 mm for a cotton layer, 1 mm for a sheet of plexiglas, provided with many little holes, or a piece of a brass net, which is held in place by a plexiglas ring). The cotton layer is moistened by a few drops of ether through a hole in the cover of the re-etherizer, which is then closed with a plexiglas plug.
3) A nonbreakable, inexpensive etherizer was constructed of polyethylene (Fig. 3). In the screw cap of a 100 ml polyethylene bottle a hole was cut and the stem of a polyethylene funnel was forced through and fused with hot air to the screw cap. The bottom end of the funnel stem, in which a few holes were drilled for the entry of the ether vapours, was closed with a round polyethylene disc. To prevent the ether-moistened cotton layer from moving, a steel spring was inserted around the funnel stem. For use, the screw cap is removed, some drops of ether poured into the bottle and the cap is replaced.

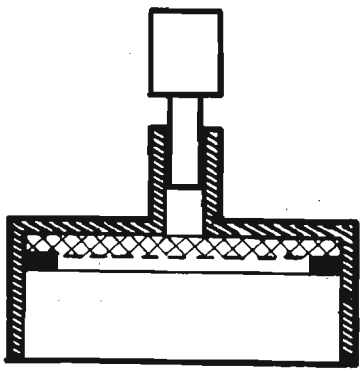


Fig. 2

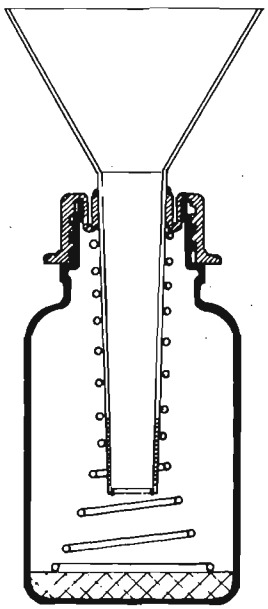


Fig. 3

Schouten, S. C. M. Genetisch Instituut der Rijksuniversiteit, Utrecht, The Netherlands. Storing standard medium in bottles for a longer period.

In order to avoid occasional peak quantities to be prepared we have tried to keep the standard cornmeal medium not provided with yeast suspension for several weeks (up to 3 months) in the store room 18°C. After preparing and pouring the food into the bottles, the bottles were capped with aluminum wrap and then sterilized for 20 min at 120°C. The bottles were then stored.

After the storage period it is only necessary to add the live yeast suspension and the bottles are ready for use.

The cultures grown on this stored medium do not differ from cultures grown on fresh-prepared food with respect to the number of offspring, generation, time, etc.

Thompson, C. F., Jr., and F. J. Gottlieb. University of Pittsburgh, Pittsburgh, Pennsylvania. A method for obtaining large numbers of accurately timed pupae.

The following method enables the collection of large numbers of pupae of very accurately known age. In order to do so, very heavy cultures of larvae are necessary. The food of David (D.I.S. 36:128) allows for extremely heavy cultures, particularly if a large number of parents are used and if several cc. of thick yeast suspension are added to the culture with the parents and again when the parents are removed several days before collection. As larvae prepare to pupate, they crawl on the side of the bottle if no dry paper is available to them. If five or six bottles are set up, larvae will pupate at a rate of up to two per minute for several days. Even though the cultures are very heavy the mature larvae are larger than those grown on normal corn-meal-molasses medium and they show no noticeable effects of crowding. At the beginning of collection, all pupae are removed or pushed down into the food from the side of the bottle. After an appropriate interval, pupae are collected from the bottles in the same order in which they were cleared of pupae, using a stiff moist brush such as Grumbacher's No.1271-F or 626-B. Thus the time of pupation of the collected pupae is the midpoint of the interval between collections plus or minus one half of the interval. With practice, collections can be done at a rate of one bottle a minute. The minimum interval between collections is a function of the number of bottles, i.e., six minutes for a six bottle series. In such a case, the order of the bottles must not be changed and an even rate of collection must be maintained so that the interval between collections from the same bottle is constant. The initiation of pupation is determined on the basis of three criteria: immobility, spiracle eversion and shape characteristic of pupae. Any individual which does not meet all three criteria is discarded. As they are collected, pupae are transferred to a small petri dish 5 cm. in diameter in which a moistened piece of filter paper has been placed. The petri dishes are sequentially numbered and all pupae collected within a minute interval are grouped together. Thus it is easy to determine the age of the groups of pupae by referring to the number of the petri dish. Occasionally, an animal which meets all three criteria will "revert" back to a larva and crawl away as a result of being transferred to the petri dish. It will frequently pupate later. To avoid including these animals, all pupae are placed on a small area marked in the center of the filterpaper. Any "revertants" will crawl off this area and pupate outside of it. Such a pupa is easily spotted and removed later. (Work supported by Public Health Service Research Grant GM11084, from the National Institute of General Medical Sciences.)

Thompson, C. F., Jr. University of Pittsburgh, Pittsburgh, Pa. A method for collecting accurately timed larvae.

The following method of obtaining accurately timed larvae, using the event of egg hatching as the base time, avoids many of the problems associated with techniques which use the time of egg collection as the base.

Eggs are collected by any convenient means. We prefer the method using Norite blackened, yeasted food on a paper milk bottle cap which fits the standard half pint milk bottles. After eggs have been laid, they are transferred under a dissecting microscope with a spear point probe, brush or fine jewelers' forceps and lined up on a small block (about a cm^2) of nutrient agar which rests on a square (about 2 cm^2) of black polyethylene tape folded over so that no stickly surface is exposed. The tape containing the agar block is placed in a petri dish containing the same nutrient agar, to insure a moist atmosphere, and the dish is covered. The eggs are observed periodically and as the larvae hatch they are collected for whatever use they may be required. The beginning of larval life is thus the midpoint of the interval since the previous collection of larvae from that block plus or minus one half of the length of that interval. The process, though seemingly laborious, is quite efficient as virtually every egg provides a timed specimen. If eggs are initially collected over a short period of time the majority will hatch within a subsequent two to three hour period although the entire range of hatching time may be quite wide. The black tape serves several purposes. It is a convenient means of handling the agar block and it provides a dark background against which the eggs and larvae are easily observed. If the tape is somewhat wider and longer than

the block, it provides a dry barrier which keeps the larvae on the block where they can easily be retrieved. Those few which do cross the barrier can usually be retrieved from the medium in the petri dish. The newly hatched larvae usually do not burrow for the first half hour after hatching and if they do, they can easily be seen in the semi transparent medium. All larvae are removed during each observation period and empty egg cases are also counted and removed to insure that all newly hatched larvae have been accounted for. It is possible by this method to watch several hundred eggs continuously and collect the larvae as they hatch. If a small interval between observations is permissible, many more blocks of eggs can be observed. The black tape cannot be replaced by cardboard or paper since these materials will curl when they come in contact with the moist agar. (Work supported by Public Health Service Research Grant GM11084, from the National Institute of General Medical Sciences.)

Corwin, H. O. and P. D. DeMarco. University of Pittsburgh, Pittsburgh, Pennsylvania. Technique for feeding chemical mutagens to adult Drosophila.

Adult male Drosophila do not survive multiple injections of chemical mutagens. In experiments where more than one mutagen is to be introduced into the adult fly at separate time intervals, feeding is necessary. The agent to be introduced must be dissolved in a medium that will stimulate fly feeding but will not induce chemical or physical alterations in the agent. The quantity and type of filter paper placed at the bottom of the shell vial, which acts as a reservoir for the mutagen solution, is also important. It must be sufficiently absorbent to remain moist, but it must not retain an excessive amount of solution or else the flies will drown. An additional problem was encountered during feeding experiments. Plugs had to be removed and replaced in the feeding vials every time new solution was added. Flies were often lost or squashed between the cotton plug and the side of the vial during the above process. The following method, adapted from the feeding technique introduced by Pelecanos and Alderson (DIS 37:116, 1963), is designed to solve the above problems.

Different concentrations of chemical mutagens were fed to Canton-S wild type males. The adult males had been aged 12-24 hours. The various concentrations of mutagens were dissolved in a 5% glucose solution. Groups of ten male flies were treated in 20 dram shell vials. The bottom of each vial was lined with three thicknesses of Whatman filter paper #3 cut to the diameter of the vials. A pasteur pipette, inserted through the cotton plug until it touched the filter paper, served as a permanent delivery tube for the different treatment solutions. This arrangement allowed for the addition of fresh solutions without the removal of the cotton plug. After the initial saturation of the filter paper, only five drops of solution every ten to twelve hours were required to keep the filter paper moist. A single agent was administered over a 48 hour period. When two agents were administered, the simple glucose medium sustained the male flies over the total 96 hour feeding period and no decrease in progeny production per male was noted following this treatment. The treated males were then tested for specific visible mutations induced at the dumpy locus. All agents fed by means of this above technique demonstrated mutation frequencies equal to or exceeding those frequencies obtained when these same agents were introduced by means of the injection technique.

Mittler, S. Northern Illinois University, DeKalb, Illinois. A rapid method for removing and discarding adult Drosophila.

For several years we have used with much success a small portable tank vacuum cleaner to remove and discard flies from bottles prior to collecting virgins. The open end of the bottle is tapped at a 45° angle on to a rubber mat about an inch from the end of the vacuum tube. The tube is not inserted into the bottle because air rushing in along the sides will cause some Drosophila to stick to the food. The cleaning attachment on the end of the flexible tube is not used, and of course, the opening is plugged after use.

Richardson, R. H. University of Texas, Austin, Texas. A safety interlock modification for the Heath IP-32 power supply

A number of laboratories are presently using the Heath IP-32 power supply for starch gel electrophoresis. The working voltages range from about 175v DC to 300v DC, with a current of 25ma to 100ma. These conditions are sufficiently dangerous to warrant precautions for preventing accidental shock.

This power supply has no indicator lights to distinguish a "standby" condition, with zero voltage across the output terminals, from the "on" condition, with up to 400 volts across the output terminals. Furthermore, the switch knob tends to loosen with use, so the switch position may be doubtful.

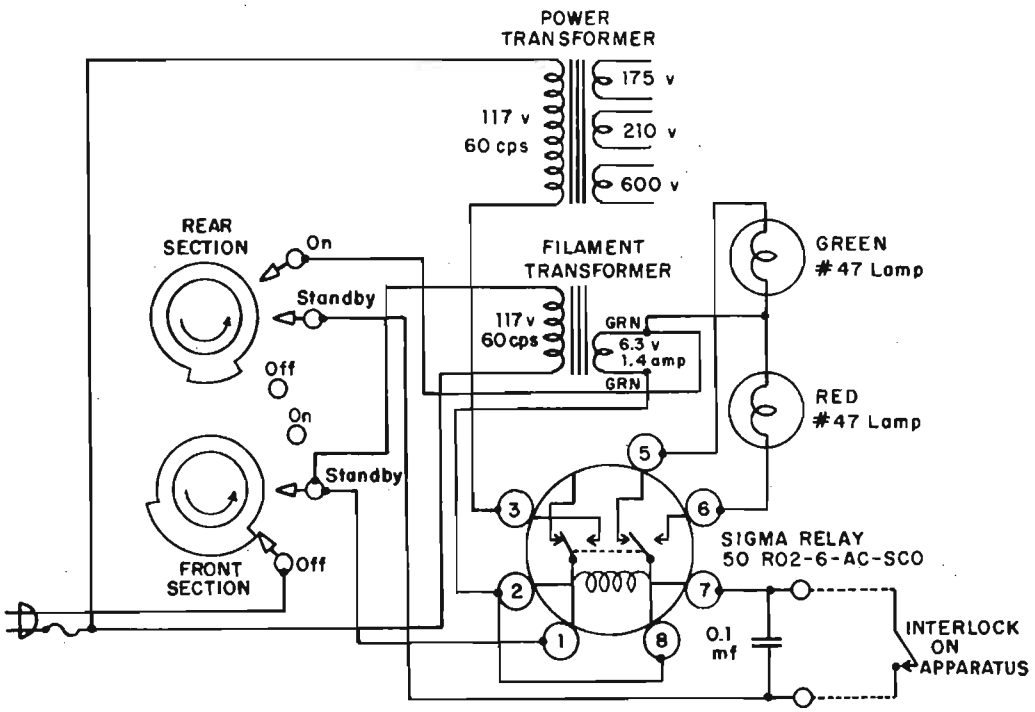


Figure 1. Schematic of modifications for Heath IP-32 power supply to incorporate interlock switching.

The power supply may easily be modified to eliminate the hazard at a cost of less than $20 for parts (see Figure 1). A power relay with an actuating voltage of 6v AC and a resistive current rating of 10 amps (for example, Sigma model 50 R02-6-AC-SCO) may be used to switch the high voltage transformer (power transformer) primary coil, instead of the rear section of the standby switch. The rear section of the standby switch is placed in series with the interlock switch at the gel, and with the relay coil. When all switches are closed, the relay is actuated.

The relay also switches on a green panel light (the one originally on the power supply front panel) when the standby condition exists, or alternatively switches on a red panel light (installed above the green one) when the on condition exists.

The jacks for the relay coil circuit are installed on the front panel above the "B+" and "Common" output jacks. Since the usual connectors to the high voltage jacks are banana plugs, the smaller pin jacks (yellow color coded) are installed for pin plug connections to the interlock switch. This prevents an accidental reversal of interlock and high voltage connections.

The interlock switch may be any of several kinds, but should be closed only when it is impossible to touch the gel, buffer solutions, etc. We have used plastic boxes to

hold the buffer containers, gel, connecting sponges, ice, etc. The interlock switch is closed when the lid of the plastic box is in place, and the switch is open at all other times.

This work was done with the support of the following grants: PHS Research Grant No. 11609 to W. S. Stone and M. R. Wheeler, PHS Training Grant No. 2 T1-GM-337-06 to R. P. Wagner, et al., and A.E.C. Contract No. AT-(40-1)-3681 to K. Kojima.

Richardson, R. H. University of Texas, Austin. A safety interlock for the Heath IP-17 power supply.

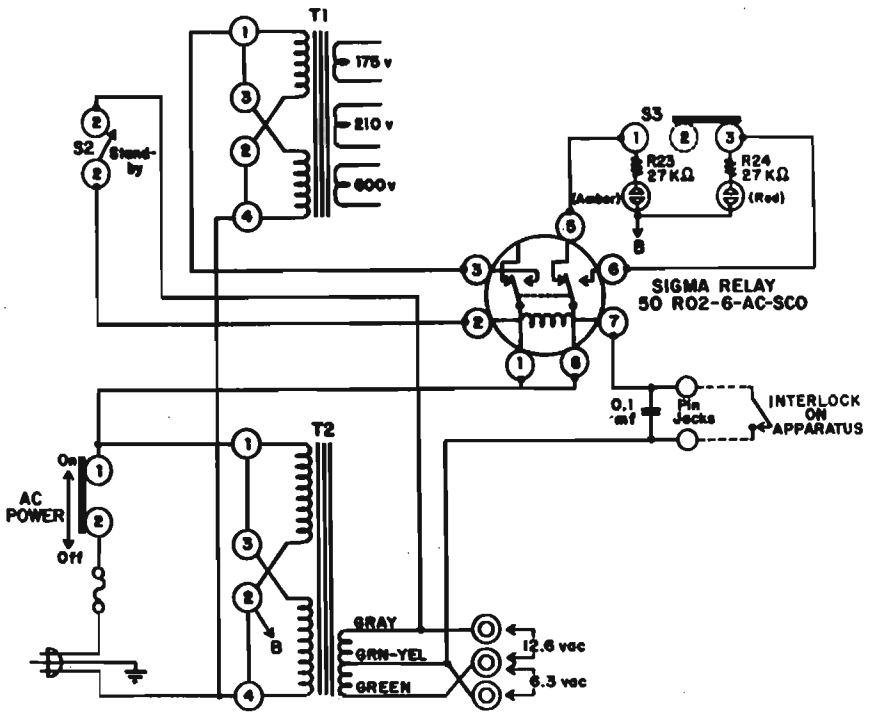
The Heath IP-17 power supply is the new model high voltage regulated power supply replacing the model IP-32. A number of peripheral circuit and cabinet modifications have been made, which result in changes from the IP-32 in details of incorporating a safety interlock. Basic operation of the interlock remains as previously given for the IP-32.

An important Heath change was separating the standby and the filament switches, and making them "positive" action without knobs to loosen. The S2 switch for the high voltage transformer is easily rewired in series with the relay coil and interlock switch.

Instead of adding a standby pilot light, the two lights supplied to indicate position of the meter switch may be rewired so that the red (labeled "B+ volts") indicates high voltage is available, while the amber (labeled "C-volts") may be relabeled "standby" and used to indicate absence of high voltage.

The only additions to the front panel of the IP-17 are two yellow tip jacks for connections of the interlock switches. The relay is mounted on the transformer chassis as near as possible to the top of the chassis between the two 6L6 tubes.

This work was done with the support of A.E.C. Contract No. AT-(40-1)-3681 to K. Kojima.



Schematic for incorporation of a safety interlock into the Heath IP-17 regulated DC Power Supply.

Gvosdev, V. A., and V. T. Kakpakov. Kurchatov's Institute of Atomic Energy, Moscow, U.S.S.R. The medium for cell culture of Drosophila melanogaster in vitro.

We have obtained growth of diploid embryonic cells of Drosophila melanogaster during 40 and more days in the C-12 medium of the following composition:

Constituents	Amount mg/100 ml	Constituents	Amount mg/100 ml
$NaH_2PO_4 \cdot 2H_2O$	50	Vitamin B_{12}	0.002
$NaHCO_3$	35	Vitamin A	0.002
KCl	256	Thiamine	0.002
$CaCl_2$	50	Riboflavin	0.002
$MgCl_2$	250	Ca-pantothenate	0.002
NaCl	290	Pyridoxine	0.002
Sucrose	500	p-aminobenzoic acid	0.002
Glucose	500	Folic acid	0.002
Malic acid	67	Biotin	0.002
Succinic acid	6	Inositol	0.002
Sodium acetate	2.5	Cholinchloride	0.002
Lactalbumin hydrolisate	1750	Tris (hydroxymethyl-aminomethane)	300
Ascorbic acid	10	Phenol red	1
Niacinamide	0.01	Streptomycin sulfate	100 units/ml
l-tryptophane	10	Penicillin sodium	100 units/ml
l-cysteine	2.5		
Glutathione	0.5	pH adjusted to 7.2 with 1 N KOH	
Nicotinamideadenine-dinucleotide	0.5		
Yeast extract	150	Osmotic pressure: Δ = 0.70°C	

The medium was supplemented with 15 per cent of bovine foetal serum. Results of observation on cell growth in this medium are communicated in the research note (43:142)

Gonzalez, F. W. and S. Abrahamson. University of Wisconsin, Madison, Wisconsin. Acti-dione, a yeast inhibitor facilitating egg counts.

Egg counts in dominant lethal studies become very laborious if a thick lawn of yeast present on the food surface obscures the eggs. D. R. Parker eliminates this problem by inhibiting yeast growth with mycostatin. Since our strain of yeast appears to be resistant to mycostatin, we tried several other yeast inhibitors. Following the suggestion of Professor Weisblum of Pharmacology, we found a 1 mg/ml solution of Acti-dione (cyclo-heximide) in water to be both a very effective yeast inhibitor and non-toxic to the developing eggs.

We allow females to lay eggs on petri dishes containing corn meal, molasses, agar, and dried brewers yeast. No living yeast is included in the food, but usually is carried to the media by flies. After 12 hours the females are transferred to fresh food units and the eggs laid on the food are counted. Immediately afterwards, the food surface is lightly sprayed with the Acti-dione solution. Thirty hours after the first count is performed, the unhatched eggs can be counted without difficulty.

Acti-dione is available from the Upjohn Company, Kalamazoo, Michigan.

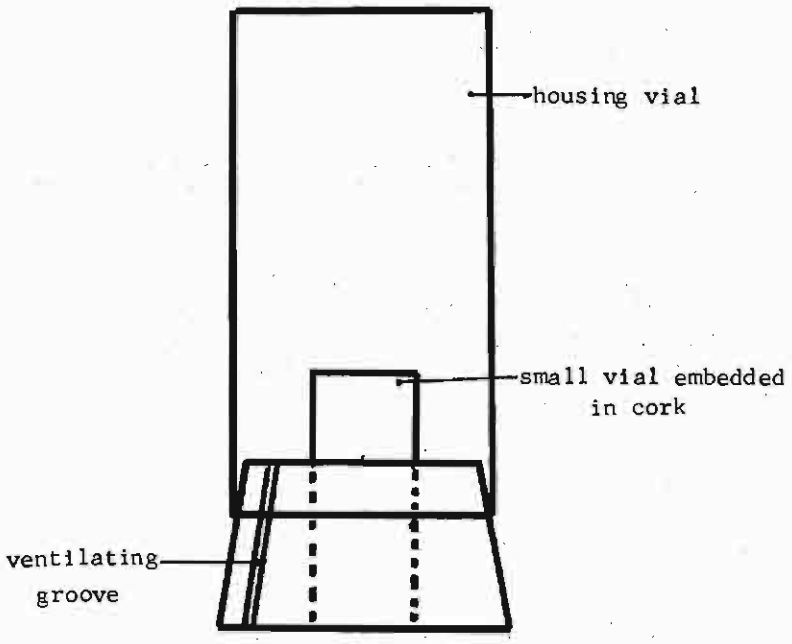


FIGURE I

Cook, R. M. and K. J. Connolly.
Department of Psychology, Sheffield University, England.

A simple method for individual egg counts in Drosophila.

A method has been devised which has the following merits: (1) individual females are used (2) repeated measures on successive days can easily be made (3) large numbers of flies may be handled.

Figure 1 illustrates the egg laying and living chamber for each female. The housing vial is 5 cm x 2.5 cm and fitted with a cork. The cork is bored out to house a smaller vial 2.5 cm x 1.5 cm which is filled with yeasted medium. A groove is cut in the cork, as illustrated, to ventilate the assembly. Egg counts are made by removing the cork and smaller vial, a bung being temporarily inserted.

After each days count the eggs are removed and the vial 'topped up' with yeasted medium. Smaller time intervals for egg counting may be employed with little disturbance to the flies.

MATERIALS REQUESTED OR AVAILABLE

Elof Axel Carlson (Zoology, U.C.L.A., Los Angeles, California 90024):

In November of 1966 I arranged with H. J. Muller to begin work on a biography of his scientific and social contributions. Plans for extensive interviews were made but Muller's illness prevented their completion. With his consent, however, I obtained some 300 handwritten letters that Edgar Altenburg had received from Muller during the period 1920-1940. I also interviewed colleagues and students who knew him during his Texas days. After Muller's death in April 1967, his widow, Dorothea, encouraged the biographical project and I arranged to survey the papers which Muller had kept in his laboratory. The library and papers are now in the Lilly Rare Book Collection at Indiana University. The donated collection includes some 30,000 letters, mostly from 1945-1967; about 500 books on radiation genetics, Drosophila genetics, and the Lysenko controversy; about 100 notebooks; and about 100 handwritten or typescript drafts of papers, mostly unpublished, including 200 pages of a monograph on Drosophila written about 1929.

The most serious weakness of the collection involves correspondence from 1910-1920 (Columbia and Rice), 1920-32 (Texas), 1933 (Germany), 1934-37 (USSR), and 1937-8 (Spain). There are sufficient papers from Edinburgh (1938-40) and Amherst)(1940-44) to round out his activities there.

I would very much appreciate xerox copies of letters to and from Muller in those periods. The correspondence from 1945 on is so complete that only handwritten letters would be lacking in the Lilly Collection.

If you have any special viewpoints, recollections, anecdotes, or suggestions which you think would be valuable for this project I would appreciate receiving that information, also.

Raymond A. Gerdes, Biology Department, Texas Woman's University, Denton, Texas 76204, would appreciate genetic reprints to build a presently non-existent department library.

Costas D. Kastritsis, Department of Anatomy, Southwestern Medical School, Dallas, Texas 75235, would appreciate receiving stocks from any of the following species: guarani, testacea, quinaria, macroptera, calloptera, rubrifrons and pallidipennis.

Henrietta G. Kalicki, Department of Biology, Adelphi University, Garden City, L. I., New York 11530, would like to receive reprints of physiological, histochemical and cytochemical studies of Drosophila.

Ross MacIntyre, Stephen O'Brien and Bruce Wallace, Cornell University, would like egg or adult samples from any experimental population of D. melanogaster which has been maintained for at least five years. We would like to analyze the extent of enzyme polymorphisms in isolated populations, and would welcome collaboration in this investigation. It is requested, however, that we be contacted by phone or letter beforehand so the flies can be immediately analyzed after their arrival.

Ilse Schwinck, Biological Sciences Group, University of Connecticut, Box U-42, Storrs, Conn. 06268, would like to receive information and/or stocks of Drosophila melanogaster mutants with partial drosopterin synthesis in the eyes. We are especially interested in new mutants not listed in Bridges and Brehme.

John Neeley, Department of Biology, University of Portland, 5000 North Willamette Blvd., Portland, Oregon, 97203, would appreciate being contacted if anyone in the course of his research has noted any of his stocks yielding more metafemales than he thought reasonable. This request is in connection with his doctoral thesis where he is attempting to analyse various genetic and environmental effects upon metafemale viability. To this end, he is looking for various stocks of Drosophila melanogaster that produce relatively high numbers (in excess of 2%) of metafemales.

ANNOUCEMENTS

A mutation of ophtalmopedia-type was found in laboratory stock of D. melanogaster. At present the mutation is under investigation, but we suffer from the shortage of necessary stocks for allelism test. Therefore, any information and especially stocks of ophtalmopedia and/or like mutations should be greatly appreciated. Information or stocks should be sent to: E. K. Ginter, Dept. General Radiobiology, Genetics., Inst. Med. Radiol., Obninsk, Kaluga District, USSR.

Marshall R. Wheeler, Department of Zoology, University of Texas at Austin, Austin, Texas 78712, reports that his Drosophila collecting net (described in DIS 29:180) is now available from General Biologial Supply House, Chicago. ("Turtox-Wheeler Insect Net", catalog no. 105A15). The plastic vial which is supplied, however, is too fragile, and negotiations are underway with a plastics manufacturer to produce the item in quantity. Individuals wishing to know more about the availability of the plastic vials shoudl write Dr. Wheeler.

Audrey M. Aubele is returning March 1, 1968 to The Ohio State University, Columbus, Ohio after spending one year (January 1967 to January 1968) as an NIH postdoctoral fellow at the Zoologisches Institut of the University of Zurich, Switzerland. She will hold a joint appointment as Assistant Professor in the Department of Pediatrics of the College of Medicine and the Department of Zoology and Entomology of the College of Biological Sciences where she will continue teaching and research in developmental and physiological genetics.

Henretta T. Band, formerly research associate in the Dept. of Zoology, Michigan State University, has moved to the Dept. of Natural Science, Michigan State University, East Lansing, where she is now an Assistant Professor.

R. C. Baumiller was appointed on July 1, 1967 Assistant Professor in Gynecology and Obstetrics and also Assistant Professor in Biology at Georgetown University, Washington, D.C.

Elof Axel Carlson has accepted a Professorship in the Biology Department, State University of New York, Stony Brook, New York, 11790, effective July 1, 1968.

Frank W. Gonzalez, currently at the University of Wisconsin, will move to the Medical Department, Brookhaven National Laboratory on January 1st, 1968.

Joseph Grossfield has joined the Department of Biological Sciences at Purdue University as an Assistant Professor.

G. D. Hanks is spending his sabbatical year at the University of Utah.

Eleanor Markowitz (formerly Himoe) has left the Department of Zoology, University of Wisconsin and is a National Cancer Institute Postdoctoral Fellow in the Department of Zoology, University of Iowa, Iowa City.

Terumi Mukai, formerly Research Member at the National Institute of Genetics, Mishima, Japan, has joined the Department of Genetics, North Carolina State University at Raleigh as Associate Professor. He is continuing his research work concerning genetic loads of Drosophila populations.

Toshio Shiomi, formerly of the Research Institute for Nuclear Medicine and Biology, Hiroshima University, had joined the Nagasaki University School of Medicine, Nagasaki, as Professor and Head of its new Department of Genetics.

J. I. Valencia will be a visiting investigator in the laboratory of W. Plaut, Zoology Department, University of Wisconsin, beginning December 1, 1967.

R. M. Valencia will be on leave from the Argentine Atomic Energy Commission to conduct research in the laboratory of S. Abrahamson, Zoology Department, University of Wisconsin, beginning December 1, 1967.

Isao Yoshikawa, formerly of the Division of Genetics, National Institute of Radiological Sciences, Chiba, has moved to the Department of Genetics, Nagasaki University School of Medicine, Nagasaki.

The John Innes Institute, Colney Lane, Norwich 70 F, England, has moved from Hertford to form an association with the University of East Anglia. The move took place mainly during the spring months and naturally much of the experimental work was disrupted, particularly the work on plants. The Institute is now functional again and new appointments should soon bring the depleted staff back to its full complement.

PGONE

A group, affectionately referred to as PGONE (population geneticists of New England) has been meeting over the last year and a half in various schools in the New England area. Anyone interested in information about future meetings should contact David Weisbrot, secretary.

A splinter group, headed by Francisco Ayala, Rockefeller University has been organized in New York City and Dr. Ayala will be glad to forward information about the group to interested persons.

GEOGRAPHICAL DIRECTORY

ARGENTINA

Buenos Aires: Atomic Energy Commission, Dept. of Radiobiology
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Kirschbaum, Werner F. B.Sc. Agr. Research Associate. Salivary cytology.
Mansilla, Purificación (Mrs.) Technical Assistant
Marinic, Susana E. (Mrs.) Research Assistant
Mazar Barnett, Beatriz (Mrs.) Doctora en Ciencias Naturales. Chemical induction of mutations.
Munoz, Enzo R. M.D. Radiation genetics
Paz, Carmen (Miss) Research Assistant Curator of stocks
Pereyra, Edith (Miss) Research Assistant
Spirito, Sonia (Miss) Undergraduate student research fellow
Valencia, Ruby M. (Mrs.) Ph.D. Chief of Genetics Division. Radiation genetics. (on leave during 1968 at University of Wisconsin)
Valenica, J. I. Professor. Radiation cytogenetics

AUSTRALIA

Adelaide, South Australia: Flinders University of South Australia, Bedford Park, S. A.
The School of Biological Sciences

Boettcher, B. B.Sc., Ph.D. Lecturer. Population genetics and immunogenetics
Brink, N. G., B.Sc. Ph.D. Lecturer. Mutagenesis
Clark, A. M. M.Sc. Ph.D. Professor and Chairman. Mutagenesis
Clark, E. G. (Mrs.) Research assistant
Hynes, M. B. Ag. Sc., research student
Lloyd, B. (Miss) Senior technician
Pateman, J. A., B.Sc., Ph.D. Professor. Biochemical genetics
Tancock, R. (Miss) Laboratory assistant
Williams, P. (Miss) Laboratory assistant and Curator of stocks.

Adelaide, South Australia: University of Adelaide, Dept. of Genetics

Hayman, D. L. Ph.D. Lecturer. Recombination, melanogaster
Levy, Anne (Miss) M.Sc. Demonstrator, Population studies, melanogaster
Mayo, M. Jean. (Mrs.) Ph.D. Lecturer

Brisbane, Queensland: University of Queensland, Dept. of Zoology, Genetics Laboratory

Angus, D., Ph.D. Research Associate.
Baimai, V., B. Sc. (Hons) Graduate student
Bock, I. R. B.Sc. (Hons) Graduate student
Plowman, Catherine Research Assistant
Mather, Wharton B. Ph.D. Head of Laboratory. Chromosomal polymorphism, isolating mechanism, Speciation.

Bundoora, Victoria, 3083: La Trobe University, Dept. of Genetics
Tel. 478-2722

Arnold, J.T.A. B.Sc. (Agr.) Graduate student. Behavior and population genetics.
MacBean, I. T. B.Sc. Demonstrator. Behaviour genetics. Selection experiments.
Parsons, P. A. Ph.D. Behaviour, population and human genetics. Selection experiments.

Canberra City, Australia: Commonwealth Scientific and Industrial Research Organization, Division of Plant Industry.

Childress, D. (Miss) Graduate Student. Fulbright Fellow 1967-1968. Chromosome Behavior
Faulkner, K. (Miss) Technical Assistant. Chromosome Behavior
Latter, B. D.H. Ph.D. Quantitative Genetics
MacKenzie, I. (Mrs.) Technical Assistant. Quantitative Genetics
Malecky, H. (Mrs.) Technical Assistant. Quantitative Genetics
Myszewski, M. Ph.D. Chromosome Behavior (at Drake University, Des Moines, Iowa, after Sept. 1968.)
Novitski, E. Ph.D. Chromosome Behavior. (on Sabbatical leave from Eugene, Oregon, 1967-1968)
Pasztor, L. M. Ph.D. Chromosome Behavior.
Peacock, W. J. Ph.D. Chromosome Behavior
Scowcroft, W. R. Ph.D. Quantitative Genetics
Taylor, W. Graduate Student. Chromosome Behavior.
Whitten, M. J. Ph.D. Quantitative and developmental genetics.

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Carrick, M. C. B.Sc. (Agr.) Research Assistant. Population genetics
Cummins, L. J. Research Student. Quantitative genetics
Hollingdale, B. J. (Miss) B.Sc. (Agr.) Research Student. Quantitative genetics
Rathie, K. A. B.Sc. (Agr.) Research Student. Quantitative genetics

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Baker, E. P. Ph.D. Senior Lecturer. Cytogenetics
McKinley, A. B.Sc. (Agr.) Teaching Fellow.

Sydney, N.S.W.: Commonwealth Scientific and Industrial Research Organization, Div. of Animal Genetics.
Tel. No. 88-0221

Bong Kug Ohh M.S. (Seoul) M.S. (Minn.) Research Student. Canalization, animal breeding
Finlay, D. B.Sc. (Agr.) Canalization
Rendel, J. M. Ph.D. (Chief) Population genetics, canalization
Sheldon, Ph.D. Quantitative genetics, canalization, mutation studies.

AUSTRIA

Vienna, University of Vienna, Dept. Institut für allgemeine Biologie
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Feuerbach, Heidemarie (Miss) Ph.D. Population genetics
Karlik, Anna (Miss) Ph.D. Population genetics
Kunze-Mühl, Elfriede (Mrs.) Ph.D. Cytogenetics
Mainx, Felix Ph.D. M.D. Professor, Head of the Dept.
Ruderer-Doschek, Elfriede (Mrs.) Ph.D. Megaselia genetics
Sperlich, Diether Ph.D. Population genetics
Springer, Robert Ph.D. Subobscura, sexual behaviour

BELGIUM

Heverlee-Louvain: Agricultural Institute of the University, F.A. Janssens Memorial Laboratory for Genetics.

Adriansens, J. Student. Cross-over
Berckmans, L. (Miss) Secretary
Courtin, Fr. Student. In vitro culture of embryonic cells
Delcour, J. Aspirant au F.N.R.S. In vitro culture of embryonic cells
Heuts M. J. Professor. Genetics; population genetics
Lints, C. V. (Mrs.) Research Assistant
Lints, F. A. Ph.D. Premier Assistant U.C.L. Physiological genetics
Ringelé, M. M. Technician
Stiers, R. O. Curator of Stocks
Tsien, H. C. B.Sc. Biochemical genetics
Verlinden, J. M. Licentiaat in Plantkunde. Age effects
Vloeberghs, J. V. Research Assistant
Wattiaux, J. M. Ph.D. Chargé de Recherches au F.N.R.S. Age effects.

BRAZIL

Saõ Jose do Rio Preto, Saõ Paulo: Governo do Estado de Saõ Paulo, Faculdade de Filosofia, Ciencias e Letras, Departamento de Biologia Geral, Tel. No. 2741 and 2179

Bicudo, H. E. Melara de C. (Mrs.) Ph.D. Assistant Professor. Cytogenetics and speciation.
Gallo, A. J. Assistant Professor. Genetic Load in Drosophila melanogaster. Taxonomy.
Mourao, C. A. Ph.D. Professor. Head of Department. Ecology and Taxonomy.
Silva, Elza (Miss) Laboratory Technician

Pôrto Alegre: Universidade do Rio Grande do Sul, Departamento de Genética, Caixa Postal 1953

Almeida, A.C. Undergraduate Student. Fellow of UFRGS C. of Res. Molecular Biophysics, Hemoglobins.

Araujo, A. M. Undergraduate Student. Fellow of UFRGS C. of Res. Human Genetics. Demography and congenital malformations.

Benevides Filho, F.R.S. Dent. Research Assistant. Visitor, University of Rio Grande do Norte. Fellow of CAPES. Human Genetics

Benevides, Maria José Dent. Research Assistant. Visitor. Fellow of Brazil C. of Res. (CNP_q) Human Genetics

de Castro, Ignez V. Sc. Lic. Fellow of Brazil C. of Res. (CNP_q). Human Genetics. Haptoglobins, G6PD.

Cordeiro, A. R. Ph.D. Associate Professor. Head of the Animal Genetics, Division of the Dept. Chromosome polymorphism, cytogenetics, Natural irradiated populations.

Contel, Eucléia P. B. Undergraduate Student. Fellow of Brazil C. of Res. (CNP_q). Animal Genetics. Enzymes in Drosophila.

Desmazeau, B.E. Biochem. Fellow of French Technical Cooperation. Visitor. Molecular Biophysics. Vertebrate hemoglobins.

Fernandes, Maria Irene B.M. Sc.Lic. Research Assistant. Head of the Vegetal Genetics, Division of the Dept. Cytogenetics of Paspalum.

Lewgoy, F. Chem. Assistant Professor. Molecular Biology and chemistry.

Ludwig, Maria R. Technician. Animal Genetics

Ludwig, Nilda Conceicao. Technician. Human Genetics

Machado, Dinarcy M. Administration Assistant

Mallmann, Maria Clara Sc. Lic. Fellow of UFRGS C. of Res. Human Genetics. Human cytogenetics.

Marques, E. K. Sc. Lic. Assistant Professor. Animal Genetics. Radiation Genetics of populations. Radioresistence.

Mendez, Heiri Undergraduate student. Fellow of Brazil C. of Res. (CNP_q). Molecular Biophysics. Vertebrates hemoglobins.

Morales, Nena B. Technician. Animal Genetics. Cytogenetics of Drosophila willistoni and D. paulistorum.

Newmann, D. Technician. Animal Genetics

Palatinik, M. Ph.D. Associated Research. Visitor. University of La Plata. Fellow of the Nac. C. of Res. (Argentina). Human Genetics. Population Genetics, human and animal blood groups.

Peres, A. P. Technician. Human Genetics

Possani, L. P. Sc.Lic. Research Assistant. Fellow of the UFRGS C. of Res. Molecular Biophysics. Abnormal hemoglobins.

Ramila, D. Technician. Foodmaker. Animal Genetics.

Reguly, Maria Luiza Bc.Sc.Lic. Research Assistant. Fellow of Brazil C. of Res.(CNP_q) Animal Genetics. Enzymes in Drosophila.

Reis, D. Technician. Animal Genetics

Reischl, E. Undergraduate Student. Fellow of Brazil C. of Res. (CNP_q). Molecular Biophysics. Vertebrates hemoglobins.

Rocha, F. J. Bc.Sc.Lic. Research Assistant. Human Genetics. Primitive populations genetics.

Rocha, Zita Marly A. Technician. Biophysics.

Sá, F.A. Technician. Human Genetics.

Salzano, F. M. Ph.D. Associate Professor. Head of the Dept. Head of Human Genetics, Division of the Dept. Human blood groups. Indian population genetics.

Simoes, C. V. Technician. Biophysics.

Simoes, G. V. Technician. Field work. Human Genetics

Simoes, Neiva Technician. Vegetal Genetics

Sune, Margarete V. Sc.Lic. Instructor Assistant. Human Genetics. Human cytogenetics.

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Vianna, Francisca M.M. Lic.Sc. Fellow of CAPES. Vegetal Genetics. Cytogenetics and Embriology of Paspalum.
Winge, Helga B.Sc. Lic. Research Assistant. Animal Genetics. Relations in the sibling group willistoni. Cytogenetics in the sibling group willistoni.
Xavier, Juracy Technician. Animal Genetics
Zanete, V.A. B.Sc.Lic. Fellow of Brazil C. of Res. (CNP_q). Animal Genetics. Chromosomal polymorphism and Radiation.

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Cordeiro, Antonio R. Professor. Cytogenetics Population Genetics.
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Morales, Nena Basilio Technician
Napp, Marly Assistant Disruptive selection
Ramila, Danubio Technician
Reguly, Maria Luiza Assistant Molecular polymorphism.
Veiga Neto, Alfredo J. Fellow. Radiation effects on population.
Winge, Helga Assistant Professor. Speciation.

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Bortolozzi, J. Post-Graduate student. Population genetics.
Brito Da Cunha, A. Ph.D. Associate Professor. Population genetics. Diseases with chromosomal effects.
Franca, Z.M. (Mrs.) Post-Graduate student. Analysis of recombination in Drosophila.
Magalhaes, L. E. de. Ph.D. Assistant Professor. Population genetics.
Pavan, C., Ph.D. Head of Department. Cytogenetics of Rhynchosciara; Drosophila infections with micro-organisms.
Santos, E. P. dos B.Sc. Instructor. Population dynamics.
Sene, F. M. Post-Graduate student. Population genetics
Souza, H.M.L. de (Mrs.) B.Sc. Instructor. Population genetics.
Targa, H. J. B.Sc. Instructor. Transplantation of gonads.
Tedeschi, M. V. (Mrs.) Post-Graduate student. Population genetics.
Toledo, J.S. de (Mrs.) B.Sc. Instructor. Speciation and evolution.
Zanardi, S. M. M. (Mrs.) Post Graduate student. Population genetics.

CANADA

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Bell, John B. B.A. Graduate Student
Dimsdale, Christine H. B.Sc. (Hon.) Curator of Laboratory Stocks
Hash, David Ph.D. Assistant Professor. Autoradiography of polytene chromosomes. Nutritional requirements of lethal mutants.

Nath, Edward J. M.Sc. Graduate student
Ping-Ting, Keh Ph.D. Research Fellow. Effect of Selenium substituted compound on crossing-over.
Vyse, Ernest R. M.Sc. Graduate student
Walker, George R.W. Ph.D. Professor. Effects of physiological treatments on crossing-over.
Woloshyn, Effie P. B.Sc. Graduate Assistant

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Chan, V. L. B.Sc. Graduate student. Biochemical genetics
Davis, Gale Ph.D. Assistant Professor. Chromosome behavior and developmental genetics
Keenleyside, M. H.A., Ph.D. Associate Professor Behavior
Misra, R. K. Ph.D. Assistant Professor. Biometrical genetics
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Ayles, Burton B.Sc. Graduate student
Bahng, Kyr Wahng M.Sc. Graduate student
Baldwin, Madeleine (Miss) M.Sc. Graduate student
Cameron, Jill (Miss) Honors undergraduate student
Erasmus, Udo B.Sc. Graduate student
Hayashi, Susi (Miss) B.Sc. Graduate student
Holden, Jeanette (Miss) Honors undergraduate student
Korinek, Ella (Mrs.) Technical help
Piternick, Leonie K. (Mrs.) Ph.D. Research Associate
Pratt, Rachel (Miss) B.Sc. Research Assistant
Procunier, Douglas, B.Sc. Graduate student
Shaw, Lorne Honors undergraduate student
Suzuki, David T. Ph.D. Associate Professor of Zoology
Tarasoff, Mary (Mrs.) B.Sc. Graduate student
Yang, Katherine (Mrs.) M.Sc. Research Assistant

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Landa, Z. Dr. Research worker. Chemical mutagenesis.
Ondrej M. Dr. Research worker. Chemical mutagenesis.
Prokopová, A. (Mrs.) Dr. Post-graduate student. Chemical mutagenesis
Grunwaldová, E. (Miss) Technician

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Šrám, Radim M.D. Chemical mutagenesis
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Zudová, Zdena (Miss) Student
Benes, V. Dr. Chemical mutagenesis
Kresslová, Zdenka Technician

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Simonsen, V. (Mrs.) Assistant. Biochemical markers
Prout, Timothy Visiting Professor. Population genetics.

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Lakovaara, Seppo Ph.D. Assistant Teacher. Developmental genetics, Population genetics.
Lokki, Juhani B.A. Research Associate. Biostatistics.
Sorsa, Marja (Mrs.) Ph.Lic. Assistant Teacher. Electron microscopy of chromosomes.
Suomalainen, Esko Ph.D. Professor. Head of Department.
Tigerstedt, Peter M.A. Research Associate. Biostatistics.
Tiivola, Airi (Mrs.) Technical Assistant. Curator of Stocks

Turku: University of Turku, Department of Genetics

Arajärvi, Pirkko (Mrs.) Cand. Nat. Sc. Research Assistant. Melanogaster: mutations, salivaries.
Hannah-Alava, Aloha (Mrs.) Ph.D. Research Associate. Melanogaster; developmental genetics, mutations.
Oksala, T.A., Ph.D. Professor, Head of Department. Melanogaster: mechanism of segregation, interchromosomal effects.
Portin, P., Mag.Phil. Melanogaster: mechanism of segregation.
Puro, J., Ph.D. Assistant teacher. Melanogaster: mutations.
Savolainen, Salme (Mrs.) Technical Assistant
Savontaus, Marja-Liisa (Mrs.) Mag. Phil. Melanogaster: mechanism of segregation.

FRANCE

Clermont-Ferrand: LFaculté des Sciences, Laboratoirè de Zoologie, 1 Ave. Vercingétoux
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Perrin-Waldemer, Cl. Assistant. Spermatogenesis and Chemosterilants of Drosophila.

Lyon, Villeurbanne: Université de Lyon, Faculté des Sciences, Section de Biologie Générale et Appliquée, Biologie Cellulaire, 43, Boulevard du 11 Novembre 1918.
Tel. No. 52.82.57

Bigonnet C. (Miss) Etudiante DEA. Ovogenesis in Drosophila
Daillie J. Maitre-Assistant. Nucleic Acid Metabolism
Fourche J. Maitre-Assistant. Respiratory metabolism in Drosophila. Ecdysone action on larva.
Gillot, S. (Mrs.) Maitre-Assistant. Ovogenesis in insects.
Mourgues C. (Mrs.) Assistant. Heretability in Drosophila.
Nigon V. Professor, Head of Department. Nucleic Acid Metabolism.
Sillans D. Assistant. Anesthesis in Drosophila

Lyon: Faculté des Sciences, Laboratoire d'Entomologie expérimentale et de Génétique, 16 quai Claude Bernard.

Bouletreau, M. Assistant. Biology of Pteromalus
Clavel, M. F. (Miss) Technician. Temperature effects in Drosophila
David, J. Maitre de Conférences. Quantitative inheritance and nutrition in Drosophila
Merle, J. (Miss) Stagiaire de Recherches. Physiology of Drosophila female
Rougier, M. Attaché de Recherches. Physiology of the dorsal vessel of Cybister
Van Herrewege, C. Graduate student. Nutrition of Blattella

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Strasbourg: Faculté des Sciences, Laboratoire de Zoologie

Sigot, André Professor sans chaire. CO_2 sensitivity in Drosophila

GERMANY

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Freye, Renate (Miss) Technician
Hess, Oswald Structural modifications of the Y-chromosome in D. hydei and their relations to gene activity
Runk, Christel (Miss) Technician
Sander, Klaus Embryology, experimental analysis of development

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Becker, Gweneth L. (Mrs.) Lethals
Becker, Hans Joachim, Puffing and variegation
Haendle, Jutta (Mrs.) Somatic crossing over
Illmensee, Karl Developmental genetics
Janning, Wilfried Position-effect variegation
Kalisch, Wolf-Ekkehard Unstable loci
Kasten, Renate (Miss) Curator of stocks
Korge, Günter Puffing
Kress, Horst Puffing
Moritz, Karl Cytochemistry
Röper, Hella (Miss) Technician

Münster (Westf.) Institut für Strahlenbiologie der Universität

Kraft, Marion (Miss) Technician
Scheid, Wolfgang Dr. cytology
Schmidt, Peter Graduate student Radiation genetics
Traut, Anneliese (Mrs.) Technician
Traut, Professor Radiation genetics

Tübingen: Max-Planck-Institut für Biologie, Abteilung Reichardt, 74 Tübingen, Spemannstr. 38
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Davidson, Terence Graduate student. Genetics of behaviour
Franceschini, Nicolas Graduate student. Properties of the visual system
Götz, Karl G. Ph.D. Research Associate. Nervous integration, mutant behaviour
Hengstenberg, Roland Graduate student. Electrophysiology of the visual system
Köhler, Bärbel (Miss) Technician. Curator of Stocks

74 Tübingen: Max-Planck-Institut für Biologie, Abteilung Beermann, Spemannstr 34
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Arcos, Laura (Miss) DNA-replication in salivary glands
Beermann, Wolfgang Physiology of salivary gland chromosomes
Boyd, James Biochemistry of salivary glands
Berendes, H. D. Salivary glands of Drosophila hydei (on leave: Dept. of Physiology, College of Physicians and Surgeons, Columbia University, 630 W. 130th St., N. Y.)
Brandt, J. W. Technical Assistant
Hennig, Wolfgand Biochemical aspects of Y-chromosome function in spermatogenesis (RNA synthesis and metabolism)
Hertling, Sigrid (Miss) Technician
Meyer, Günther F. Gametogenesis, light and electron microscopy; fine structure of chromosomes

WESTERN-GERMANY

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Kosswig, Curt. Professor Dr.

GREAT BRITIAN

Birmingham, England: The University, Department of Genetics
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Barnes, B. W., Ph.D. Research Associate. Genetical architecture and natural selection in Drosophila
Gale, J. S., Ph.D. Lecturer. Biometrical genetics of Papaver.
Jinks, J. L. Ph.D. D.Sc. Professor. Systems of variation of fungi; extra-chromosomal inheritance; biometrical genetics
John, B. Ph.D. Reader. Chromosome variation in wild and cultured populations
Jones, D. A., Ph.D. Lecturer. Differentiation in fungi; population genetics of Lotus corniculatus.
Jones, G. H. Ph.D. Assistant Lecturer. Genotypic control of chromosome behaviour
Kearsey, M. J. Ph.D. Lecturer. Genetic architecture in Drosophila and natural selection.
Killick, R. J. B.Sc. Research Student. Genetic architecture of continuous variation in Drosophila.
Lawrence, M. J. Ph.D. Lecturer. Experimental studies with higher plants.
Smith, D. A. Ph.D. Senior Lecturer. Methionine synthesis in Salmonella typhimurium.

Cambridge, England: Cambridge University, Department of Genetics, Milton Road

Alderson, T. Ph.D. Mutagenesis
Ashburner, M. Research Assistant. Puffing
Charlesworth, B. Research Student. Population genetics
Davies, R. W. Research student. Quantitative and developmental genetics
Gibson, J. B. Ph.D. Quantitative and developmental genetics
Hudson, G. J. Laboratory assistant
Lawrence, P. Ph.D. Developmental genetics
Thoday, J. M. Sc.D. F.R.S. Professor. Selection
Walker, S. (Mrs.) Research student. Population genetics

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Auerbach, C. (Miss) D.Sc., F.R.S. Professor. Mutagenesis.
Basden, E. B. Research Assistant. Wild species.
Boyman, M. J. Stock-keeper
Clayton, G. Lecturer. Selection
Knight, G. R. Research Assistant. Subobscura salivaries.
Nguyen thi Man (Miss) Research Visitor. Drosophila Nucleic acids.
Perry, M. Research Assistant. Autoradiography
Ratnayake, W. C. Graduate student. Chemical Mutagenesis.
Robertson, A., D.Sc., F.R.S. Quantitative genetics
Robertson, E. (Mrs.) Research Assistant. Embryology
Robertson, F. W., D.Sc. Biochemical and population genetics
Rogers, J. (Miss) Body colour polymorphism
Russel, M. Graduate student. Drosophila growth
Slizynska, H. (Mrs.) Ph.D. Cytological analysis
Slizynski, B. M. Ph.D. Salivaries
Waddington, C. H. Sc.D. F.R.S. Professor. General Genetics.

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Dyer, K.F. B.Sc. Radiation Genetics, population genetics
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Hiroyoshi, T. Japan, Osaka
Hirschler, D. E. Duarte, California
Hittle, M. (Mrs.) Duarte, California
Hizikuro, S. (Miss) Japan, Osaka
Ho, F. Berkeley, California
Hoar, D. I. Davis, California
Hochman, B. Knoxville, Tennessee
Hodges, C. (Mrs.) Raleigh, North Carolina
Hoffman, F. Syracuse, New York
Holden, J. (Miss) Canada, Vancouver, B. C.
Hollander, J. Netherlands, Haren (Gr.)
Hollander, W. F. Ames, Iowa
Hollingdale, B. J. (Miss) Australia, Sydney
Hollingsworth, M. J. England, London
Holm, D. G. Storrs, Connecticut
Holmgren, P. Sweden, Umea
Holt, A. C. E. (Mrs.) England, Reading
Homyk, T. Chapel Hill, North Carolina
Honda, Y. (Miss) Japan, Nagasaki
Hooper, G. B. Poughkeepsie, New York
Hossainy, E. (Mrs.) Austin, Texas
Hottinger, S. L. Bowling Green, Ohio
Howard, E. F. Madison, Wisconsin
Howland, M. F. (Mrs.) Stony Brook, New York
Huang, S. L. Austin, Texas
Hubby, J. L. Chicago, Illinois
Huck, R. E. (Miss) St. Louis, Missouri
Hudson, G. J. England, Cambridge
Hughes, J. Urbana, Illinois
Hunt, D. M. England, London
Hunt, P. (Mrs.) Berkeley, California
Hürlimann, R. Switzerland, Zürich
Hynes, M. Australia, Adelaide
Ichimaru, K. (Miss) Japan, Nagasaki
Ikeda, H. Japan, Tokyo
Illmensee, K. Germany, München
Imaizumi, Y. (Miss) Japan, Chiba
Inagaki, E. Japan, Chiba
Irvine, S. R. Logan, Utah
Ito, R. Ann Arbor, Michigan
Ito, S. (Miss) Japan, Tokyo
Ives, P. T. Amherst, Massachusetts
Iyama, S. Japan, Misima
Jackson, D. (Mrs.) Chapel Hill, North Carolina
Jackson, M. Long Beach, California
Jacobs, G. E. (Miss) Madison, Wisconsin
Jacobs, M. E. Goshen, Indiana
Jain, H. K. India, New Delhi
Janning, W. Germany, München
Jeffery, D. Berkeley, California
Jenkins, J. B. Los Angeles, California
Jensen, R. Tucson, Arizona
Jha, A. P. India, Bhagalpur
Jinks, J. L. England, Birmingham
Joardar, B. India, Calcutta
John, B. England, Birmingham
Johnsen, R. Providence, Rhode Island
Johnsen, R. C. Garden City, LI, New York
Johnson, C. S. (Miss) Bowling Green, Ohio
Johnson, F. M. Austin, Texas
Johnson, L. (Mrs.) Houston, Texas
Johnson, W. E. Austin, Texas
Johnson, W. W. Albuquerque, New Mexico
Johnston, F. (Mrs.) Storrs, Connecticut
Jones, D. A. England, Birmingham
Jones, D. M. Seattle, Washington
Jones, G. H. England, Birmingham
Judd, B. H. Austin, Texas
Jungen, H. Switzerland, Zürich
Jurow, K. Ithaca, New York
Kaji, S. Japan, Kobe
Kale, P. G. India, Varanasi
Kalicki, H. G. (Mrs.) Garden City, LI, NY
Kalisch, W-E. Germany, München
Kambysellis, M. Austin, Texas
Kan, Y. S. Korea, Seoul
Kanehisa, T. Japan, Kobe
Kaneko, A. D. Japan, Sapporo
Kang, S-J. (Miss) Korea, Seoul
Kaplan, M. A. Eugene, Oregon
Kaplan, M. L. New York, New York
Kaplan, R. A. (Miss) Madison, Wisconsin
Kaplan, W. D. Duarte, California
Karlik, A. (Miss) Austria, Vienna
Kasten, R. (Miss) Germany, München
Kastritsis, C. D. Dallas, Texas
Kauffman, B. P. Ann Arbor, Michigan
Kaufman, T. C. Austin, Texas
Kawabe, M. Japan, Kobe
Kawaguchi, M. (Miss) Japan, Nagasaki
Kawanishi, M. Japan, Misima
Kearsey, M. J. England, Birmingham
Keeler, E. (Mrs.) Iowa City, Iowa
Keenleyside, M. H. A. Canada, London
Keith, A. Berkeley, California
Kelker, A. (Miss) Austin, Texas
Kellermann, J. Chapel Hill, North Carolina
Kelly, N. (Mrs.) Eugene, Oregon
Kemp, R. England, Harwell
Kenyon, A. M. (Mrs.) Athens, Georgia
Kernaghan, R. P. Stony Brook, New York

Khan, A. H. West Pakistan, Tandojam
Khishin, A. F. United Arab Republic, Assuit
Kikkawa, H. Japan, Osaka
Killick, R. J. England, Birmingham
Kim, K. W. Korea, Kwangju
Kimura, M. Japan, Misima
King, A. L. Salt Lake City, Utah
King, R. C. Evanston, Illinois
King, R. J. DeKalb, Illinois
Kircher, H. Tucson, Arizona
Kirchgessner, T. L. Albuquerque, New Mexico
Kiriazis, W. C. Madison, Wisconsin
Kirschbaum, W. F. Argentina, Buenos Aires
Kishimoto, Y. (Miss) Japan, Kobe
Kitagawa, O. Japan, Tokyo
Kitazume, Y. Japan, Kobe
Klenk, K. Switzerland, Zürich
Klingele, D. (Sr.) Notre Dame, Indiana
Klug, M. A. (Mrs.) Evanston. Illinois
Klug, W. S. Evanston, Illinois
Knight, G. R. Scotland, Edinburgh
Kobelt, D. Switzerland, Zürich
Koch, E. A. (Mrs.) Evanston, Illinois
Koenig, P. B. (Miss) Chapel Hill, N. C.
Koepfer, R. (Mrs.) New York, New York
Köhler, B. (Miss) Germany, Tübingen
Kojima, K-I. Austin, Texas
Komma, D. J. Urbana, Illinois
Kono, S. (Miss) Japan, Osaka
Korge, G. Germany, München
Korinek, E. (Mrs.) Canada, Vancouver, B.C.
Kossevig, C. West Germany, Hamburg
Kosuda, K. Japan, Tokyo
Kraft, M. (Miss) Germany, Münster
Krause, C. E. (Mrs.) Storrs, Connecticut
Krause, E. South Orange, New Jersey
Krauss, M. I. (Miss) Storrs, Connecticut
Kress, H. Germany, München
Kresslová, Z. Czechoslovakia, Prague
Krider, H. M. (Miss) Madison, Wisconsin
Krikortz, M. Sweden, Umea
Krimbas, C. B. Greece, Athens
Krivshenko, E. D. Rochester, New York
Krivshenko, J. D. Rochester, New York
Kroeger, H. Switzerland, Zürich
Kroman, R. Long Beach, California
Krunić, M. Yugoslavia, Belgrade
Kubli, E. Switzerland, Zürich
Kuhn, D. T. Tempe, Arizona
Kunze-Mühl, E. (Mrs.) Austria, Vienna
Kuroda, Y. Japan, Misima
Kurokawa, H. Japan, Tokyo
Kusyk, C. (Miss) Chapel Hill, North Carolina
Kvelland, I. (Miss) Norway, Blinden
Kyselka, C. F. (Miss) Trenton, New Jersey
Laban, B. S. (Mrs.) Bowling Green, Ohio
Lahiri, S. India, Calcutta
Lakhotia, S. C. India, Calcutta
Lakovaara, S. Finland, Helsinki
Lamb, M. J. England, London
Landa, Z. Czechoslovakia, Prague
Landner, L. Sweden, Stockholm
Lange, E. L. Lafayette, Indiana
Langer, B. (Mrs.) Pittsburgh, Penn.
Latter, B. D. H. Australia, Canberra
Lauge, G. (Miss) France, Lyon
Laughnan, J. R. Urbana, Illinois
Lawrence, M. J. England, Birmingham
Lawrence, P. England, Cambridge
Lee, B. W. Korea, Seoul
Lee, C. C. Korea, Seoul
Lee, C. S. Korea, Seoul
Lee, T. J. Korea, Seoul
LeFever, H. M. Emporia, Kansas
Lefevre, G. Northridge, California
Lenhard, L. Topeka, Kansas
Lesnik, L. H. Philadelphia, Pennsylvania
Leuthold, U. Switzerland, Zürich
Leventhal, E. A. (Mrs.) Madison, Wisconsin
Levins, R. Chicago, Illinois
Levitan, M. Fairfax, Virginia
Levy, A. (Miss) Australia, Adelaide
Lewgoy, F. Brazil, Porto Alegre
Lewis, A. (Miss) Milwaukee, Wisconsin
Lewis, E. B. Pasadena, California
Lewis, H. W. Washington, D. C.
Lewis, K. Washington, D. C.
Lewis, R. (Mrs.) England, Brighton
Lewontin, R. G. Chicago, Illinois
Lifschytz, E. Israel, Jerusalem
Limburn, S. W. England, London
Lindsay, H. A. (Miss) Austin, Texas
Ling, L. (Miss) New Haven, Connecticut
Lints, C. V. (Mrs.) Belgium, Heverlee-Louvain
Lints, F. A. Belgium, Heverlee-Louvain
Lippold, T. Milwaukee, Wisconsin
Lloyd, B. (Miss) Australia, Adelaide
Lokki, J. Finland, Helsinki
Lott, R. G. Tempe, Arizona
Louw, J. H. México, México
Louw, J. H. South Africa, Stellenbosch
Low, P. (Mrs.) Logan, Utah
Lowenstein,L. L. (Miss) Madison, Wisconsin
Lu, C. C. (Mrs.) Houston, Texas
Lucchesi, J. C. Chapel Hill, North Carolina
Luce, W. M. Urbana, Illinois
Luchowski, E. (Mrs.) Buffalo, New York
Ludwig, M. Brazil, Porto Alegre
Ludwig, M. R. Brazil, Porto Alegre
Ludwig, N. C. Brazil, Porto Alegre
Lundelius, J. (Mrs.) Austin, Texas
Lundin, M. Sweden, Umea
Lüning, K. G. Sweden, Stockholm
MacBean, I. T. Victoria, Bundoora
Machado, D. M. Brazil, Porto Alegre
Machida, I. Japan, Chiba
MacIntyre, R. Ithaca, New York
MacKenzie, I. (Mrs.) Australia, Canberra

Maeda, Y. Japan, Kobe
Magalhaes, L. E. De Brazil, Sao Paulo
Magnusson, J. Sweden, Stockholm
Mahowald, A. P. Milwaukee, Wisconsin
Mainx, F. Austria, Vienna
Majoral, J. (Miss) Spain, Barcelona
Makino, S. Japan, Sapporo
Malecky, H. (Mrs.) Australia, Canberra
Malich, C. W. Moffett Field, California
Mallmann, M. C. Brazil, Porto Alegre
Mancebo, V. Berkeley, California
Manchester, W. San Diego, California
Manek, V. (Mrs.) Uganda, Kampala
Manning, J. Salt Lake City, Utah
Mansilla, P. (Mrs.) Argentina, Buenos Aires
Marble, J. R. (Mrs.) Eugene, Oregon
Marien, D. New York, New York
Marinic, S. E. (Mrs.) Argentina, Buenos Aires
Marinković, D. Yugoslavia, Belgrade
Markowitz, E. (Mrs.) Iowa City, Iowa
Marques, E. K. Brazil, Porto Alegre
Martell, K. (Miss) Stony Brook, New York
Maruyama, T. Japan, Misima
Marzluf, G. A. Milwaukee, Wisconsin
Masterson, J. E. Ames, Iowa
Masuda, H. (Miss) Japan, Misima
Mates, J. W. Eugene, Oregon
Mather, W. B. Australia, Brisbane
Matter, B. Switzerland, Zürich
Mauldin, V. (Mrs.) Austin, Texas
Mayer, E. Milwaukee, Wisconsin
Mayo, M. J. (Mrs.) Australia, Adelaide
Mazar Barnette, B. Argentina, Buenos Aires
McCrady, W. B. Arlington, Texas
McFadyen, M. (Miss) Tucson, Arizona
McFarlane, J. L. (Mrs.) Riverside, California
McGary, P. Fairfax, Virginia
McKinley, A. Australia, Sydney
McKinley, K. (Miss) Austin, Texas
McReynolds, M. M. (Mrs.) Austin, Texas
McWhorter, C. G. (Mrs.) Austin, Texas
Mead, C. G. Oak Ridge, Tennessee
Mead, R. M. (Mrs.) Austin, Texas
Megahih, M. A. United Arab Republic, Assuit
Meiss, L. H. (Mrs.) Urbana, Illinois
Mendez, H. Brazil, Porto Alegre
Mensua, J. L. Spain, Barcelona
Merle, J. (Miss) France, Lyon
Merriam, J. R. Pasadena, California
Mettler, L. E. Raleigh, North Carolina
Meyer, G. F. Germany, Tübingen
Meyer, H. U. (Mrs.) Madison, Wisconsin
Micheli, A. France, Roma
Milkman, R. D. Syracuse, New York
Miller, D. D. Lincoln, Nebraska
Miller, D. M. (Mrs.) Pullman, Washington
Miller, M. A. (Mrs.) Bowling Green, Ohio
Miller, M. F. (Mrs.) Oak Ridge, Tennessee
Minato, K. Japan, Misima
Mindek, G. Switzerland, Zürich
Minis, D. (Mrs.) Princeton, New Jersey
Misra, R. K. Canada, London, Ont.
Mitchell, H. K. Pasadena, California
Mitra, N. India, Calcutta
Mittler, J. E. (Miss) DeKalb, Illinois
Mittler, S. DeKalb, Illinois
Mizushima, T. (Miss) Japan, Osaka
Mohler, B. (Mrs.) Iowa City, Iowa
Mohler, J. D. Iowa City, Iowa
Moisand, R. E. Buffalo, New York
Momma, E. Japan, Sapporo
Monclus, M. (Mrs.) Spain, Barcelona
Montalenti, G. France, Roma
Montelius, I. Sweden, Stockholm
Moon, K. W. Korea, Seoul
Moore, B. M. (Mrs.) Houston, Texas
Moore, M. (Mrs.) Duarte, California
Morales, N. B. Brazil, Porto Alegre
Moree, R. Pullman, Washington
Mori, S. (Miss) Japan, Nagasaki
Moritz, K. Germany, München
Moriwaki, D. Japan, Tokyo
Morrow, D. L. Ithaca, New York
Morton, M. (Mrs.) Charlottesville, Virginia
Moskwinski, T. (Miss) Notre Dame, Indiana
Motylinski, C. Cleveland, Ohio
Mourao, C. A. Brazil, Sao Paulo
Mourgues, C. (Mrs.) France, Lyon
Moyer, S. Boston, Massachusetts
Muckenthaler, F. A. Albany, New York
Mukai, P. (Miss) Los Angeles, California
Mukai, T. Raleigh, North Carolina
Mukerjee, D. Houston, Texas
Mukherjee, A. S. India, Calcutta
Munoz, E. R. Argentina, Buenos Aires
Murakami, A. Japan, Misima
Murata, M. Japan, Chiba
Murphy, C. (Mrs.) Berkeley, California
Murphy, J. P. Austin, Texas
Mycock, W. (Mrs.) Princeton, New Jersey
Myszewski, M. Australia, Canberra
Nakagawa, K. (Miss) Japan, Tokyo
Nakai, S. Japan, Chiba
Nakanishi, H. Y. Japan, Chiba
Nakao, Y. Japan, Chiba
Nakashima-Tanaka, E. Japan, Osaka
Napp, M. Brazil, Porto Alegre
Narayanan, Y. (Mrs.) St. Louis, Missouri
Narise, S. Japan, Misima
Narise, T. Japan, Misima
Nash, D. Canada, Edmonton
Nash, D. J. Fort Collins, Colorado
Nash, K. R. Carbondale, Illinois
Nash, N. (Miss) Moffett Field, California
Nath, E. J. Canada, Edmonton
Nawa, S. Japan, Misima
Nawar, M. M. United Arab Republic, Assuit
Neeley, J. C. Portland, Oregon

Nei, M. Japan, Chiba
Neupol, S. Ithaca, New York
Newlin, L. Salt Lake City, Utah
Newmann, D. Brazil, Porto Alegre
Newton, J. A. Chapel Hill, North Carolina
Nguyen thi, M. (Miss) Scotland, Edinburgh
Nicholl, L. T. (Mrs.) Bowling Green, Ohio
Nickerson, R. Syracuse, New York
Nickla, H. Tempe, Arizona
Nicoletti, B. France, Roma
Nielsen, J. T. Denmark, Aarhus
Nigon, V. France, Lyon
Nilsson, B. (Mrs.) Sweden, Stockholm
Nolte, D. J. Johannesburg, Johannesburg
Norman, S. D. (Miss) DeKalb, Illinois
Novitski, E. Australia, Canberra
O'Brien, S. Ithaca, New York
Ogaki, M. Japan, Osaka
Ogawa, A. (Miss) Japan, Chiba
Ogita, Z. Japan, Osaka
Ogonji, G. Washington, D. C.
Oguma, M. Japan, Tokyo
Ohba, S. Japan, Tokyo
Ohta, T. Japan, Misima
Oishi, K. New Haven, Connecticut
Okada, T. Japan, Tokyo
Oksala, T. A. Finland, Turku
Okuno, T. (Miss) Japan, Osaka
Olembo, R. J. Uganda, Kampala
Oliver, C. P. Austin, Texas
Oliver, M. Lafayette, Indiana
Olivieri, G. France, Roma
Olofsson, E. Sweden, Umea
Olvera, R. O. (Miss) México, México
O'Neal, L. (Mrs.) Newark, New Jersey
Ondrej, M. Czechoslovakia, Prague
Orem, J. Seattle, Washington
Oshima, C. Japan, Misima
Oster, I. I. Bowling Green, Ohio
Oster, P. (Mrs.) Bowling Green, Ohio
Ostrowski, R. DeKalb, Illinois
Pak, W. Lafayette, Indiana
Pakonen, C. Z. (Mrs.) Pullman, Washington
Pal, T. K. India, Calcutta
Palatinik, M. Brazil, Porto Alegre
Palmer, T. Washington, D. C.
Park, M. S. Korea, Kwangju
Parker, D. R. Riverside, California
Parker, M. (Mrs.) Houston, Texas
Parks, C. (Mrs.) St. Louis, Missouri
Parry, D. Seattle, Washington
Parsons, P. A. Victoria, Bundoora
Pasternak, M. E. Duarte, California
Pasteur, G. New York, New York
Pasztor, L. M. Australia, Canberra
Pateman, J. A. Australia, Adelaide
Paterson, H. E. Australia, Nedlands
Patton, J. Tucson, Arizona
Paulay, R. Milwaukee, Wisconsin
Pauming, E. Princeton, New Jersey
Pavan, C. Brazil, Sao Paulo
Pavlovsky, O. (Mrs.) New York, New York
Paz, C. (Miss) Argentina, Buenos Aires
Peacock, W. J. Australia, Canberra
Peeples, E. E., Jr., Austin, Texas
Peletz, B. (Miss) DeKalb, Illinois
Pendley, J. San Diego, California
Peres, A. P. Brazil, Porto Alegre
Pereyra, E. (Miss) Argentina, Buenos Aires
Perez, R. S. Duarte, California
Perez-Davila, Y. (Mrs.) Chicago, Illinois
Perreault, W. J. Ann Arbor, Michigan
Perrin, M. Switzerland, Zürich
Perrin-Waldemer, C. France, Clermont-Ferrand
Perry, M. Scotland, Edinburgh
Persson, K. Sweden, Umea
Peternick, L. K. (Mrs.) Canada, Vancouver
Petersen, K. (Miss) Ann Arbor, Michigan
Peterson, S. San Diego, California
Petkovic, D. (Mrs.) Yugoslavia, Belgrade
Petri, W. Berkeley, California
Petroff, E. Knoxville, Tennessee
Pettit, B. Philadelphia, Pennsylvania
Pettit, B. (Miss) Milwaukee, Wisconsin
Pettitt, A. J. (Mrs.) Ann Arbor, Michigan
Philip, U. England, Newcastle Upon Tyne
Phillips, D. C. Syracuse, New York
Phillips, J. P. Logan, Utah
Ping-Ting, K. Canada, Edmonton
Pipkin, S. Washington, D. C.
Pittendrigh, C. S. Princeton, New Jersey
Plati, L. Medford, Massachusetts
Plaut, W. S. Madison, Wisconsin
Plough, H. H. Amherst, Massachusetts
Plowman, C. Australia, Brisbane
Polson, D. S. (Mrs.) Houston, Texas
Portin, P. Finland, Turku
Pospisil, Z. V. (Mrs.) Eugene, Oregon
Possani, L. P. Brazil, Porto Alegre
Poulson, D. F. New Haven, Connecticut
Prakash, S. Chicago, Illinois
Pratt, R. (Miss) Canada, Vancouver, B. C.
Prendergast, L. (Mrs.) San Diego, California
Prevosti, A. Spain, Barcelona
Procunier, D. Canada, Vancouver, B. C.
Prokopova, A. (Mrs.) Czechoslovakia, Prague
Prout, T. Denmark, Aarhus
Prout, T. Riverside, California
Puro, J. Finland, Turku
Pyati, J. Albuquerque, New Mexico
Pyle, A. (Mrs.) Iowa City, Iowa
Quamme, H. (Mrs.) Madison, Wisconsin
Quinn, R. Princeton, New Jersey
Radcliffe, C. Urbana, Illinois
Rahman, Z. India, Bhagalpur
Rai-Chaudhuri, A. India, Calcutta
Raikow, R. (Mrs.) Berkeley, California
Ramamurthy, G. Berkeley, California

Ramel, C. Sweden, Stockholm
Ramila, D. Brazil, Porto Alegre
Randolph, D. (Miss) Lafayette, Indiana
Rangaswamy, V. S. Bowling Green, Ohio
Rasch, E. M. (Mrs.) Milwaukee, Wisconsin
Rasch, R. W. Milwaukee, Wisconsin
Rasmuson, B. Sweden, Umea
Rasmuson, M. Sweden, Umea
Rathanasabapathy, V. India, Madras
Rathie, K. A. Australia, Sydney
Ratnayake, W. C. Scotland, Edinburgh
Ratty, F. J. San Diego, California
Raut, R. N. India, New Delhi
Ray, J. H. Lexington, Kentucky
Rayburn, M. (Mrs.) Raleigh, North Carolina
Rayle, R. E. Davis, California
Reguly, M. L. Brazil, Porto Alegre
Reichert, D. F. Austin, Texas
Reis, D. Brazil, Porto Alegre
Reischl, E. Brazil, Porto Alegre
Remensberger, P. Switzerland, Zürich
Rendel, J. M. Australia, Sydney
Rengo, M. East Lansing, Michigan
Resch, K. M. (Miss) Austin, Texas
Rha, C. H. Korea, Kwangju
Rhoads, J. Lafayette, Indiana
Ribó, G. (Miss) Spain, Barcelona
Richards, B. Cleveland, Ohio
Richardson, R. H. Austin, Texas
Richmond, R. New York, New York
Ricky, L. Ithaca, New York
Rico, M. Spain, Valencia
Riker, C. Providence, Rhode Island
Rinehart, R. R. San Diego, California
Ringelé, M. M. Belgium, Heverlee-Louvain
Robb, J. A. New Haven, Connecticut
Robbins, L. Seattle, Washington
Roberts, R. Chicago, Illinois
Robertson, A. Scotland, Edinburgh
Robertson, E. (Mrs.) Scotland, Edinburgh
Robertson, F. W. Scotland, Edinburgh
Robertson, G. Raleigh, North Carolina
Robins, S. England, London
Rocha, F. J. Brazil, Porto Alegre
Rocha, Z. M. A. Brazil, Porto Alegre
Rockwell, R. Dayton, Ohio
Rockwood, E. S. (Miss) Austin, Texas
Roe, S. (Miss) Houston, Texas
Rogers, C. K. Knoxville, Tennessee
Rogers, J. (Miss) Scotland, Edinburgh
Romrell, L. J. Logan, Utah
Ronen, A. Israel, Jerusalem
Röper, H. (Miss) Germany, München
Rose, R. W. Philadelphia, Pennsylvania
Rosenfeld, A. (Mrs.) Seattle, Washington
Rougier, M. France, Lyon
Rowan, M. J. (Sister) Rochester, New York
Roy, H. Baltimore, Maryland
Ruderer-Doschek, E. (Mrs.) Austria, Vienna
Rudkin, G. Philadelphia, Pennsylvania
Rüegg, M. Switzerland, Zürich
Runge, R. Lake Forest, Illinois
Runk, C. (Miss) Germany, Freiburg
Rushing, D. Chapel Hill, North Carolina
Russel, M. Scotland, Edinburgh
Russell, J. (Mrs.) Tucson, Arizona
Russell, P. (Mrs.) Amherst, Massachusetts
Sá, F. A. Brazil, Porto Alegre
Sabol, E. M. (Miss) Madison, Wisconsin
Sack, C. East Lansing, Michigan
Sacks, J. M. Pittsburgh, Pennsylvania
Saeki, T. Japan, Chiba
Sakai, K. I. Japan, Misima
Salceda, S. V. México, México
Salverson, H. (Mrs.) Madison, Wisconsin
Salzano, F. M. Brazil, Porto Alegre
Sander, K. Germany, Freiburg
Sandler, L. M. Seattle, Washington
Sang, J. H. England, Brighton
Sanger, W. G. Lincoln, Nebraska
Santos, E. P. D. Brazil, Sao Paulo
Sanyal, C. India, Calcutta
Sarkar, D. N. India, Varanasi
Sasaki, F. (Miss) Japan, Tokyo
Satter, K. L. Tempe, Arizona
Savolainen, S. (Mrs.) Finland, Turku
Savontaus, M-L. (Mrs.) Finland, Turku
Sayers, E. R. Tuscaloosa, Alabama
Schabtach, E. Baltimore, Maryland
Schaffer, H. E. Raleigh, North Carolina
Schalet, A. Storrs, Connecticut
Scharloo, W. Netherlands, Haren (Gr.)
Scheid, W. Germany, Münster
Scheidt, G. Pasadena, California
Schewe, M. J. Chapel Hill, North Carolina
Schmidt, M. E. (Miss) Austin, Texas
Schmidt, P. Germany, Münster
Schneider, I. (Miss) Washington, D. C.
Schouten, S. C. M. Netherlands, Utrecht
Schubiger, G. Switzerland, Zürich
Schuitema, K. A. Netherlands, Haren (Gr.)
Schultz, J. Philadelphia, Pennsylvania
Schwinck, I. (Miss) Storrs, Connecticut
Scott, D. (Miss) Johannesburg, Johannesburg
Scowcroft, W. R. Australia, Canberra
Seaman, R. Fort Collins, Colorado
Seecof, R. L. Duarte, California
Seiger, M. B. Dayton, Ohio
Seki, T. Japan, Osaka
Sen, L. India, Calcutta
Sene, F. M. Brazil, Sao Paulo
Sergio, D. Washington, D. C.
Shafer, S. J. Philadelphia, Pennsylvania
Shaheen, S. (Mrs.) United Arab Republic, Assuit
Shamay, E. Israel, Jerusalem
Shannon, M. P. (Mrs.) Austin, Texas

Sharma, R. P. India, New Delhi
Sharp, L. (Miss) Galesburg, Illinois
Shaw, B. A. (Mrs.) Philadelphia, Pennsylvania
Shaw, L. Canada, Vancouver, B. C.
Sheldon Australia, Sydney
Shelton, E. E. Austin, Texas
Shen, M. W. (Mrs.) Austin, Texas
Sheppard, P. M. England, Liverpool
Sherald, A. Charlottesville, Virginia
Sherwin, R. N. Chicago, Illinois
Sherwood, E. (Mrs.) Berkeley, California
Shidler, D. (Mrs.) Lafayette, Indiana
Shields, G. England, Brighton
Shima, T. Japan, Sapporo
Shiomi, T. Japan, Nagasaki
Shorrocks, B. England, Newcastle Upon Tyne
Sibiga, S. M. (Mrs.) Storrs, Connecticut
Siervogel, R. M. Eugene, Oregon
Sigot, A. France, Strasbourg
Sillans, D. France, Lyon
Silva, E. (Miss) Brazil, Sao Paulo
Simmons, J. R. Logan, Utah
Simoes, C. V. Brazil, Porto Alegre
Simoes, G. V. Brazil, Porto Alegre
Simoes, N. Brazil, Porto Alegre
Simonsen, V. (Mrs.) Denmark, Aarhus
Singer, B. S. (Mrs.) Storrs, Connecticut
Singer, K. M. Storrs, Connecticut
Singh, B. N. India, Varanasi
Singh, V. K. India, Bhagalpur
Slatis, H. M. East Lansing, Michigan
Slizynska, H. (Mrs.) Scotland, Edinburgh
Slizynski, B. M. Scotland, Edinburgh
Smaldino, J. Albany, New York
Smit, A. (Mrs.) Pasadena, California
Smith, D. A. England, Birmingham
Smith, J. D. College Station, Texas
Smith, J. M. England, Brighton
Smith, J. (Mrs.) Chapel Hill, North Carolina
Smith, P. D. Chapel Hill, North Carolina
Smith, P. A. Evanston, Illinois
Smolker, R. England, Brighton
Södergren, A. Sweden, Umea
Sofer, W. H. Baltimore, Maryland
Sokoloff, A. San Bernardino, Calif.
Sondhi, G. (Mrs.) Newark, New Jersey
Sondhi, K. C. Newark, New Jersey
Sonnenblick, B. P. Newark, New Jersey
Sorsa, M. (Mrs.) Finland, Helsinki
Souza, H. M. L. De (Mrs.) Brazil, Sao Paulo
Spanton, S. (Mrs.) Bowling Green, Ohio
Spark, M. W. Chapel Hill, North Carolina
Spassky, B. New York, New York
Sperlich, D. Austria, Vienna
Spiegelman, S. Urbana, Illinois
Spiess, E. B. Chicago, Illinois
Spiess, L. D. (Mrs.) Chicago, Illinois
Spieth, H. T. Davis, California
Spieth, P. T. Eugene, Oregon
Spirito, S. (Miss) Argentina, Buenos Aires
Spofford, J. B. Chicago, Illinois
Sprackling, L. (Mrs.) Fort Collins, Colorado
Sprang, G. J. (Miss) Netherlands, Haren (Gr.)
Springer, R. Austria, Vienna
Srám, R. Czechoslovakia, Prague
Stalker, H. D. St. Louis, Missouri
Stanley, J. G. (Miss) Bowling Green, Ohio
Stanton, G. DeKalb, Illinois
Staub, M. Switzerland, Zürich
Stauffer, H. Berkeley, California
Stern, C. New York, New York
Stern, C. Berkeley, California
Stevenson, R. Kirksville, Missouri
Stewart, D. Berkeley, California
Stiers, R. O. Belgium, Heverlee-Louvain
Stoddard, A. (Miss) Pittsburgh, Pennsylvania
Stone, W. S. Austin, Texas
Stout, H. (Miss) Philadelphia, Pennsylvania
Stroh, A. (Mrs.) Garden City, L.I., New York
Strömnaes, Ö. Norway, Blinden
Stuart, F. (Mrs.) Eugene, Oregon
Sturtevant, A. H. Pasadena, California
Sulerud, R. L. Minneapolis, Minnesota
Sune, M. V. Brazil, Porto Alegre
Suomalainen, E. Finland, Helsinki
Surridge, J. F. Lincoln, Nebraska
Surver, W. Notre Dame, Indiana
Suzuki, D. T. Canada, Vancouver, B. C.
Svahlin, H. Sweden, Umea
Sved, J. New York, New York
Swaminathan, M. S. India, New Delhi
Szebenyi, A. Syracuse, New York
Szwabowski, D. (Miss) New Haven, Connecticut
Takahashi, F. (Miss) Japan, Chiba
Takaya, H. Japan, Kobe
Tallantire, A. C. (Miss) Uganda, Kampala
Tamulevich, P. Berkeley, California
Tancock, R. (Miss) Australia, Adelaide
Tanzarella, C. France, Roma
Tarasoff, M. (Mrs.) Canada, Vancouver, B.C.
Targa, H. J. Brazil, Sao Paulo
Taylor, W. Australia, Canberra
Tedeschi, M. V. (Mrs.) Brazil, Sao Paulo
Temin, R. G. (Mrs.) Madison, Wisconsin
Terwedow, H. Notre Dame, Indiana
Thoday, D. D. England, Sheffield
Thoday, J. M. England, Cambridge
Thomas, W. H. Carbondale, Illinois
Thompson, C. F., Jr. Pittsburgh, Pennsylvania
Thompson, I. (Mrs.) Syracuse, New York
Thompson, M. E. (Mrs.) Bowling Green, Ohio
Thompson, P. E. Ames, Iowa
Thompson, S. R. Notre Dame, Indiana
Thornell, E. (Mrs.) Houston, Texas
Throckmorton, L. H. Chicago, Illinois
Tiffany, B. (Miss) Amherst, Massachusetts
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Tiivola, A. (Mrs.) Finland, Helsinki
Tobari, I. Japan, Chiba
Tobari, Y. N. (Mrs.) Japan, Tokyo
Tobari, Y. (Mrs.) Austin, Texas

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Tokuda, T. (Miss) Japan, Misima
Tokumitsu, T. Japan, Sapporo
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Tolby, B. E. Eugene, Oregon
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Tonomura, Y. (Mrs.) Japan, Tokyo
Tonzetich, J. Durham, North Carolina
Torgerson, R. Salt Lake City, Utah
Townsend, J. I. Richmond, Virginia
Traut, A. (Mrs.) Germany, Münster
Traut, (Professor) Germany, Münster
Travaglini, E. C. Philadelphia, Pennsylvania
Tripp, T. Buffalo, New York
Trippa, G. France, Roma
Trosko, J. E. East Lansing, Michigan
Trout, W. E. Duarte, California
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Tsien, H. C. Belgium, Heverlee-Louvain
Tuinstra, E. J. (Miss) Netherlands, Utrecht
Turoczi, L. Newark, New Jersey
U, R. Durham, North Carolina
Ulrich, H. Switzerland, Zürich
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Umetani, K. (Mrs.) Madison, Wisconsin
Unanue, R. Duarte, California
Ursprung, H. Baltimore, Maryland
Valencia, J. I. Argentina, Buenos Aires
Valencia, J. I. Madison, Wisconsin
Valencia, R. M. (Mrs.) Argentina, Buenos Aires
Valencia, R. M. (Mrs.) Madison, Wisconsin
Valentin, J. Sweden, Stockholm
Valenzuela, M. (Miss) México, México
Vander Mey, K. Bellingham, Washington
Vander ooi, V. (Mrs.) Iowa City, Iowa
Van Herrewege, C. France, Lyon
Van Valen, P. (Mrs.) Chicago, Illinois
Varak, E. (Mrs.) Ithaca, New York
Vasistha, H. C. India, Varanasi
Veiga Neto, A. J. Brazil, Porto Alegre
Verlinden, J. M. Belgium, Heverlee-Louvain
Vianna, F. Brazil, Porto Alegre
Vinson, W. C. Cambridge, Massachusetts
Vlist, J. van der (Miss) Netherlands, Utrecht
Vloeberghs, J. V. Belgium, Heverlee-Louvain
Voelker, R. A. Austin, Texas
Voigtlander, J. A. (Mrs.) Madison, Wisconsin
von Borstel, R. C. Oak Ridge, Tennessee
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Vyse, E. R. Canada, Edmonton
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Walker, S. (Mrs.) England, Cambridge
Wallace, B. Ithaca, New York
Wambera, E. C. (Mrs.) Canada, London
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Warren, H. (Mrs.) Pittsburgh, Pennsylvania
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Watanabe, T. (Miss) Japan, Kobe
Watanabe, T. K. Japan, Misima
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White, N. Lafayette, Indiana
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Wick, A. Berkeley, California
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Widmer, B. Switzerland, Zürich
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Williams, J. (Mrs.) Raleigh, North Carolina
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Williams, S. (Miss) Ann Arbor, Michigan
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Woodruff, R. C. Logan, Utah

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Yamaguchi, O. Japan, Tokyo
Yamamoto, M. Japan, Tokyo
Yamazaki, H. I. (Mrs.) Japan, Tokyo
Yamazaki, T. Chicago, Illinois
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Yang, K. (Mrs.) Canada, Vancouver, B. C.
Yasuda, N. Japan, Misima
Yearick, M. (Miss) Pittsburgh, Pennsylvania
Yen, T. T. T. Indianapolis, Indiana
Yones, M. H. United Arab Republic, Assuit
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Yoon, S. B. Madison, Wisconsin
Yoshikawa, I. Japan, Nagasaki
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Young, W. J. Baltimore, Maryland
Ytterborn, K. H. Sweden, Stockholm
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Zimmerman, C. Charlottesville, Virginia
Zimmerman, W. F. Amherst, Massachusetts
Zouros, E. Greece, Athens
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Zweep, A. Netherlands, Haren (Gr.)

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