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FROM THE EDITOR

The editorial work on this issue has been done at the Department of Radiation Genetics and Chemical Mutagenesis, University of Leiden. We are indebted to Professor F. Sobels for making the facilities available for this effort, as well as for help in other ways.

The delay in producing this issue was caused in part by the move to Leiden, but the book is larger as a consequence, as we did not want timely material to "age" waiting for the next issue. As we are now well established until the fall of 1976, the next issue will not take as long to produce. However, financial necessity has caused us to return to our original program of one issue per year.

All matters pertaining to the receipt of issues may still be addressed to the University of Oregon. Until the end of August, 1976, contributions of material and other editorial matters may be sent to Drosophila Information Service, c/o E. Novitski, Sylvius Laboratory, University of Leiden, Leiden, The Netherlands.

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RESEARCH NOTES

TECHNICAL NOTES

MATERIALS REQUESTED OR AVAILABLE I

T.R.F. Wright reports that the stocks listed below constructed in Charlottesville, Virginia, have been sent to both the Pasadena, California and Bowling Green, Ohio stock centers to make them generally available for use.

1. Temperature-sensitive suppressor of forked stocks (see Dudick et al. 1974, Genetics 76: 487-510).

   car $\underline{1}(1)su(f)^{ts67g}$
   $f^{s}\underline{1}(1)su(f)^{ts67g}$
   $f^{u}\underline{1}(1)su(f)^{ts67g}/FM4,y^{31d}sc^{8}dm$ B

2. Stocks carrying chromosomes for use in various mutant screening schemes.

$\underline{1}(1)mys/FM4,y^{31d}sc^{8}dm$ B($\underline{1}^{ts}$)	Position of $\underline{1}^{ts}$ in FM4 chromosome unknown
sc ec cv ct^{6} v g^{2}f/FM7c,y^{31d} sc^{8}	w^{a} sn^{x2} v B($\underline{1}$(1)TW-9)
	Position of $\underline{1}$(1)TW-9, larval lethal, unknown
sc ec cv ct^{6} v g^{2} f/FM7c,y^{31d} sc^{8}	w^{a} sn^{x2} v B ($\underline{1}$(1)TW-24)
	Position of $\underline{1}(\underline{1})$TW-24, an embryonic lethak, unknown
DTS-91/In(2LR)O,$dp\underline{1}^{vI}$ Cy pr cn^{2}	A balanced, very reliable DTS for screens (see Wright, 1970, DIS 45:140)
b Tft $\underline{1}$(2)?/In(2LR)O,$dp\underline{1}^{vI}$ Cy pr	cn^{2}(DTS-513)
	A balancer chromosome with an EMS-induced DTS in it (see Falke and Wright, 1972, DIS 45:89-91)
b Tft $\underline{1}$(2)?/In(2LR)O,$dp\underline{1}^{vI}$ Cy pr	cn^{2}(DTS-100) Ditto
In(2LR)bw^{VI} ds^{33k}(DTS-18)/In(2LR)O,$dp\underline{1}^{vI}$ Cy pr cn^{2}	Ditto
DTS-2,Sb/TM3,y^{+} ri p^{p} sep bx^{34e} e^{s} Ser	A balanced, very reliable DTS for screens (see Wright, 1970, DIS 45:140)
FM6,y^{31d} sc^{8} B;Sp bw^{D}/SM5,al^{2} Cy	lt^{v} cn^{2} sp^{2};TM3,y^{+} ri pP sep bx^{34e} e^{s} Ser/Sb
	A balancer stock for the three major chromosomes. Goes best at room temperature (22°C)

3. Virginator stocks.

C(1)DX,y f/$\underline{1}$(1)$su(f)^{ts67g}$	At 29-30° an excellent virginator stock for attached-X virgins
oc ptg^{3} $\underline{1}$(1)TW-1^{cs}/FM7c,y^{31d} sc^{8}	w^{a} sn^{x2} v B($\underline{1}$(1)TW-9;DTS-91/In(2LR)O,$dp\underline{1}^{vI}$ Cy pr cn^{2}
	A cold sensitive virginator stock for a balanced DTS
oc ptg^{3} $\underline{1}$(1)TW-1^{cs}/FMc,y^{31d} sc^{8}	w^{a} sn^{x2} v B($\underline{1}$(1)TW-24);DTS-91/In(2LR)O,$dp\underline{1}^{vI}$ Cy pr cn^{2}
	Ditto
T(Y;2)CyO,DTS-513/+	At 30°C an excellent virginator for wild type females and the basic stock for making other virginator stocks (see Wright and Green, DIS 51:108)

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Division of Biology

Note: The symbol, *, is used for cross indexing and signifies that the mutant is carried in a stock whose number is shown at the right. (For recent additions see Appendix page 16)

Wild Stocks

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

Chromosome 1

* ac 174
8 ac^3 w^a
9 amx/FM3, y^{31d} sc^8 dm B 1
10 amx lz^g v/y f:=
* amx . . . . . (see lz^K)
11 Ax
* bb . . . . . 26, 63, etc.
* bb^{G3} 133
* bb^1 . . . . . 738, 780
* bb^N 167
* bb^{poi} . . . . . (see bb^{G3})
12 B
13 B bx^r car/y f:=
* B^3 42
* B^iB^i 45
* BB 43
* BB^{36b} 44
14 Bg B/In(1)AM
15 bi ct^6 g^2
16 bo v
17 br
18 br w^e ec rb t^4/FM1, y^{31d} sc^8 w^a lz^s B
* br^3 664
19 Bx
20 Bx^2
21 Bx^3
22 Bx^J
* Bx^r 13
* Bx^{r49k} 144
23 car
* cho 101
* cho^2 187
24 cm
25 cm ct^6
* Co 184
26 cs^{53}/y w bb
* ct^6 . . . . . 15, 25, etc.
* ct^K 175
27 ct^n oc/FM1, y^{31d} sc^8 w^a lz^s B
* cu-X 787
* cv . . . . . 102, 103, etc.
28 cx
29 cx^{tg} oc/FM1, y^{31d} sc^8 w^a lz^s B
30 dm/y f:=
31 dor/y f:=
32 dor^1/FM6, y^{31d} sc^8 dm B (nub/+); see also 764
33 dow/FM6, y^{31d} sc^8 dm B
* dx 38, 125
* dx^{st} 664, 665
34 dy
* e(bx) . . . (= en-bx) . . . 788
* $e(bx)^2$ . . . (= en^2-bx) . . 678
* $e(S)^x$ . . . (= en^x-S) . . . 666
35 Eag
36 ec
37 ec ct^6 s car/FM6, y^{31d} sc^8 dm B
38 ec dx
* eq 102, 737
39 Ext/FM6, y^{31d} sc^8 dm B
40 f
41 f B/y f:=
42 f B^3/y f:=
43 f BB/y f:=
44 f BB^{36b}/y f:=
45 f B^iB^i/y f:=
46 f fu/y f:=
* f^3 167
* f^5 157
47 f^{36a}
* f^{B15} 774
* f^{B27} 775
48 fa
* fa^n
* flp . . . . . (see flw)
49 flw
50 fo
51 fx/y f:=
* fu 46
* fw 146, 170
* fw^{34e} 194
52 g^2
53 g^2 pl/FM3, y^{31d} sc^8 dm B 1
54 g^2 ty/y f:=
* g^4 . . . . . 667, 733
55 gg^2/FM6, y^{31d} sc^8 dm B
56 gg^3
57 gt w^a
58 Hk^2
* Hw . . . . . 114, 733, etc.
59 Hw^{49c}/FM1, y^{31d} sc^8 w^a lz^s B
60 if^3
61 kz
* 1(1)7 . . . . . (see dor^1)
62 I(1)J1 sc^{J1}/I(1)J1 sc^{J1}/Dp(1;f)24
63 lh B car bb/y f:=
64 lz/FM3, y^{31d} sc^8 dm B 1

65 lz^3/y f:=
66 lz^{34}/y f:=
67 lz^{36}/y f:=
68 lz^{37}
69 lz^{48f}/y f:=
* lz^{50e} 766
70 lz^{BS} lz^{46} lz^{g} ras^4 v/y f:=
* lz^{D} 667
* lz^{g} . . . . . 10, 180, etc.
71 lz^{K}
* lz^{s} . . . . . 18, 27, etc.
* lz^{y4} 120, 766
72 m
* m^2 . . . . . 667, 733
73 m^{D}/FM3, y^{31d} sc^8 dm B l
74 M9L0n/FM6, y^{31d} sc^8 dm B
75 M(1)o f/FM6, y^{31d} sc^8 dm B
76 M(1)o^{Sp}/FM6, y^{31d} sc^8 dm B
* M(1)Sp . . . . . (see M(1)o^{Sp})
77 mal/y f:=
* mal^{bz} 183
78 na/y f:=
79 ny f/FM1, y^{31d} sc^8 w^a lz^s B (ri)
80 oc ptg^3/In(1)C1B
* od . . . . . (see os^o)
81 os^o
82 os^s

84 peb v
* pl 53
* pn . . . . . 109, 176, etc.
85 pn^2
* pn^3 105
* ptg_1 679
86 ptg^2
* ptg^3 80
* ptg^4 141
87 r^9/y f:=
88 r^{39k} f B/In(1)AM
89 ras_1dy
90 ras^2
91 ras^3 m
* ras^4 70
* ras^v 845
92 rb
93 rb cx
94 rg
95 rst^2/FM1, y^{31d} sc^8 w^a lz^s B
96 rux/FM6, y^{31d} sc^8 dm B
97 rux^2
98 s
99 sbr
100 sc
101 sc cho t
102 sc cv v eq (sc reverted)
103 sc cv v f
104 sc ec cv ct^6 v_1 g^2 f/FM3, y^{31d} sc^8 dm B l
105 sc pn^3 g^{2rv} Bx^2 ... (g^2 reverted) (sc reverted)
106 sc z ec ct^6
107 sc z swb^{62b} (Ives)
108 sc z w^{17G2} ec ct^6
109 sc^2 pn/y f:=
110 sc^{3B}
111 sc^{3-1} w/y f:=
* sc^4 794
112 sc^5
113 sc^6 w^a
* sc^7 . . . . . 722, 796, etc.
* sc^8 . . . . . 784, 801, etc.
* sc^9 805
* sc^{10} . . . . . (see ac^3)
114 sc^{10-1}/y Hw
* sc^{19} 838
* sc^{D2} 182
* sc^{J1} 62
* sc^{J4} 769
* sc^{S1} 178
* sc^{S2} 837
* sc^{260-14} 806
* sc^{260-15} 847
* sc^{260-22} 807
* sc^{z} 846
115 scp t
116 sd^{58d14}/y f:=
117 Sh^5
118 shf^2
* sl . . . . . (in C1B, $C1B^{36d}$)
* sl^2 723
* sn 713
* sn^2 161
119 sn^3
120 sn^3 lz^{y4} v/y f:=
121 sn^4
122 sn^{34e}
123 sn^{36a}/y f:=
* sp-w . . . . . (see w^{sp})
124 spl
* sta . . . . . (see T(1;3)sta)
* su(Cbx) 677
125 su(dx) dx
* su(f) . . (= su^{W}-f) . . . 145
126 $su(s)^2$ v (bw)
127 $su(s)^2$ w^a cv t

129 $su(f)^{S}$v/FMA3, y^2 (bw)
* $su(w^a)$ . . (= su-w^a) . . . 700, 708, etc.
* su^{S2}-v-pr . . . . . (see $su(s)^S$)
130 svr
131 svr w^a
132 svr^{poi}
133 svr^{poi}-dish bb^{G3}
134 sw
135 sx vb^2 os^s/FM6, y^{31d} sc^8 dm B
* sy . . . . . (see os^s)
136 t
137 t^2 v f
138 t^3
* t^4 18
139 t^5 v r^{12}
* tuh-1 . . . (= tu-h) . . 673
140 tw/FM1, y^{31d} sc^8 w^a lz^s B

* ty 54
* tyl (- ty-l) . . . 779, 780
141 un Bx^2/In(1)AM, ptg^4
142 un^4
143 v
144 v f Bx^{r49k} car/y f:=
145 v f su(f)
146 v^2 fw
147 v^{36f}
* v^{0f} 781
148 vb
* vb^2 135
149 vs
150 w
151 w m f
152 w sn^3 m
* w^{11E4} 199
* w^{17G2} 108
153 w^a
154 w^a mw mit
155 w^{a2}
156 w^{a3}
157 w^{a4}
158 w^{bf} f^5
159 w^{bf2}
* w^{bf3} 697
160 w^{Bwx}
161 w^{ch} wy
162 w^{co} sn^2
163 w^{col}
164 w^e
165 w^{e2}
166 w^{ec3}
167 w^h
168 w^i f^3 bb^N
169 w^{sat}
170 w^{sp}
171 w^t fw
172 w^u
173 wy
* wy^2 192
174 y
175 y ac v
176 y ct^K (bw)
177 y pn
178 y pn w cm ct^6 sn^3 oc ras^2 v dy g^2 f os^o car sw/FM7b, y^{31d} w^a lz^s B
179 y pn w cm ct^6 sn^3 oc ras^Z v dy g^2 f os^o car sw/In(1)sc^{S1}, In(1)dl-49, y v B
180 y sc
181 y sc lz^g v f/y f:=
182 y sc^5
183 y sc^{D2}
184 y v f mal ^{bz}
185 y w Co/y f:=
186 y w spl
187 y z^a w^{65a25} spl sn^3/y w f:=
188 y z^a w^{cn}/y w f:=
189 y^2
190 y^2 cho^2
191 y^2 cv v f
192 y^2 sc w^a ec
193 y^2 w^a
194 y^2 w^a w
195 y^2 wy^2 g^2 (g^2 partly reverted)
196 y^{2S}
197 y^{2S} fw^{34e}
198 y^{3d}/y f:=
* y^{3P} 812
* y^4 814
* y^{31d} . . . . . 9, 18, etc.
199 y^{34c}
* y^{59b} 709
200 y^{td}
201 y^{v2}
201A z w^{11E4}

Chromosome 2

202 a px or
203 a px sp
204 ab
205 ab^2/T(Y;2)E
206 ab^2 ix^2 bw sp^2/In(2L+2R)Cy, Cy dp^{lvI} Bl L^4 sp^2
* abb 403
207 abr/In(2L+2R)Cy, Cy hk^2
208 abr/SM5, al^2 Cy lt^v sp^2
209 ad
210 al
211 al b c sp^2
212 al dp b bw 1(2)ax/SM5, al^2 Cy lt^v sp^2
213 al dp b pr ap^{blt} bw/SM5, al^2 Cy lt^v sp^2
214 al dp b pr Bl c px sp/SM1, al^2 Cy sp^2
215 al dp b pr Bl c px sp/In(2LR)O, dp^{lvI} Cy pr cn^2
216 al dp b pr c px sp
217 al dp b pr Hx
218 al S ast ho/SM1, al^2 Cy sp^2
* al^2 . . . . . 210, 211, etc.
* alpha-l . . . . . (see tyr-1)
219 Alu
220 an/SM5, al^2 Cy lt^v sp^2
221 an^2/SM1, al^2 Cy sp^2
222 ang
223 ant(ro)
224 ap^4/SM5, al^2 Cy lt^v sp^2
225 ap^{wow}
226 arch ch1/SM5, al^2 Cy lt^v sp^2
227 ast ho cl
* ast^3 815
228 ast^4 sp cl
* ast^X 300
* Ata 868
229 b
230 b tyr-1
231 b cn beta
232 b el rd^s pr cn
233 b Go/In(2LR)Gla
234 b Go/SM5, al^2 Cy lt^v sp^2
235 b gp

236 b j
237 b l(2)Bld pr c px sp/SM5, al^2 Cy lt^v sp^2
238 b lt wx^{wxt} bw
239 b pr tk/T(T;2)G
240 b sf
241 b vg
* ba 251
242 Bl/In(2L+2R)Cy, Cy bw^{45a} sp^2 or^{45a}
243 Bl/T(2;3)dp
244 Bl L^2/SM5, al^2 Cy lt^v sp^2
245 Bl stw^{48} ap^{blt} tuf sp/SM5, al^2 Cy lt^v sp^2
246 Bla/SM5, al^2 Cy lt^v sp^2
247 blo
* blt . . . . . (see ap^{blt})
248 bri
* bs 380
249 bs^2
* bs^3 328
250 bur fs(2)El/SM5, al^2 Cy lt^v sp^2
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252 bw ba
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* bw^{V34k} 342
256 c
257 c wt px
258 cg c/SM5, al^2 Cy lt^v sp^2
259 cg c/In(2LR)U
260 ch
261 chl
262 chl en/SM5, al^2 Cy lt^v sp^2
263 chl l(2)bw bw^{2b} mr^2/SM5, al^2 Cy lt^v sp^2
264 chy
265 ck/SM5, al^2 lt^v sp^2
266 cl
267 cl^2/T(Y;2)E
268 cn
* cn^2 (in all stocks containing In(2R)Cy)
269 cn bw
270 cn en/SM5, al^2 Cy lt^v sp^2
271 cn l(2)crc/SM5, al^2 Cy lt^v sp^2
272 cn^3/T(Y;2)C
273 cn^{35k}
* cq . . . . . (see rk^4)
274 cru/In(2L+2R)Cy, Cy (w^e)
275 d/SM5, al^2 Cy lt^v sp^2
276 d b/SM5, al^2 Cy lt^v sp^2
277 da/SM1, al^2 Cy sp^2
278 dil^2 hv bw sp/SM5, al^2 Cy lt^v sp^2
279 dke c
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286 dp^{ovN}
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* dpTh . . . . . (see dplvI)
* dp^{tx} . . . . . (see dplv)
* dp^v 690
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* dp^{vl} . . . . . (see dp^{vM})
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289 ds dp
290 ds^{rv} ft dp^{v2} l(2)M b pr/SM5, al^2 Cy lt^v sp^2
291 ds S G b pr/In(2L+2R)Cy, al^2 Cy lt^v L^4 sp^2
292 ds^W/In(2L)Cy^{LtR}, Su(S) dplvw pr
* ds^{33k} . . . . . 328, 353, etc.
293 ds^{38k}/In(2L)Cy, Cy dplv2 b pr
294 dsr
295 dw-24F cl/SM5, al^2 Cy lt^v sp^2
296 dw-24F l(2)cg, cg/SM5, al^2 Cy lt^v sp^2
* E(S) . . . (= EN-S) . . . 335, 395, etc.
297 ed Su(dx)^2
298 el
* en 261, 269, 748
* esc 816
299 ex
300 ex ds S^X ast^X/SM1, al^2 Cy sp^2
* fes . . . . . (see fs(2)B)
301 fj l(2)Su(H)/SM5, al^2 Cy lt^v sp^2
302 fj wt/SM5, al^2 Cy lt^v sp^2
303 fr/In(2L+2R)Cy, Cy dplv2
304 fr wt/SM5, al^2 Cy lt^v sp^2
305 Frd/In(2L+2R)Cy, Cy sp^2
* fs 2.1 . . . . . (see fs(2)El)
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* Go 232, 233
* gp 234
* gt-4 416
* Hia 439, 440
309 hk
310 hk pr
* hk^2 205
311 ho
312 hv/SM5, al^2 Cy lt^v sp^2
313 Hx/ see also 215
314 hy/SM5, al^2 Cy lt^v sp^2
315 hy a px sp/SM1, al^2 Cy sp^2
* ix 374
* ix^2 204
316 j
317 J/In(2L)NS
318 J^{34e}
319 kn

320 L
321 L^2
322 L^4
323 L^5
324 L^G
325 L^r
326 L^{si}
* 1(2)301 367
327 T(2)39 a px slt sp/SM5, al^2 Cy lt^v sp^2
328 T(2)a bs^3, In(2L)t/bw^{V1}, ds^{33k}
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329 T(2)ay b c sp^2/SM5, al^2 Cy lt^v sp^2
* T(2)Bld 236
* T(2)bw 262
* T(2)C 399
* T(2)cg 296
* T(2)cre 270
330 T(2)gl a px or/SM5, al^2 Cy lt^v sp^2
331 T(2)H l^2/SM5, al^2 Cy lt^v sp^2
* T(2)M 290
332 T(2)mat/SM5, al^2 Cy lt^v sp^2
333 T(2)me/SM1, al^2 Cy sp^2
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* T(2)R 411
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* Tl 363
334 Tl^2
335 Tm/In(2L+2R)Cy, Cy S^2 $dp lv^2$ E(S)
336 Tt/T(Y;2)A
337 Tt std/SM2, al^2 Cy lt^v sp^2
338 Tt stw^3
* Tt^3 . . . . . 291, 864, 888
* Tt^v . . . . . 206, 210, etc.
339 Ttd
340 Tw
* Tys 691
341 M(2)173/SM5, al^2 Cy lt^v sp^2
* M(2)B . . . . . (see $M(2)z^B$)
342 $M(2)e^S$/In(2L+2R)Cy, Cy, In(2R)bw^{V34}
343 $M(2)H^{S5}$/SM5, al^2 Cy lt^v sp^2
344 $M(2)l^2$/XM1, al^2 Cy sp^2
345 $M(2)m^{S6}$/SM5, al^2 Cy lt^v sp^2
346 $M(2)S2^3$/SM2, al^2 Cy lt^v sp^2
347 $M(2)S2^9$/SM5, al^2 Cy lt^v sp^2
* M(2)S3 . . . . . (see $M(2)S2^3$)
* M(2)S5 . . . . . (see $M(2)H^{S5}$)
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349 M(2)z/SM5, al^2 Cy lt^v sp^2
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351 $M(2)z^B$/SM5, al^2 Cy lt^v sp^2
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353 mr bs^2/bw^{V1}, ds^{33k}
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366 pi/SM5, al^2 Cy lt^v sp^2
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* Pin 415
368 pk cn
369 pk tuf (sp^2/+)
* Pm . . . . . (see bw^{V1})
* Pm^2 . . . . . (see bw^{V32g})
370 po vg
371 po^2
372 pr
373 pr cn/T(Y;2)C
374 pr cn ix/SM5, al^2 Cy lt^v sp^2
375 pr^{bw}
376 pu
* Pu^{Gr} 881
377 puf
378 pw-c/SM5, al^2 Cy lt^v sp^2
379 px
380 px bs (old Berlin stock of Goldschmidt)
381 px bw sp/T(Y;2)J
382 px bw mr sp/bw^{V1}, ds^{33k}
383 px slt sp
384 pym/In(2L+2R)Cy, Cy
385 pys
386 Q
* rc 691
387 rd/SM5, al^2 Cy lt^v sp^2
* rd^s 231
388 rdo
389 rdo^2 pr
* Rev 823
* Rev^B 753
390 rh
391 rk^4
392 rl
* rn 882
* Roi 441
393 rub
394 Ruf/bw^{V1}, ds^{33k}
* Rvd . . . . . (see Rev^B)
395 S/In(2L+2R)Cy, Cy E(S) (K-pn)
396 S Sp ab^2 ltd/SM5, al^2 Cy lt^v sp^2
* S^2 . . . . . 335, 771
397 SR/bw^{V1}, ds^{33k}
* S^X 300
398 sca
399 sca 1(2)C/SM5, al^2 Cy lt^v sp^2
400 SD-5/SM1, al^2 Cy sp^2
401 SD-72/SM5, al^2 Cy lt^v sp^2

* sf 239
402 sf^2
403 shr bw^{2b} abb sp/SM5, al^2 Cy lt^v sp^2
404 shv
405 shv ho
* Sk 350
* slt 327, 383
406 sm px/SM5, al^2 Cy lt^v sp^2
407 sm px pd/SM5, al^2 Cy lt^v sp^2
408 so
409 so^2 b cn
* sp . . . . . 201, 212, etc.
410 sp^2 bs^2
411 Sp/In(2L)t, l(2)R
412 Sp/SM5, al^2 Cy lt^v sp^2
413 Sp Bl N-2G/SM5, al^2 Cy lt^v sp^2
414 Sp J/SM5, al^2 Cy lt^v sp^2
415 Sp J L^2 Pin/SM5, al^2 Cy lt^v sp^2
416 spd gt-4/SM5, al^2 Cy lt^v sp^2
417 sple
418 spt
419 std/SM5, al^2 Cy lt^v sp^2
420 stw
421 stw^2
422 stw^3/T(Y;2)B
423 stw^5
424 stw^{48} ap^{blt} tuf sp^2
* Su(dx) . . . (= Su-dx) . . 665
* $Su(dx)^2$ . . (=Su^2-dx) . . 358, 664
* Su(er) . . . (= Su-er) . . 693
425 Su(H)/In(2L+2R)Cy, Cy pr
426 Su(H) whd l(2)Su(H)/SM5, al^2 Cy lt^v sp^2
* Su(S) 292
* tet 668
427 Tft/SM1, al^2 Cy sp^2
* Tg 818
* tk 238
428 tkd/SM5, al^2 Cy lt^v sp^2
429 tkv
430 tri vg^{No2}/SM5, al^2 Cy lt^v sp^2
* tu 252
* tu-36a . . . (=tu^{36a}) . . 604
431 tuf ltd
432 tyr-l (p^p); see also 229
433 Uf
434 vg
435 vg bw
436 vg^D/SM5, al^2 Cy lt^v sp^2
437 vg^{ni}
* vg^{No2} 430
438 vg^{np}
439 vg^{nw} Hia/SM5, al^2 Cy lt^v sp^2
440 vg^{nw} Hia/T(2;3)Sb^M, In(2L+2R)Cy, Cy
441 vg^U/In(2L)t, Roi, In(2R)Cy, bw sp^2 or
442 vst/SM5, al^2 Cy lt^v sp^2
443 whd
444 wt
* wx^{wxt} . . . (= wxt) . . . 237

Chromosome 3

445 a(3)26
* a-3 . . . . . (see a(3)26)
446 aa h
447 aa tu-36e
448 abd
* $Antp^B$ 826
449 app
450 as^{hg}
451 as^{hg} e^s
* Ata 868
452 bar-3
* Bd 566
453 Bd^G/In(3R)C, l(3)a
454 bf/TM6, ss^- bx^{34e} Ubx^{P15} e
* bod 563
* bp . . . . . (see bul^{bp})
455 bul
456 bul^{bp}/TM1, Me ri sbd^1
457 bv
* bx 594, 608
458 bx^3 Cbx Ubx bxd pbx/T(2;3)ap^{Xa}
459 bx^{34e}
* bxd 458, 595, 873
* bxd^{107} 902
* by 576, 577
* c(3)G . . . (= c3G) . . . 600
460 ca
461 ca bv
462 ca_2K-pn
463 ca^2
* ca^{nd} 497
464 Cbx
465 cd
466 cmp ca/TM6, ss^- bx^{34e} Ubx^{P15} e
467 cp
468 cp in ri p^p
469 cu
470 cu kar
471 cu kar ry^8
472 cur
473 cv-c
474 cv-c sbd^2
475 cv-d
* Cyd 489
476 D/Gl
477 D^3 Sb ca^2/In(3L+3R)P
478 det
479 Dfd/In(3LR)Cx
480 Dfd^r
481 Dl_3H e^s cd/In(3R)P, spr
482 Dl^3/In(3R)C, e
483 Dl^5/In(3R)C, l(3)a
484 Dl^7/In(3LR)Ubx^{130}, Ubx^{130} e^s
485 Dl^9/In(3R)C, e
486 Dl^{11}/In(3L+3R)P, Dfd ca
487 Dl^{12}/In(3L+3R)P, Dfd ca
488 Dl^{131}/In(3R)C, Sb e l(3)e
489 Dl^{14}/In(3R)Cyd, Cyd
* Dl^B 827

490 Dl^{x}/In(3L+3R)P
* Dr 556
491 Dr^{Mio}/TM6, ss^{-} bx^{34e} Ubx^{P15} e
492 drb
* dsx 551
493 dwh/In(3L+3R)P, Dfd ca
* e . . . . . 681, 682, In(3R)C
* $e(dp)^{v}$ 690
494 e^{4} wo ro
495 e^{11}
496 e^{s}
497 e^{s} ca^{nd}/TM6, ss^{-} bx^{34e} Ubx^{P15} e
498 eg/In(3LR)Cx
499 eg^{2}/In(3LR)Cx
* er 684, 693
500 eyg
* fl 541
501 fz
502 gl
503 gl^{2} e^{4}
504 gl^{3}
505 gl60j
506 Gl Sb/LVM
* gm 559, 623
507 gro
508 gs
509 h_{2}
510 h^{2}
511 H/In(3R)P
512 H Pr/In(3R)C, e
513 H^{2}/T(2;3)ap^{Xa}
514 H^{3}/In(3R)C, Sb e l(3)e
* H^{57c} 620
* Hm 878
* Hn 879
* Hn^{r} 518
515 Hn^{r3} sr
* Hu 828
516 in
517 jv
518 jv Hn^{r} h
519 jvl
* k 588
* K-pn 395, 462
* kar 470, 471, 614
520 kar^{2}
521 Ki
522 l(3)dl0/In(3LR)Cx, D
* l(3)a 453, 483
523 l(3)ac e^{s} M(3)w/LVM
* l(3)e . . . . . 488, 514, etc.
* l(3)PL . (In(3L+3R)P; In(3L+3R)P, Dfd ca)
* l(3)PR . " " "
524 l(3)tr Sb/In(3L)P, In(3R)P18, Me Ubx e^{4}
525 l(3)tr Ubx/TM1, Me ri sbd^{1}
* l(3)W 605, 827
* l(3)XaR 867
526 ld
527 Ly/D^{3}
528 Ly Sb/LVM
* M(3)36e . . . . . (see M(3)be^{36e})
529 M(3)40130/In(3L+3R)P, Dfd ca
* M(3)124 . . . . . (see M(3)w^{124})
* M(3)B . . . . . (see M(3)w^{B})
* $M(3)B^{2}$ . . . . . (see M(3)w^{B2})
530 M(3)be^{36e}/In(3R)C, l(3)a
531 M(3)h^{S37}/In(3L)P, Me
532 M(3)h^{y}/In(3L)P, Me
533 M(3)S32/T(2;3)Me
534 M(3)S34/T(2;e)Me
535 M(3)S36/T(2;3)Me
* M(3)S37 . . . . . (see M(3)h^{S37})
* H(3)w 523
536 M(3)w/In(3R)C, e l(3)e
537 M(3)w^{124}/In(3R)C, e l(3)e
538 M(3)w^{B}/In(3R)C, e l(3)e
539 M(3)w^{B2}/In(3R)C, e l(3)e
* M(3)y . . . . . (see M(3)h^{y})
540 ma
541 ma fl
542 mah
543 Mc/T(2;3)ap^{Xa}
544 mwh
545 N-X/T(2;3)ap^{Xa}
546 obt
547 p
548 p^{p}
549 p^{p} bx sr e^{s}
550 p^{p} cu
551 p^{p} dsx/TM6, ss^{-} bx^{34e} Ubx^{P15} e
552 pb/In(3LR)Cx
553 pbx/T(2;3)ap^{Xa}
554 Pc/TM1, Me ri sbd^{1}
* Pdr 692
555 Pr/In(3R)C, e
556 Pr Dr/TM3, y^{+} ac^{+} ri p^{p} sep bx^{34e} e^{s}
557 Pt/T(2;3)ap^{Xa}, ca
558 pyd
559 R Ly/In(3L)P, gm
560 ra
561 red
562 ri
563 ri bod e^{s}/In(3L)P, Me, In(3R)C, Sb e l(3)e
564 ri p^{p}/T(Y;2;3)F, st
565 ro
566 ro Bd ca/In(3R)C, l(3)a
567 ro ra ca/T(2;3)Me
568 rs^{2}
569 rsd
* rt^{2} 587
570 ru
571 ru h th st p^{p} H e^{s} ro/TM6, ss^{-} bx^{34e} Ubx^{P15} e
572 ru h th st cu sr e^{s} ca
573 ru h th st cu sr e^{s} ca/TM3, ru Sb Ser
574 ru h th st cu sr e^{s} Pr ca/TM6, ss^{-} bx^{34e} Ubx^{P15} e
575 ru h th st p^{p} cu sr e^{s}
576 ru lxd by
577 ru^{g} jv se by
578 ry
* ry^{8} 471

579 Sb/In(3LR)Ubx^{101}, Ubx^{101}
580 Sb H/In(3R)C, cd
581 Sb Ubx/T(2;3)ap^{Xa}
582 Sb^{63b}/In(3LR)Ubx^{130}, Ubx^{130} e^{s}
583 Sb^{Spi}/In(3LR)Cx
* Sb^{V} 885, 886
* sbd 603
584 sbd^{2}
* sbd^{105} 757
* sbd^{1} 456, 525
585 se
586 se h
587 se rt^{2} th/In(3L)P, Me
588 se ss k e^{s} ro
* sed . . . . . (see Hn^{r3})
* sep 556, 825, 885
589 Ser/In(3R)C, e 1(3)e
590 snb
591 sr
592 sr gl
593 ss
594 ss bx Su(ss)^{2}
595 ss bxd k e^{s}/T(2;3)ap^{Xa}
596 ss^{a}
597 ss^{aB}
598 ss^{a40a}
599 st
600 st c(3)G ca/TM1, Me ri sbd^{1}(sp^{2})
601 st in ri p^{p}
602 st Ki p^{p}
603 st sbd e^{s} ro ca
604 st sr e^{s} ro ca (tu-36a)
605 st sr H^{2} ca/In(3R)P^{W}, st 1(3)W ca
606 st^{sP}
* su(pd) . . (= su-pd) . . . 679
607 su(pr)^{B}/TM6, ss^{-} bx^{34e} Ubx^{P15} e (pr)
608 su(Hw)^{2} bx bxd/TM1, Me ri sbd^{1} (sp^{2})
* Su(ss)^{2} . . (= su^{2}-ss) . . 594
609 su(t) (t)
* su(tu) . . (= su-tu) . . . 693
610 su(ve) ru ve h th
611 th
612 th st cp
613 th st pb p^{p}/TM6, ss^{-} bx^{34e} Ubx^{P15} e
614 th st pb p^{p} cu kar su(Hw)^{2} jv1 ss bx sr gl(TM6, ss^{-} bx^{34e} Ubx^{P15} e
* tra 674
* tra^{D} 844
615 Tri/In(3LR)DcxF
616 tt wo
617 Tu (= Tubby)
* tu-36e . . (=tu^{36e}) . . 447
* tuh-3 673
618 tx
619 Ubx e^{4}/In(3L+3R)P, Dfd ca
620 Ubx^{61d}/H^{57c}
* Ubx^{101} 579
* Ubx^{130} . . . . . 484, 582, 674, etc.
621 ve
622 ve h th
623 ve R/In(3L)P, gm
* vo-3 . . . . . (see e(dp^{v}))
624 W
625 W Sb/In(3LR)Cx
626 We/In(3L)P, Me, In(3R)C, e 1(3)e
627 wk/In(3L+3R)P, Dfd ca
628 wo
* Xa . . (= T(2;3)ap^{Xa}) . . 458, 543, etc.

Chromosome 4

629 ar/ey^{D}
630 bt
631 bt ey^{R} sv^{n}
632 bt^{D}/ci^{D}
633 Ce^{2}/spa^{Cat}
634 ci ey^{R}
635 ci ey^{R} sv^{n}
636 ci gv1 bt
637 ci gv1 ey^{R} sv^{n}
638 ci sv^{n}
639 ci^{361}
640 co^{57g}
641 ci^{D}/ey^{D}
642 ci^{W}
643 ey
644 ey^{2}
645 ey^{4}
* ey^{D} 629, 641, 662
* ey^{R} . . . . . 634, 635, etc.
646 gv1
647 gv1 ey^{R}
648 gv1 ey^{R} sv^{n}
649 1(4)2^{f}/ci^{D} (Hochman)
650 1(4)4^{c}/ci^{D} "
651 1(4)6^{b}/ci^{D} "
652 1(4)14^{b}/ci^{D} "
653 1(4)15^{2}/ci^{D} "
654 1(4)21/ci^{D} "
655 1(4)22/ci^{D} "
656 1(4)25/ci^{D} "
* 1(4)AM-1 . . . . . (see 1(4)22)
* 1(4)PT-1 . . . . . (see 1(4)6^{b})
* 1(4)PT-2 . . . . . (see 1(4)2^{f})
* 1(4)PT-3 . . . . . (see 1(4)4^{c})
* 1(4)SLC-1 . . . . . (see 1(4)15^{2})
* 1(4)ST-1 . . . . . (see 1(4)21)
* 1(4)ST-2 . . . . . (see 1(4)14^{b})
* 1(4)ST-3 . . . . . (see 1(4)25)
* Ma1 694
657 spa
658 spa^{Cat}/ci^{D}
659 spa^{pol}
660 spa^{p65}
661 sv^{de}/ey^{D}
662 sv^{n}

Multichromosomal

663 br^{3} dxst;ed Su(dx)^{2} (1;2)
664 dxst;Su(dx) (1;2)

665 e(S)^{x}/FMA3, y^2;al S ast ho/SM1, al^2 Cy sp^2 (1;2)
666 lz^D/In(1)d1-49, m^2 g^4;bw^{V1}/In(2L+2R)Cy, Cy (1;2)
667 os^s;tet (1;2)
668 v;bw (1;2)
669 v;In(2R)bw^{VDel}/SM1, al^2 Cy sp^2 (1;2)
670 y ac w^{ch} fa/FMA3, y^2;Su(w^{ch})/ In(2L+2R)Cy, Cy (1;2)
672 sc z w^z rst;halo (1;3)
673 tuh-1;tuh-3 (1;3)
674 w^a v/FMA3, y^2;tra/In(3LR)Ubx^{130}, Ubx^{130} es (1;3)
675 w^e/FMA3, y^2;Dp(2;3)P/TM6, ss^- bx^{34e} Ubx^{P15} e (1;3)
676 y;mwh (1;3)
677 y su Cbx v/FMA3, y^2; su-Hw bx bxd/ ap^{Xa} (1;3)
678 y^2 $e(bx)^2$ w^{bf}/FMA3, y^2;sbd^2 ss bx^{34e}/ TM1, Me ri sbd^1 (1;3)
679 ptg;px pd;su(pd) (1;2;3)
680 FMA3, y^2;net;sbd^2;spa^{pol} (1;2;3;4)

682 y f:=;bw;e;ci ey^R (1;2;3;4)
683 y f:=;bw;e;spa^{pol} (1;2;3;4)
684 al dp b Fl c px sp/In(2L+2R)Cy, Cy; D/In(3L+3R)P (2;3)
685 b Su(er)$^+$ bw;st er (2;3)
686 bw;st (2;3)
687 bw^4;st (2;3)
688 bw^{V1}, sp b/In(2L+2R)Cy, Cy sp^2;Sb/ In(3LR)DcxF (ru h ca?) (2;3)
689 bw^{V1}, ds^{33k}/In(2L+2R)Cy, Cy;H/In(3R)Mo, sr (2;3)
690 cn;ry^2 (2;3)
691 dp^v;e(dp^v) (2;3)
692 lys rc;ss (2;3)
693 px pd;Pdr H, Dp(2;3)P/Pdr (2;3)
694 Su(er) tu bw;st er su(tu) (2;3)
695 pr;Mal (2;4)

Attached-X

696 br ec/y^{3d}
697 f B/su(s)^S v
* FM3, y^2 . . (= FMA3) . . 128, 129, etc.
698 w^{bf3}/sn^{36a}
699 y/g^2 ty
700 y pn/FM6, y^{31d} sc^8 dm B
* y pn v 709
* y v bb 786
* y v f 720
* y v f car 780
* y w bb 26
* y w f 783
* y^2 sc w^a ec 712

Attached Autosomal Arms

701 C(2L)P3, +;C(2R)P3, +
702 C(2L)P3, j^{63};C(2R)P4, px
703 C(2L)P4, dp;C(2R)P4, px
704 C(3L)P3, ri;C(3R)P3, sr
705 C(3L)P6, +;C(3R)P6, +
706 C(4)P1, ci ey^R/gv1 sv^n
707 C(4)P2, ci ey^R/gv1 sv^n

Attached-XY

708 v f B XY/y^2 su w^a w^a bb

710 XY^LY^S In(1)En/y^2 su-w^a w^a bb

711 Y^S/g^2 B.Y^L & y f:= ($dp^{ol}v$)(Stern)
712 Y^S X.Y^L, In(1)EN, In(1)d1-49, Y^S y.Y^L/y X.Y;bw;e;ci ey^R

Triploids

713a C(1)RM, In(1)d1-49, v^{Of} f/FM7
713b C(1)RM, y w fa^{no}/FM6 ♀ & FM6, y^{31d} sc^8 dm B/B^S Y y^+ ♂
713c C(1)RM, y^2 sc w^a ec/FM6 ♀ & FM6, y^{31d} sc^8 dm B/B^S Y y^+ ♂

Extra-Y

714 In(1)w^{m4L} $N^{264-84R}$, y sn/FM3, y^{31d} sc^8 dm B 1/Y ♀;dm sn ♂ (DIS 28:137)
715 y v f mal/mal^+ Y ♀;In(1)d1-49, B^{M1}, Df(1)mal^6, y v sn^{X2}/mal^+ Y ♂
716 y w f/y w^a/w^+.Y
* Y-bb 786
717 In(X)$^{c2}w^{vc}$/In(1)d1-49 l(1)J1, y w lz^s f^{36a} ♀ & In(1)d149 l(1)J1 y w lz^s f^{36a}/sc^8 Y♂
718 X^{cl}, y/y f:=/y^+ Y
719 X^{c2}, cv v f/ClB, v
720 R(1)5A, y w f B/In(1)d1-49, y v f car/B^S.Y (Pasztor's unstable ring)

Closed-Y

721 R(Y)bw^+/X;bw ("MYR")
* Y^c, bw^+. . . . . (see R(Y)bw^+)
722 Y^{Lc}/y w Y^S & FMA3, y^2

Deficiencies

Deficiencies-X

723 Df(1)260-1 Df(1)260-1/FM4, y^{31d} sc^8 dm B
724 Df(1)B^{263-20} Df(1)B^{263-20}/In(1)sc^7, In(1)AM, sc^7 car
725 Df(1)bb Df(1)bb, y sl^2 bb-/FM4, y^{31d} sc^8 dm B

726 Df(1)bb — Df(1)bb, y v car bb-/In(1)AM
* Df(1)bb^{1} — 738
727 Df(1)ct$^{268-42}$ — Df(1)ct$^{268-42}$, y/FM4, y^{31d} sc^{8} dm B
728 Df(1)g^{1} — Df(1)g^{1}, f B/In(1)AM
729 Df(1)m^{259-4c} — Df(1)m^{259-4c}/FM4, y^{31d} sc^{8} dm B
730 Df(1)N^{8} — Df(1)N^{8}/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
731 Df(1)N$^{264-39}$ — Df(1)N$^{264-39}$, w^{ch}/FM4, y^{31d} sc^{8} dm B
732 Df(1)N$^{264-105}$ — Df(1)N$^{264-105}$/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
* Df(1)rst^{2} — 95
* Df(1)sc^{4L} sc^{8R} — 795
* Df(1)sc^{8} — 769
733 Df(1)svr — Df(1)svr, Dp(1;f)101, sp1/y f:=
734 Df(1)w$^{258-11}$ — Df(1)w$^{258-11}$, y/In(1)d1-49, y Hw m^{2} g^{4}
735 Df(1)w$^{258-42}$ — Df(1)w$^{258-42}$, y/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
736 Df(1)w$^{258-45}$ — Df(1)w$^{258-45}$, y/FM4, y^{31d} sc^{8} dm B
737 Df(1)w$^{258-48}$ — Df(1)w$^{258-48}$, y sc^{5} sp1;Dp(1;3)w^{vco};y f:=

Deficiencies-Y

738 Df(Y)Y^{bb-} — Df(Y)Y^{bb-}, y^{2} eq
739 Df(Y)Yst — w^{e} bb^{1}/w^{e} bb^{1};Yst & w^{e} bb^{1};Y^{+};In(2L+2R)NS, px sp/1(2)mr^{2}

Deficiencies-2

740 Df(2)M33a — Df(2)M33a/bw^{V32g}
* Df(2)MB — . . . . . (see Df(2L)M-z^{B})
741 Df(2)MS4 — Df(2)MS4/SM1, al^{2} Cy sp^{2}
742 Df(2)MS8 — Df(2)MS8/SM1, al^{2} Cy sp^{2}
743 Df(2)MS10 — Df(2)MS10/SM1, al^{2} Cy sp^{2}
744 Df(2)rl^{10a} — Df(2)rl^{10a} lt cn/bw^{V1}, ds^{33k}
745 Df(2L)al — Df(2L)al/In(2L+2R)Cy, Cy E(S)
746 Df(2L)M-z^{B} — Df(2L)M-z^{B}/SM1, al^{2} Cy sp^{2}
747 Df(2L)S2 — Df(2L)S2/In(2L+2R)Cy, Cy E(S)
748 Df(2L)S3 — Df(2L)S3/SM1, al^{2} Cy sp^{2}
749 Df(2R)42 — Df(2R)42, en/SM1, al^{2} Cy sp^{2}
750 Df(2R)bw^{5} — Df(2R)bw^{5}, sp^{2}/T(2;3)ap^{Xa}
751 Df(2R)bw^{VDe2L}Cy^{R} — Df(2R)bw^{VDe2L}, In(2R)Cy^{R}/Gla
752 Df(2R)Px^{2} — Df(2R)Px^{2}, bw sp/SM1, al^{2} Cy sp^{2}
753 Df(2R)vg^{B} — Df(2R)vg^{B}/SM5, al^{2} Cy lt^{v} sp^{2}
754 Df(2R)vg^{C} — Df(2R)vg^{C}/In(2LR)Rev^{B}
755 Df(2R)vg^{C} — Df(2R)vg^{C}/SM5, al^{2} Cy lt^{v} sp^{2}
* Df(2R)vg^{D} — . . . . . (= vg^{D}) 436

Deficiencies-3

* Df(3L)Hn — 879
* Df(3L)Ly — . . . . . (= Ly) 527, 528
756 Df(3R)M-S31 — Df(3R)M-S31/T(2;3)Me
757 Df(3R)ry^{75} — kar^{2}, Df(3R)ry^{75}/In(3LR), M(3)S34 kar^{4} ry^{2} Sb
758 Df(3R)sbd^{105} — Df(3R)sbd^{105}, p^{p} sbd^{105} bx sr e^{5}/LVM

Deficiencies-4

759 Df(4)M4 — Df(4)M4/ey^{D}

Duplications

* Dp(1;f)24 — . . . . . (= Del(1)24) 62, 808
760 Dp(1;f)101 — Dp(1;f)101;In(1)sc^{8}, Df(1)sc^{8}, w^{a}
761 Dp(1;f)107 — Dp(1;f)107;In(1)sc^{8}, Df(1)sc^{8}, w^{a}
762 Dp(1;f)118 — Dp(1;f)118;In(1)sc^{8}, Df(1)sc^{8}, w^{a}

763 Dp(1;f)135 Dp(1;f)135;Df(1)sc^{8}, w^{a}
764 Dp(1;f)R Dp(1;f)R/y dor^{1}/y dor^{1}
* Dp(1;f)X^{c2} . . . . . (see Dp(1;f)R)
765 Dp(1;f)z^{9} Dp(1;f)z^{9}, Df(1)sc^{J4}/y f:=
766 Dp(1;1)112 Dp(1;1)112, y f (homozygous stock)
767 Dp(1;Y^{L})sc^{S1} sc^{S1}.Y^{L}/y.Y^{S};y f:=;cn bw;(e/+)
768 Dp(1;3)126 Dp(1;3)126;v f/In(3L+3R)P, Dfd ca
769 Dp(1;3)sc^{J4} Dp(1;3)sc^{J4}/Df(1)sc^{8}, w^{a}
* Dp(1;3)w^{vco} 736
770 Dp(2;2)S Dp(2;2)S, (S ast) (Sast^{4}) net dp cl/In(2L+2R)Cy, Cy E(S)
* Dp(2;3)P 692
771 Qn(2;2)S Qn(2;2)S, (ast)$_{5}$, al ho/In(2L+2R)Cy, Cy S^{2} E(S)

Inversions

Inversions-X

772 In(1)AB In(1)AB/y f:=
* In(1)AM . . . . . 14, 88, etc.
773 In(1)B^{M1} In(1)B^{M1}, v B^{M1} (tan-like); see also 784, 785, etc.
774 In(1)B^{M2} In(1)B^{M2}(rv f^{B15} (reinv.; mosaic)
775 In(1)B^{M2} In(1)B^{M2}, f^{B27} B^{M2}/ClB
776 In(1)B^{M2} In(1)B^{M2} v^{rv} B^{M2}
* In(1)bb 723, 724
* In(1)ClB In(1)Cl, sc t^{2} v sl B (= ClB) 80, 718
* In(1)ClB^{36d} In(1)Cl, sc t^{2} v sl B^{36d} (= ClB^{36d}) 851
779 In(1)dl-49 In(1)dl-49, tyl
780 In(1)dl-49 In(1)dl-49, tyl bb^{1}/y v f car
781 In(1)dl-49 In(1)dl-49, v^{Of} f
782 In(1)dl-49 In(1)dl-49, y fa^{n}
* In(1)dl-49 In(1)dl-49, y Hw m^{2} g^{4} 733, 798
783 In(1)dl-49 In(1)dl-49, y Su(Hw) Hw m^{2} g^{4}/y w f;(nub/+)
784 Ins(1)dl-49, B^{M1} In(1)dl-49, In(1)B^{M1}, l(1)J1 sc^{J1} oc ptg B^{M1}/In(1)sc^{S1}L sc^{8R}, y sc^{S1} sc^{8} pn w ec rb cm ct^{6} sn^{3} ras^{2} g^{2} f os^{s} os^{o} car l/l(1)J1^{+}.Y (= "Maxy")
785 Ins(1)dl-49, B^{M1} In(1)dl-49, In(1)B^{M1}, sc v B^{M1} (homozygous)
786 Ins(1)dl-49, B^{M1} In(1)dl-49, In(1)B^{M1}, y/Y^{-bb} & y v bb/Y^{-bb}
787 Ins(1)dl-49, B^{M1} In(1)dl-49, In(1)B^{M1}, y sc v cu-X B^{M1}
788 In(1)e(bx) In(1)e(bx), e(bx), e(bx)/y f:=
* In(1)EN 711
* Ins(1)FM1 In(1)FM1, In(1)dl-49, y^{31d} sc^{8} w^{a} lz^{s} B (=FM1). 18, 27, etc.
* In(1)FM3 In(1)FM3, y^{31d} sc^{8} dm B l (= FM3) . . . . . 9, 53, etc.
* In(1)FM4 In(1)FM4, y^{31d} sc^{8} dm B (= FM4) 712, 723
789 In(1)FM6 In(1)FM6, y^{31d} sc^{8} dm B/y f:=; see also 32, 33, etc.
790 In(1)FM7a In(1)FM7a, y^{31d} w^{a} v^{Of} B (homozygous) (see DIS 44:101)
* In(1)FM7b 178
* In(1)FMA3 In(1)FMA3, y^{2} (= FMA3) 128, 129
791 In(1)N$^{264-84}$ In(1)N$^{264-84}$, y/FM6, y^{31d} sc^{8} dm B
792 In(1)rst^{3} In(1)rst^{3}, rst^{3} (homozygous)
793 In(1)rst^{3} In(1)rst^{3}, y rst^{3} car bb
* In(1)S 809, 813
794 In(1)sc^{4} In(1)sc^{4}, y sc^{4}
795 In(1)sc^{4L} sc^{8R} In(1)sc^{4L} sc^{8R}, y; see also 700
796 In(1)sc^{7} In(1)sc^{7}, sc^{7}
797 In(1)sc^{7} In(1)sc^{7}, sc^{7} w^{a}
798 Ins(1)sc^{7}, AM In(1)sc^{7}, In(1)AM, sc^{7}/In(1)dl-49, y Hw m^{2} g^{4}
799 Ins(1)sc^{7}, AM In(1)sc^{7}, In(1)AM, sc^{7} car/FM4, y^{31d} sc^{8} dm (without B)
800 Ins(1)sc^{7}, B^{M1} In(1)sc^{7}, In(1)B^{M1}, sc^{7} w^{43b} B^{M1}/y f:=
801 In(1)sc^{8} In(1)sc^{8}, sc^{8}
802 In(1)sc^{8} In(1)sc^{8}, sc^{8} cv v f/y f:=
803 In(1)sc^{8} In(1)sc^{8}, y^{31d} sc^{8} w^{a}
804 In(1)sc^{8}, dl-49 In(1)sc^{8}, In(1)dl-49, y^{31d} sc^{8} (homozygous)

*	In(1)sc^{8R}	. . . . . 700, 784
805	In(1)sc^{9}	In(1)sc^{9}, sc^{9} Bx f t w^{a} (homozygous)
806	In(1)$sc^{260\text{-}14}$	In(1)$sc^{260\text{-}14}$, $sc^{260\text{-}14}$
807	In(1)$sc^{260\text{-}22}$	In(1)$sc^{260\text{-}22}$, $sc^{260\text{-}22}$
808	In(1)sc^{J1}	In(1)sc^{J1};Dp(1;f)24
*	Ins(1)sc^{S1}, dl-49	In(1)sc^{S1}, In(1)dl-49, y v b 178
*	Ins(1)sc^{S1L}, sc^{8R}	In(1)sc^{S1L}, In(1)sc^{8R}, y sc^{S1} sc^{8} pn w ec rb cm ct^{6} sn^{3} ras^{2} g^{2} f os^{s} os^{o} car l 784
809	Ins(1)sc^{S1L}, S, sc^{8R}	In(1)sc^{S1L}, In(1)sc^{8R}, sc^{S1} sc^{8} w^{a} B (= Muller-5)
*	In(1)sc^{S1R}	 813
810	In(1)w^{m4}	In(1)w^{m4} (bb?)
811	In(1)w^{m4}	In(1)w^{m4}, y cv m f/y f:=
812	In(1)y^{3P}	In(1)y^{3P}, y^{3P} B (B reverted)
813	Ins(1)y^{3PL}, S, sc^{S1R}	In(1)y^{3PL}, In(1)S, In(1)sc^{S1R}/ y f:=;sc^{19i}/In(2L+2R)Cy, Cy
814	In(1)y^{4}	In(1)y^{4}, y^{4}

2L Inversions

815	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 293, 350
*	In 2L)$Cy^{L}t^{R}$	In(2L)$Cy^{L}t^{R}$, Su(S) dp^{lv2} pr 292
*	In(2L)NS	 317
*	In(2L)t	 328
816	In(2L)t	In(2L)t, esc c sp/SM5, al^{2} Cy lt^{v} sp^{2}
817	In(2L)t	In(2L)t, lt l L^{4} sp^{2}/bw^{V1}, ds^{33k}
*	In(2L)t	In(2L)t, I(2)R 411
818	In(2L)Tg	In(2L)Tg, Tg/SM5, al^{2} Cy lt^{v} sp^{2}

2L + 2R Inversions

819	In(2L+2R)Cy	In(2L+2R)Cy, al^{2} E(S) cn^{2} sp^{2} (does not carry Cy mutant)
*	In(2L+2R)Cy	In(2L+2R)Cy, al^{2} Cy lt^{3} L^{4} sp^{2} 291, 864, 888
*		In(2L+2R)Cy, Cy . . . . . 273, 384, etc.
*		In(2L+2R)Cy, Cy bw^{45a} sp^{2} or^{45a} 241
*		In(2L+2R)Cy, Cy dp^{lv2} 303
*		In(2L+2R)Cy, Cy dp^{lvI} Bl L^{4} sp^{2} 204
*		In(2L+2R)Cy, Cy dp^{lvI} pr 838
*		In(2L+2R)Cy, Cy E(S) . . . . . 395, 744, etc.
*		In(2L+2R)Cy, Cy hk^{2} 205
*		In(2L+2R)Cy, Cy L^{4} sp^{2} 862, 869
*		In(2L+2R)Cy, Cy pr . . . . . 425, 887, etc.
*		In(2L+2R)Cy, Cy sp^{2} 305, 687
*		In(2L+2R)Cy, Cy S^{2} E(S) 771
*		In(2L+2R)Cy, Cy S^{2} dp^{lv2} E(S) 335
*	Ins(2L+2R)Cy, bw^{V34k}	In(2L+2R)Cy, Cy In(2R) bw^{V34k} 342
*	Ins(2L)Cy, (2R)NS	In(2L)Cy, Cy dp^{lv2} pr, In(2R)NS, l px l(2)NS sp . 361
820	In(2L+2R)NS	In(2L+2R)NS, b mr/In(2L+2R)Cy, Cy
*	In(2L+2R)NS	In(2L+2R)NS, px sp 738
*	Ins(2L)t, (2R)Cy	In(2L)t, Roi In(2R)Cy, bw sp^{2} or (= Roi) 441

2LR Inversions

821	In(2LR)102	In(2LR)102, ds^{W} sp^{2}/SM1, al^{2} Cy sp^{2}
*	In(2LR)bw^{V1}	In(2LR)bw^{V1}, ds^{33k} . . . . . 328, 353, etc.
*	In(2LR)bw^{V32g}	. . . . . 352, 739
*	In(2LR)dp	. . . . . (see T(2;3)dp)
*	In(2LR)Gla	. . . . . (= Gla) 232
*	In(2LR)O	 681
*	In(2LR)Pm	. . . . . (see In(2LR)bw^{V1})
*	In(2LR)Pm^{2}	. . . . . (see In(2LR)bw^{V32g})
*	In(2LR)Rev	. . . . . (= Rev) 823

*	In(2LR)Rvd	. . . . . (see In(2LR)Rev^{B})	
*	In(2LR)SM1	In(2LR)SM1, al^{2} Cy sp^{2} (= SM1)	212, 216, etc.
*	In(2LR)SM5	In(2LR)SM5, al^{2} Cy lt^{v} sp^{2} (= SM5)	206, 210, etc.
*	In(2LR)U	. . . . . (= U)	258

2R Inversions

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

3L Inversions

*	In(3L)D	. . . . . (= D)	476, 522, etc.
*	In(3L)D^{3}	. . . . . (= D^{3})	477, 527
*	In(3L)P	In(3L)P, gm	559, 623
*	In(3L)P	In(3L)P, Me	531, 532, etc.
*	In(3L)P	In(3L)P, Me ca	892, 894
824	In(3L)P	In(3L)P, mot-36e/R	

3L + 3R Inversions

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

3LR Inversions

*	In(3LR)Cx	. . . . . (= Cx)	479, 498, etc.
*	In(3LR)Cx	In(3LR)Cx, D	522, 893
*	In(3LR)DcxF	. . . . . (= In(3LR)CxF)	615, 687
*	In(3LR)DcxF	In(3LR)DcxF, ru h ca	840
825	In(3LR)sep	In(3LR)sep, sep ri p^{p}	
*	In(3LR)P35	. . . . . (= In(3LR)Pasadena-35)	886
*	In(3LR)TM1	In(3LR)TM1, Me ri sbd^{1} . . . . . (= TM1) . . .	456, 525, etc.
*	In(3LR)TM3	In(3LR)TM3, y^{+} ac^{+} ri p^{p} sep bx^{34e} e^{s} (= TM3) . .	556, 885
*	In(3LR)Ubx^{101}	. . . . .	579
*	In(3LR)Ubx^{130}	In(3LR)Ubx^{130}, Ubx^{130} e^{s} . . . (= Ubx^{130}) . .	484, 582, 674, etc.

3R Inversions

826	In(3R)Antp^{B}	In(3R)Antp^{B}, Antp^{B}/TM1, Me ri sbd^{1}	
*	In(3R)C	In(3R)C, cd	580
*		In(3R)C, e	482, 485, etc.
*		In(3R)C, e l(3)e	536, 537, etc.
*		In(3R)C, l(3)a	453, 483
*		In(3R)C, Sb e l(3)e	488, 514
*	In(3R)Cyd	In(3R)Cyd, Cyd . . . . . (= Cyd)	489
827	In(3R)Dl^{B}	In(3R)Dl^{B}, st Dl^{B}/In(3R)P^{W}, st l(3)W ca	
828	In(3R)Hu	In(3R)Hu, Hu Sb^{Spi}/In(3L+3R)P	
829	In(3R)Mo	In(3R)Mo, sr/T(2;3)ap^{Xa}, ca; see also 688	
*	In(3R)P	. . . . .	481, 511
*	In(3R)P18	. . . . . (= In(3R)Pasadena-18)	524
830	In(3R)P^{Fla}	In(3R)P^{Fla} (homozygous)	
*	In(3R)P^{W}	In(3R)P^{W}, st l(3)W ca	605, 827

Translocations 1;Y

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

Translocations 1;2

833 T(1;2)Bld — T(1;2)Bld, Bld/ClB (carries In(2R)Cy)
834 T(1;2)f^{Z57-15} — T(1;2)f^{Z57-15}/In(1)AM
835 T(1;2)lt — T(1;2)lt/In(2L+2R)Cy, Cy (carries eq & possibly su(s)^{3})
836 T(1;2)N$^{264-10}$ — T(1;2)N$^{264-10}$/FM6, y^{31d} sc^{8} dm B
837 T(1;2)sc^{S2} — T(1;2)sc^{S2}/In(2L+2R)Cy, Cy
838 T(1;2)sc^{19} — T(1;2)sc^{19}/y f:=;fs(2)B sc^{19i} b pr/In(2L+2R)Cy, Cy dp lvI pr

Translocations 1;3

839 T(1;3)263-4 — T(1;3)263-4, y sc B l(In(1)AM
840 T(1;3)143-3 — T(1;3)143-3, ru e^{s} ca/In(3LR)DcxF, ru h ca
* T(1;3)Del-143" — . . . . . (see T(1;3)143-3)
841 T(1;3)N$^{264-6}$ — T(1;3)N$^{264-6}$, y/y w dm (= N^{6})
842 T(1;3)04 — T(1;3)04/ClB
843 T(1;3)05 — T(1;3)05, D/y f:=
844 T(1;3)OR60 — T(1;3)OR60/In(3LR)Ubx^{130}, Ubx^{130} e^{s}female;tra^{D} Sb e/In(3LR) Ubx^{130}, Ubx^{130} e^{s} male
845 T(1;3)ras^{V} — T(1;3)ras^{V}/y f:=
* T(1;3)sc^{J4} — 856
846 T(1;3)sc^{Z} — T(1;3)sc^{Z}/y f:=
847 T(1;3)sc$^{260-15}$ — T(1;3)sc$^{260-15}$/FM6, y^{31d} sc^{8} dm B
848 T(1;3)sta — T(1;3)sta/FM3, y^{31d} sc^{8} dm B l
849 T(1;3)sta — T(1;3)sta/y f:=
850 T(1;3)v — T(1;3)v, v/FM6, y^{31d} sc^{8} dm B
851 T(1;3)w^{vco} — T(1;3)w^{vco}, v f/ClB^{36d}

Translocations 1;3;4

852 — Df(1)sc^{8}, w^{a}/y f:=;T(1;3;4)sc^{J4} ey^{D}

Translocations 1;4

853 T(1;4)B^{S} — T(1;4)B^{S}/y f:=
* T(1;4)N^{8a} — . . . . . (see T(1;4)N$^{264-12}$)
854 T(1;4)N$^{264-12}$ — T(1;4)N$^{264-12}$/FM6, y^{31d} sc^{8} dm B
855 T(1;4)sc^{8} — T(1;4)sc^{8}, B w^{a}/y f:=
856 T(1;4)w^{m5} — T(1;4)w^{m5}/ey^{D}
857 T(1;4)w$^{m258-18}$ — T(1;4)w$^{m258-18}$, y/ci^{D}
858 T(1;4)w$^{m258-21}$ — T(1;4)w$^{m258-21}$/FM1, y^{31d} sc^{8} w^{a} lz^{s} B
859 T(1;4)w$^{m258-21}$ — 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 — 336
860 T(Y;2)B — T(Y;2)B/b; see also 422
* T(Y;2)C — 271, 373
* T(Y;2)E — 203, 266
* T(Y;2)G — 238
* T(Y;2)J — 381
861 T(Y;2)rl — T(Y;2)rl, lt cn/b lt bw

Translocations Y;2;3

* T(Y;2;3)F — 564

Translocations 2;3

862	T(2;3)101	T(2;3)101, al^{2} sp^{2}/In(2L+2R)Cy, Cy L^{4} sp^{2}
863	T(2;3)101	T(2;3)101; ru h e^{4} ro ca/In(3L+3R)P, Dfd ca
864	T(2;3)108	T(2;3)108, al c sp^{2}/In(2L+2R)Cy, al^{2} Cy lt^{3} L^{4} sp^{2}
865	T(2;3)109	T(2;3)109, p^{p}/In(3L+3R)P, Dfd ca
866	T(2;3)A	T(2;3)A, Bl;ru h D TA ss e^{s}/In(3L+3R)P
*	T(2;3)ap^{Xa}	. . . . . (= Xa) . . . . . 458, 543, etc.
*	T(2;3)ap^{Xa}	T(2;3)ap^{Xa}, ca 557, 829
867	T(2;3)ap^{Xa}	T(2;3)ap^{Xa}/l(3)XaR
868	T(2;3)Ata	T(2;3)Ata, Ata/In(2R)Mo^{K}
869	T(2;3)B	T(2;3)B, al sp^{2}/In(2L+2R)Cy, Cy L^{4} sp^{2}
870	T(2;3)B	T(2;3)B;ru h D TB ss e^{s}/In(3L+3R)P
871	T(2;3)bw^{V4}	T(2;3)bw^{V4}/SM1, al^{2} Cy sp^{2}
872	T(2;3)bw^{V5}	T(2;3)bw^{V5}/SM5, al^{2} Cy lt^{v} sp^{2}
873	T(2;3)bw^{VDe3}	T(2;3)bw^{VDe3};Ubx bxd/In(3LR)Cx
874	T(2;3)bw^{VDe4}	T(2;3)bw^{VDe4}/SM5, al^{2} Cy lt^{v} sp^{2}
875	T(2;3)C	T(2;3)C;ru h D TC ss e^{s}/In(3L+3R)P
*	T(2;3)dp	 242
876	T(2;3)dp^{D}	T(2;3)dp^{D}, dp^{D}/SM1, al^{2} Cy sp^{2}
877	T(2;3)E	T(2;3)E/SM5, al^{2} Cy lt^{v} sp^{2}
878	T(2;3)Hm	T(2;3)Hm, Hm/In(2L+2R)Cy, Cy
879	T(2;3)Hn	T(2;3)Hn, Df(3L)Hn, Hn/In(3LR)Ubx^{130}, Ubx^{130} e^{s}
*	T(2;3)Me	. . . . . 533, 534, etc.
*	T(2;3)P	T(2;3)P, P 675, 692
*	T(2;3)p^{Gr}	. . . . . (see T(2;3)Pu^{Gr})
880	T(2;3)Pu^{4}	T(2;3)Pu^{4}, Pu^{4}/C(3)x
881	T(2;3)Pu^{Gr}	T(2;3)Pu^{Gr}, Pu^{Gr}/SM1, al^{2} Cy sp^{2}
882	T(2;3)rn	T(2;3)rn/In(2R)Cy
883	T(2;3)Dp-S	T(2;3)Dp-S, ho/In(2L+2R)Cy, Cy E(S) (homozygous viable)
884	T(2;3)S^{L}	T(2;3)S^{L}/In(2L+2R)Cy, Cy E(S)
*	T(2;3)S^{M}	 440
885	T(2;3)Sb^{V}	T(2;3)Sb^{V}, Sb^{V}, In(3R)Mo/TM3, y^{+} ac^{+} ri pP sep bx^{34e} e^{s}
886	T(2;3)Sb^{V}	T(2;3)Sb^{V}, Sb^{V}, In(3R)Mo, In(3LR)P35/Sm1, al^{2} Cy sp^{2};In(3LR) Ubx^{130} Ubx^{130} e^{s}
*	T(2;3)Xa	. . . . . (see T(2;3)ap^{Xa})

Translocations 2;4

887	T(2;4)a	T(2;4)a/In(2L+2R)Cy, Cy pr;ey^{2}
888	T(2;4)ast^{v}	T(2;4)ast^{v}/In(2L+2R)Cy, al^{2} Cy lt^{3} L^{4} sp^{2}
889	T(2;4)b	T(2;4)b/In(2L+2R)Cy, Cy pr;ey^{2}
890	T(2;4)d	T(2;4)d, al dp px sp/In(2L+2R)Cy, Cy pr;ey^{2}
891	T(2;4)d	T(2;4)d/In(2L+2R)Cy, Cy pr

Translocations 3;4

892	T(3;4)A2	T(3;4)A2/In(3L)P, Me ca
893	T(3;4)A12	T(3;4)A12/In(3LR)Cx, D
894	T(3;4)A13	T(3;4)A13, ve ca/In(3L)P, Me ca
895	T(3;4)A28	T(3;4)A28, ve ca (homozygous)
896	T(3;4)c	T(3;4)c/In(3L+3R)P, Dfd ca
897	T(3;4)e	T(3;4)e/In(3LR)Ubx^{130}, Ubx^{130} e^{s}
898	T(3;4)e	T(3;4)e, h th st cu sr e^{s} ca/In(3L+3R)P, Dfd ca
899	T(3;4)f	T(3;4)f/In(3L)P, Me
900	T(3;4)f	T(3;4)f, h th st cu sr e^{s} ca/In(3L+3R)P, Dfd ca

Transpositions

901	Tp(3)bxd^{100}	Tp(3)bxd^{100}, ri/T(2;3)Me
902	Tp(3)bxd^{107}	Tp(3)bxd^{107}, bx bxd^{107} sr e^{s}/bx^{34e} Mc
903	Tp(3)Vno	Tp(3)Vno/H^{2}

APPENDIX (Recent additions to Pasadena Stock List)

23a carl(1)su-f^{ts67g}
47a f^{s} 1(1)su-f^{ts67g}
47b f^{u} 1(1)su-f^{ts67g}/FM4, y^{31d} sc^{8} dm B
62a 1(1)mys/FM4, y^{31d} sc^{8} dm B
62b 1(1)mys^{ts2}, 1(1)su-f^{ts67g}/y f:=
79a oc ptg^{3} 1(1)TW-1^{cs}/FM7c, y^{31d} sc^{8} w^{a} sn^{x2} v B 1(1)TW-9;DTS-91/In(2LR)O, dp^{lvI} Cy pr cn^{2}
79b oc ptg^{3} 1(1)TW-1^{cs}/FM7c, y^{31d} sc^{8} w^{a} sn^{x2} v B 1(1)TW-24;DTS-91/In(2LR)O, dp^{lvI} Cy pr cn^{2}
80a Oce
104a sc ec cv ct^{6} v g^{2} f/FM7c, y^{31d} sc^{8} w^{a} sn^{x2} v B 1(1)TW-9 - larval lethal
104b sc ec cv ct^{6} v g^{2} f/FM7c, y^{31d} sc^{8} w^{a} sn^{x2} v B 1(1)TW-24 - embry. lethal
240a b Tft/In(2LR)O, dp^{lvI} Cy pr cn^{2} (DTS-513)
240b b Tft/In(2LR)O, dp^{lvI} Cy pr cn^{2} (DTS-100)
294a DTS-91/In(2LR)O, dp^{lvI} Cy pr cn^{2}
354a Ms bw/In(2LR)O, dp^{lvL} Cy pr cn^{2}, bw & Fs/In(2LR)O, dp^{lvI} Cy pr cn^{2}, bw
607a su-pr^{e3};pr cn
607b su-pr^{e4}/TM3;pr cn
608a su-Hw^{69k}/Ubx^{130}, Ubx^{130};sc cv ct^{6} v
608b su-Hw^{70} e^{s}/Ubx^{130}, Ubx^{130};sc cv ct^{6} v
669a v;rs^{2}
716a R(1)w^{vc}/d1-49, 1(1), y w lz^{s} f^{36a}/sc^{8} Y
789a In(1)FM7, y^{31d} w^{a} sn^{x2} v^{of} B/y f:=/Y^{bb74}
866a T(2;3)$Antp^{Yu}$-In(2LR)SM1 al^{2} Cy sp^{2} cn^{2};Sb

PHILADELPHIA, PENNSYLVANIA: UNIVERSITY OF PENNSYLVANIA
School of Veterinary Medicine

Deficiencies-X

1	Df(3C1)w^{258-45}	w^{258-45}/Ins(1)sc^{S1}, S, sc^{8}, sc^{S1} sc^{8} w^{a} B
2	Df(3C2)w^{m4L} w^{mJr}	Ins(1)w^{m4L} w^{mJR}, w/Y/y w f:=
3	Df(3C3)w^{mJL} rst^{3R}	Ins(1)w^{mJL} rst^{3R}/FM6
4	Df(3C2.3)w^{m4L} rst^{3R}	Ins(1)w^{m4L} rst^{3R}, w/FM6
5	Df(3C4)w^{m5L} $w^{258-18R}$	Ts(1;4)w^{m5L} $w^{258-18R}$, w^{m} Bx/FM6
6	Df(3C5.6)$w^{258-18L}$ N^{8aR}	Ts(1;4)$w^{258-18L}$ N^{8aR}, y w^{m} N Bx/FM6, y^{31d} sc^{8} dm B
7	Df(bb)w^{m4L} $w^{m51b19R}$	Ins(1)w^{m4L} $w^{m51b19R}$/Y/y w f:=

Duplications-X

8	Dp(1;1)3C2, w^{mJL} w^{m4R}	Ins(1)w^{mJL} w^{m4R}, car f/Y/y w f:=
9	Dp(1;1)3C2, w^{mJL} $w^{m51b19R}$	Ins(1)w^{mJL} $w^{m51b19R}$, car f/Y/y w f:=
10	Dp(1;1)3C4, $w^{258-18L}$ w^{m5R}	Ts(1;4)$w^{258-18L}$ w^{m5R}, y w^{m} Bx/FM6:=
11	Dp(1;1)3C5.6, N^{8aL} $w^{258-18R}$	Ts(1;4)N^{8aL} $w^{258-18R}$, Bx/FM6:=
12	Dp(1;1)w^{rG}	Dp(1;1)w^{rG}, y ac z/Y/y f:=
13	Dp(1;1)w^{rG2}	Dp(1;1)w^{rG2}, y ac z/Y/y f:=
14	Dp(1;1)z^{59d}	Dp(1;1)z^{59d}/Y/y f:=

Inversions

15	In(1)w^{m51b19}	homozygous
16	In(3LR)264-100r27	y w; Dp(1;3)$w^{m264-100a}$In(3LR)r27/+

Translocations

17	T(1;4)20G1	T(1;4)20G1, fa sp1 m B/Y/y w f:=
18	T(1;4)N^{8a}	T(1;4)N^{8a}, w rst^{m} N/FM6, y^{31d} sc^{8} dm B
19	T(1;4)w^{m5}	homozygous
20	Ts(1;4)$20G1^{L}$ w^{m5R}	Ts(1;4)$20G1^{L}$ w^{m5R}, Bx/Y/y w f:=
21	Ts(1;4)w^{m5L} $20G1^{R}$	Ts(1;4)w^{m5L} $20G1^{R}$, w^{m}/FM6

BASEL, SWITZERLAND: BIOZENTRUM DER UNIVERSITÄT BASEL

Chromosome 1

7 sn^3 dor/ClB
46 $l(1)El2^{ts}$
92 $l(1)El2^{ts}$/ClB
19 y mal^{f3}
11 y sn^3
94 y w f^{36a}
41 ♂ Y^S X Y^L, In(1)EN;Y^S B f v Y^L/0 ♀ C(1)RM, y v bb/o
54 ♂ y^2 sc w^a w ♀ y f:=

Chromosome 2

55 da pr cn/SM5
61 fs(2)B Alu lt/SM5
40 $Df(2)vg^D$/Cy L^4 sp^2
50 L^2/Cy sp^2
35 L^2 If
43 L^2 If 1(2)gl/SM5

Chromosome 3

1 $Antp^B$ ss^a/TM1, Me(ri^2)sbd^1
56 DTS-4/TM3, Sb Ser
12 Gl/TM1
5 In(3)$Antp^B$ ss^a/LVM
32 In(3LR)C269, mwh e
33 In(3LR)C269, Ns/TM1
62 1(3)e20 mwh red e/TM1
63 1(3)f26 mwh red e/TM1
64 1(3)g30 mwh red e/TM1
65 1(3)k43 mwh red e/TM1
66 1(3)2004 mwh red e/TM1
67 1(3)II B 1 mwh red e/TM1
68 1(3)XII10 mwh red e/TM1
69 1(3)XIY-3 mwh red e/TM1
93 mwh e
2 mwh ss^{a-68} e/TM1
4 Ns
15 p^p Ki e
36 Pr^k Dr/In(3L)+(3R)P
25 ry^2
6 ss^a
26 TM1/TM3

Multichromosomal

21 mal^1;st
3 T(2;3)Hm/Cy
9 y;1(2)gl/SM5 (a px or ?)
22 y;1(4)29^b/ci^d
23 y;1(4)39/1(4)29^b
20 y mal^{f3};st
10 y sn^3;mwh
8 y sn^3;T(1;3)sc^{j4}

SYDNEY, AUSTRALIA: UNIVERSITY OF SYDNEY
Department of Animal Husbandry

Wild Stocks

5 strains from N.S.W. and Victoria

Chromosome 1

ct^6 t^3 dy
cx
cx kz
dy
ec
ec Oce
In rst^3
Oce
Oce cv wy^2 f
t^3
w
w^{bl}
y
y cv f
y cv wy^2 f
y cx wy^2 f
y ec Oce wy^2 f
y kz cx wy^2 f
y w
y ct^6 & y f:=
m^D/FM3

Chromosome 2

b j
net
vg

Chromosome 3

e^{11}
e wo ro gl

SPAIN, SANTIAGO DE COMPOSTELA: UNIVERSIDAD DE SANTIAGO
Facultad de Ciencias, Departamento de Genética

Wild Stocks

Oregon
Valencia
Rojos
Portomouro
Cumbraos
Goyas
Rivadulla

Chromosome 1

w
car
v
sn^{qr}
sn^3
sn^{36a}
sn^{34e}
w^{co}, sn^2
y, w^a, cv, sn^{qr}

Chromosome 2

α-$Gpdh^S$
α-$Gpdh^F$
Adh^S
Adh^F
dp
vg
cn
bri
bw
cn^{rbr}
vg, cn^{rbr}
b, cn, vg

Chromosome 3

e
tx
ri se
Est-6^S
Est-6^F
Est-C^F

Chromosome 4

ci^D/ey^D

Inversion-X

Muller-5

Attached-X

- y f

Multichromosomal Inversions

bw^{V1}, dp, b/In(2L+2R)Cy, Cy sp^2;Sb/In(3LR)DcxF

Special Stocks

α-$Gpdh^S$, Adh^S/α-$Gpdh^S$, Adh^S
α-$Gpdh^S$, Adh^F/α-Gdh^S, Adh^F
α-$Gpdh^F$, Adh^S/α-$Gpdh^F$, Adh^S
α-$Gpdh^F$, Adh^F/α-$Gpdh^F$, Adh^F

NAGASAKI, JAPAN: NAGASAKI UNIVERSITY SCHOOL OF MEDICINE
Department of Genetics

Wild Stocks	Chromosome 1	Chromosome 2	Chromosome 3
Canton-S	sc^{S1} B InS w^a sc^8	b	e^{11}
Erie, Pa. (isogenic)	sc^8.Y/In(1)dl-49, y B	cn bw	Sb/Ubx^{130} e^s
Ockley	& y f:=	$Df(s)bw^5$ sp^2/Xa	
Oregon-RS	w	Ins SM1, al^2 Cy sp^2/dp	Chromosome 4
Tanegashima	y	b Pm ds^{33k}	
	y w m f	vg	pol

Multichromosomal

Ins SM1, al^2 Cy sp /dp b Pm ds^{33k};Sb/Ubx^{130} e^s;pol
sc^{S1} B Ins w^a sc^8;Ins SM1, al^2 Cy sp^2/dp b Pm ds^{33k};Sb/Ubx^{130} e^s;pol
y sc^{S1} In49 sc^8;bw;st p^p

X-chromosome Lethals

X-ray-, neutron-indiced & spontaneous lethals, about 1200 strains

Chromosome 2 Lethals

X-ray-induced & spontaneous lethals, about 80 strains

CALCUTTA, INDIA: UNIVERSITY OF CALCUTTA
Department of Zoology, Genetics Research Unit

Wild Stock

a2 Oregon R

Chromosome 1

b1 B
b2 car bb
b3 cm
b4 cm ct^6
b5 ct
b6 f^{36a} odsy f^{+ih} & y f:=
b7 ras^4 m/ClB
b8 w^e sn/ClB
b9 rb
b10 svr
b11 w
b12 w^{ch} wy
b13 y
b14 y ac sn^3 sx vb^2 sy/y sc^1 In dl49 B w^a sc^8
b15 y sc pn w rb cm ct^6 sn^3 ras^2 v g^2 car/sc^{S1} car f In49 v (bleached)
b16 y v g f
b17 y w sn^3
b18 z w^{11E4}

Chromosome 2

d1 b
d2 b cn beta
d3 b vg
d4 bw
d5 cg c/U
d6 cn bw
d7 Cy/Pm
d8 dp^T SP cn InNSR mr/Cy
d9 fj wt/SM5, al^2 Cy lt^V sp^2
d10 Gla, InLR/S^2 Cy cn^2 bw sp
d11 InNSL InNSR/al^2 Cy, InL lt^3 L^2
d12 net
d13 vg

Chromosome 3

e1 e^s
e2 H^2/Xa
e3 red
34 red e
e5 ru e ca

Multichromosomal

g1 dp^T Sp on InNSR mr/Cy; red e
g2 vg;e^s
g3 y ac sn^3;Cy/Pm
g4 cm;st
g5 y;Cy/Pm

Translocations

h1 T(1;3)sta/FM3, y^{31d} sc^8 dm B
h2 T(1;3)sc^{260-15}/FM6, y^{31d} sl^8 dm B
h3 T(1;3)05, D/y f:=
h4 T(1;3)sc^2/y f:=
h5 T(1;3)sta/y f:=

Altered-Y & Attached-XY

j1 (Maxy) 1^{j1+} $Y/1^{J1}$ $sc^{J1(+)}$ In49 ptg oc B^{M1}/y sc^{S1} car odsy f g^2 dy v ras^2 sn^3 ct^6 cm rb ec w pn 1 sc^8
j2 f^{36a} odsy f^{+ih}/y f:=
j3 ras^4 m/y f:=
j4 w^{bf3}/ sn^{36a}
j5 y/g^2 ty

Duplications

k1 $D_p(1;3)sc^{j4}$ i

BIRMINGHAM, ENGLAND: THE UNIVERSITY OF BIRMINGHAM
Department of Genetics

Delete: Multichromosomal v;cn from list in DIS 46:34; otherwise list remains the same.

NORWICH, ENGLAND: JOHN INNES INSTITUTE

Add: Chromosome 1 w sn B to list in DIS 50:17; otherwise list remains the same.

MELBOURNE, AUSTRALIA: UNIVERSITY OF MELBOURNE
Department of Genetics

Wild Stocks

601 Local Melbourne strain
602 MEL (Canton-S)

Chromosome 1

101 w
102 w^{58c}
103 w^{e-2}
104 w^{sat}
105 w^{a55k}
106 v
107 w ct f
109 In(1)sc^7, sc^7
110 In(1)sc^8, sc^8
112 y^2 w^a
117 ct v f
121 Basc
122 In(1)$sc^{S1.L}$ In(1)S w^a $sc^{8.R}$
127 v/Y^{bb}
128 "sz" y(X.Y^S/Y^{Lc})
131 In(1)sc^8, y^{31d} sc^8 w^a
132 In(1)sc^8, sc^8 v f car
143 y, B/Y
147 y/B^sYy^+/R(1)X^{c2}, y B

Chromosome 2

201 cn
202 dp
205 bw
206 vg
209 cn bw
211 vg pr
212 fj wt/ap^{Xa} (T 2;3)
215 S/In(2R)Cy/Cn L^4 sp^2
216 Df(s)Px^2/Cy L^4 sp^2
217 1(2)gl cn bw/Cy al lt^2 L^4 sp^2
218 1(2)gl cn bw/SM5, al^2 Cy lt^v sp^2

Chromosome 3

301 se
303 ss
304 st
306 e^4
307 e
308 ss e ro-3
310 h p^p e^s
313 Dl^3/In(3r)C, e
316 Sb Ubx/ap^{Xa} (T 2;3)

Chromosome 4

401 ci
402 ey^2
404 ci^D/ey^D

Multichromosomal

504 bw;e
505 vg;se
506 vg;se
508 v;bw;e
509 y^2 w^a;bw;st
510 "sz" bw;st(X.Y^S/Y^{Lc})
511 "sz" vg;se(X.Y^S/Y^{Lc})
512 bw;st

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

Wild Stock

Wellington mass transfer

Chromosome 1

y
w
w^a

Chromosome 2

m
B
v
w m B

vg
bw
dp

vg bw
cn bw
dp b cn bw
Cy

Chromosome 3

e
ve
se

Multichromosomal

vg;se
bw;e
bw;ve
bw;st
dp;se
dp;e
lz^3;ey^R

SEOUL, KOREA: SEOUL NATIONAL UNIVERSITY
Department of Zoology

Delete: stocks numbered 003, 101, 102, 104, 105, 106, 204, 207, 209, 401, 503
Add: some inbred wild Korean strains, 304 se, 402 ey, 504 Muller-5;Sy/bw^{V1};Sb/Ubx (1;2;3)
Otherwise list stands as in DIS 46:15 (except for the addition of 210 Srf/bw^{V1} in DIS 48:15).

TAIPEI, TAIWAN, REPUBLIC OF CHINA: ACADEMIA SINICA
Institute of Zoology

Wild Stocks

Oregon-R
Oregon-R (inbred)
Samarkand
Taiwan (19 stocks)

Chromosome 1

B
y
w
sc cv v f

Chromosome 2

cl
cn bw
b
bw
vg

Chromosome 3

p

Multichromosomal

bw;h (2;3)
Cy/Pm;Sb/D (2;3)

Attached-X

y/g^2 ty
oc/g f:=

Inversion

oc ptg^3/In(1)ClB

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

Note: + means al^2 Cy lt^v sp^2

Wild Stocks

a1 Oregon K
a2 Suchumi
a3 Novosibirsk
a4 Krnov 65
a5 Lednice 71
a6 Šakvice 71
a7 Samarkand
a8 Oregon R
a9 Crkwenica
a10 Oregon R-C
a11 Lausanne S
a12 Moravský Písek

Chromosome 1

b1 w
b2 Muller-5
b3 w^a

Chromosome 2

c1 dp
c2 cn
c3 bw
c4 cn vg/Oregon K
c5 cn vg/Suchumi
c6 cn vg/Samarkand
c7 dp b cn bw
c8 Cy/Al b pr lt ltd cn a px pd bw
c9 cn l (2) cr c/SM5,+
c10 al dp b bw l (2) ax/SM5,+
c11 S Sp Bl bw^D/al^2 CyO p^3 L^4 sp^2
c12 Tft L/SM5,+
c15 Bl L^2/SM5,+
c17 Sp J L^2 Pin/SM5,+
c18 Sp rc^2/Cy Bl L
c19 b cn
c20 cn bw
c21 vg

Chromosome 3

d1 e

Chromosome 4

e1 pol

Multichromosomal

f1 M-5;SM5 +/L;Me Sb e/H e (1;2;3)
f2 sc^{S1} Ins w^a sc^8;SM5/Bla;Sb/TM3 (1;2;3)
g1 SM5;+/L;Me Sb e/H e (2;3)
g2 bw;e (2;3)
h1 bw;e;pol (2;3;4)

D. MELANOGASTER - NEW MUTANTS

Report of J.L. Farmer
Brigham Young University, Provo, Utah

w^{coJ}: white-coral J 1-1.5. EMS induced. Indistinguishable from w^{co} phenotypically or by chromatography of eye pigments. Unlike w^{co} it is suppressed by a gene which is present in many laboratory stocks.

Su(w^{coJ}): Suppressor of white-coral J 2-approx. 105. Present in majority of common laboratory stocks tested, including w^{co}. Suppresses w^{coJ} but not w^{co}. Very sensitive to genetic background, restoring drosopterins from a few percent to more than 100 percent of wild-type. RK1 in good genetic background. w^{coJ} flies heterozygous for Su(w^{coJ}) make about 65% as much drosopterins as those homozygous for Su(w^{coJ}). Dominant, homozygous viable, fecundity and viability excellent. Closely linked to Su(bw^{Vl}), not known if it is allelic. Su(w^{coJ}) does not suppress bw^{Vl}. Su(bw^{Vl})/Cy cn vg bw^{Vl} carries Su(w^{coJ}) linked to Su(bw^{Vl}). (Su(bw^{Vl}) is homozygous lethal, contrary to Lindsley and Grell.)

Report of L. Craymer
University of Wisconsin, Madison, Wisconsin

C(1)M4: Compound(1) Multiple 4 72e. An acrocentric compound X of constitution C(1)M4, In(1)w^{m4}+AB——In(1)FM7, y^-, produced by X-ray induced exchange between the proximal heterochromatin of In(1)w^{m4}+AB, y w^{m4}, and the distal heterochromatin of In(1)FM7, y^{31d} w^a v^{0f} (B reverted by unequal crossing over). In the occasional salivary nucleus in which C(1)M4 synapses, a complex figure with the eight breaks - 1B, 3C, 4D, 9F, 11F, 13F, and 20 - characteristic of the w^{m4}+AB+FM7 inversions is to be seen. Metaphase figures show five 'autosomes', and anaphase figures indicate that one of these is acrocentric. C(1)M4 is marked with y, w^a/w^{m4}, and was originally bb, but this last has since disappeared. The w^a/w^{m4} makes C(1)M4 an excellent balancer for attached XY's: strong variegation occurs in the absence of Y chromatin, moderate variegation occurs with Y^S, and almost complete suppression occurs with Y^L or a complete Y. Frequency of spontaneous triploidy is about 1/10,000; spontaneous detachment frequency is about 1/15,000. C(1)M4, y, is a powerful enhancer of autosomal recombination, but has low viability with autosomal rearrangements. A C(1)M4, y^2 stock exists which has good viability under these conditions; unfortunately the C(1)M4 in the triploid stock through which this derivation was made had become bb^-, and this suffers the same defect.

$y^+Y^SX.$, In(1)FM7, y^- w^a v^{0f} 73c. X-ray induced detachment of C(1)M4, y, with y^+YB^S.

$Y^SX.Y^LB^S$, In(1)EN, y 73l. Recombinant from $Y^SX.Y^L$, In(1)EN, y/T(Y:2)J146 male.

$y^+Y^SX.Y^LB^S$, In(1)FM7^L+EN^R, y v^{0f} 74d. Recombinant from $y^+Y^SX.$, In(1)FM7, y^- w^a v^{0f}/ $Y^SX.Y^LB^S$, In(1)EN, y. Salivaries of heterozygotes with Canton S show the sc^{8L}+EN^R, dl-49, and 15DE:20 inversions. Metaphase figures are consistent with a $Y^SX.Y^L$. This was built to provide a more flexible replacement for the dow B^{Ml}/Binsn mutation accumulation system, but should also be a good balancer for temperature-sensitives, meiotics, and other mutants for which homozygosis is undesirable. The FM7 inversions serve effectively to prevent euchromatic recombination, while the y^+ and B^S serve to follow recombinational events in heterochromatin.

Report of E.A.F. Gateff
Biologisches Institut I, Freiburg, West Germany

l(1)mbn: lethal (1) malignant blood neoplasm EMS induced. Not located. Lethal at the time of puparium formation. The haemolymph of the l(1)mbn hemizygous male larvae exhibits many more free haemocytes than the wild type haemolymph. Plasmatocytes, lamellocytes and crystal cells are the predominant cell types found in the l(1)mbn haemolymph. They participate in nodule formation and melanization of the l(1)mbn tissues during the third larval instar. The plasmatocytes show furthermore, invasiveness into the imaginal discs. Neoplastic cell masses located in the 5th, 6th and 7th segments are the most pronounced feature of the mutant. These neoplastic cell masses are constituted primarily of prohaematocytes and plasmatocytes. When pieces of these neoplasms are implanted into the body cavity of female adults, they grow rapidly and kill the host in 10 - 14 days. The cell types found in the transplanted neoplasm are the prohaematocytes and plasmatocytes. They show a high degree of malignancy. Not only do they fill the body cavity of the host, they invade also the ovaries and the single ovarioles. Thus, the l(1)mbn gene cuases the neoplastic transformation of the prohaematocytes and the plasmatocytes in the larva.

Report of W.K. Baker
University of Chicago, Chicago, Illinois

Antp^{72j}: Antennapedia-72j 3 Baker 72j26. Spontaneous in a Opt^G;mwh jv;ey^2 strain. Varies in expression from complete mesothoracic leg with sternopleura, to slight modification of antenna. Homozygous viable and thus like Nasobemia. Salivary chromosomes appear normal. Presumed allele of Antp.

Report of A.J. Gallo and V.M. Salceda
Colegio de Postgraduados ENA, Chapingo, Mexico

Mex. 156: A sex limited lethal gene A sample of 179 males, recently captured in nature, was analyzed for the presence of lethal genes in the X, II and III chromosomes. From the test for lethals on the sex chromosome, using the M-5 technique, was recovered one lethal that affects only the females, since the class +/+ did not appear. This lethal gene limited to females, is the second reported in the literature, the other having been reported by Drescher in 1964 (Amer. Nat. XCVIII No. 900:167-171). The number of flies in the test that allowed us to classify it as sex-limited are as follows: M-5/Y 177; +/Y 224; M-5/+ 204; +/+ none. The frequency of this gene in the population is 0.55%. With the only kind of females and normal males a stock was started and named Mex 156. Experiments tending to better information on this mutant will be initiated in the future.

Report of G.J. Thalmann
Dowling College, Oakdale, New York

E(A): Enhancer of Abnormal abdomen (A^{53g}) 2-$6^{\pm}$ (between al and dp). This enhancer, which has the ability to cause extreme intensification of the A^{53g} phenotype, was discovered in an inbred Notch deficiency stock, $Df(1)N^{45e}$. Flies carrying both A^{53g} and the enhancer are almost entirely denuded of tergites. The stock in which the enhancer was discovered has no greater incidence of abdominal irregularities than Ore-R. Enhancer Star, located in the same region, does not enhance expression of A^{53g} and E(A) does not enhance expression of Star.

Report of G. Lefevre, Jr.
State University, Northridge, California

fa^{1N118}: facet-lethal-N118 1-3.0. A cytologically normal, X-ray-induced male-lethal allele at the Notch locus. Heterozygous females are nearly wild-type, but show occasional slight traces of the N phenotype. The mutant fully complements with spl, but interacts with fa^{g}, showing rough, irregular eyes. It is covered by Dp51b7, Dp N^{st}, and $w^{+}Y$. It survives in heterozygous combination with the male-lethal Ax allele described below.

Ax^{J14}: Abruptex-J14 1-3.0. A cytologically normal, X-ray-induced male-lethal allele at the Notch locus. It expresses the usual Ax phenotype. It is covered by Dp51b7 and Dp N^{st}, and the covered males show the Ax phenotype. But when covered by $w^{+}Y$, the males have normal wing veins, though they lack ocellar bristles. When heterozygous with fa^{1N118}, described above, females survive, exhibiting a strong Ax phenotype as well as rough eyes.

Report of E. Valadé
Universidad de Santiago, Santiago de Compostela, Spain

cn^{rbr}: rojo brillante Chromosome 2. Recessive. Of cn complex. Spontaneous from a vg stock. Eye color brighter than cinnabar. Ocelli colorless. Eye color darkens with age. Spontaneously reverts to wild color in high frequency ($3x10^{-3}$). Abnormal segregation with excess of normals.

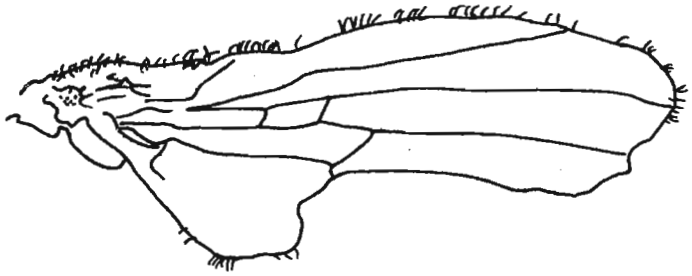
Report of J. Erickson
Western Washington State College, Bellingham, Washington

w^{sey}: white sepia-yellow Spontaneous mutant in sepia stock. With sepia, a bright lemon-yellow. Without sepia, off-white, possibly a spontaneous w^{1} (white ivory). Will be held a year after appearance of this report.

Report of P. Simpson and H.A. Schneiderman
University of California, Irvine, California

sd^{ts}: scalloped temperature sensitive 1-50.5. EMS induced in sn^3 stock. Wings vestigial-like at 29°C, also capitellum of haltere extremely reduced. At 22°C wings have parts of wing blade and/or wing margin missing, halteres normal. Viability and fertility good at 22°C.

22°



29°

DROSOPHILA SPECIES - STOCKS

Calcutta, India: University of Calcutta
Department of Zoology, Genetics Research Unit

ananassae hydei simulans (1) simulans net pseudoobscura

TAIPEI, TAIWAN, REPUBLIC OF CHINA: ACADEMIA SINICA
Institute of Zoology

Drosophila

D. albomicans (113 stocks)
D. annulipes
D. formosana (7 stocks)
D. hypocausta
D. immigrans (12 stocks)
D. (Chaetodro.) quadrilineata (4 stocks)
D. ruberrima (14 stocks)
D. sulfurigasta albostrigata (2 stocks)
D. tongpua (4 stocks)
D. xanthogaster (2 stocks)
D. asper (5 stocks)
D. sp. of daruma-like (6 stocks)
D. hydei (5 stocks)
D. lacertosa
D. repleta (2 stocks)
D. sp. of repleta group
D. sp. of robusta group (5 stocks)

Sophophora

D. ananassae (15 stocks)
D. auraria
D. bipectinata (33 stocks)
D. sp. of ficusphila-like (2 stocks)
D. kikkawai
dark form (6 stocks)
light form (27 stocks)
D. lini (2 stocks)
D. lucipennis (5 stocks)
D. malerkotliana (9 stocks)
D. sp. of montium group (6 stocks)
D. sp. of pennae-like (2 stocks)
D. prostipennis (5 stocks)
D. quadraria (2 stocks)
D. sp. of rufa-like (3 stocks)
D. sp. of suzukii subgroup (4 stocks)
D. takahashii (2 stocks)
D. trilutea (2 stocks)

Dorsilopha

D. busckii (3 stocks)

Pholadoris

D. sp.

Scaptodrosophila

D. sp.

Others

Scaptomyza elmoi
Dettopsomyia nigrovittata (5 stocks)
Chymomyza sp. (10 stocks)
Liodrosophila spp. (2 stocks)

Note: Anyone who needs Drosophila species stocks from Taiwan, please write to: Fei-Jann Lin, Institute of Zoology, Academia Sinica, Nankang, Taipei, Taiwan 115, Republic of China.

SANTIAGO DE COMPOSTELA, SPAIN: UNIVERSIDAD DE SANTIAGO
Facultad de Ciencias, Departamento de Genética

subobscura

Wild Stocks		Special Stocks				
Ons	Cumbraos	K 228	: Est-2^{100}	; Est-$4^{0.91}$;	Est-5^{100}	
Goyas	Rivadulla	S 35	: Est-2^{130}	; Est-4^{100} ;	Est-5^{S}	
Padrón	Couso	21-1-3	: Est-$2^{1.65}$;	Est-4^{100} ;	Est-5^{S}	; Est-6^{100}
Portomouro	Rojos	7	:	Est-$4^{0.89}$;	Est-5^{S}	; Est-6^{102}

Other Species
(Maintained in the laboratory as natural populations)

phalerata	repleta	immigrans	simulans
funebris	hydei	busckii	

MELBOURNE, AUSTRALIA: UNIVERSITY OF MELBOURNE
Department of Genetics

simulans	immigrans	serrata	pseudoobscura
701 SIM + S 51	703 IMM + C 60	705 SER + Q 59	713 Standard, Mather California
			714 Chiricahua, Mather California
hydei	**funebris**	**persimilis**	
702 HYD + S 51	704 FUN + C51	707 D1;or;Cy	

LEEDS, ENGLAND: UNIVERSITY OF LEEDS
Department of Pure and Applied Zoology

ambigua	confusa	phalerata	subobscura
bifasciata	obscura	pseudoobscura	transversa
cameraria			

EASTON, PENNSYLVANIA: LAFAYETTE COLLEGE
Department of Biology

mojavensis	mulleri	pseudoobscura	virilis

SYDNEY, AUSTRALIA: UNIVERSITY OF SYDNEY
Department of Animal Husbandry

simulans	st e	net b py sd pm	ananassae - Wild (Rockhampton)
	$y\ w$	st Ubx pe/st pe	buzzatii
net	f^2		hydei
jv se	v	Other Species	serrata
jv st pe	bw		simulans
H^h pe	dh b pm	aldrichi	wheeleri

simulans

Report of J.L. Agnew
University of the Witwatersrand, Johannesburg, South Africa

rp: rotated penis Agnew 74b.
Spontaneous in progeny of wild female from Mapumulo, Kwa-Zulu. ♂ genitalia rotated thru 180°; male sterile. Apparently same as rp in melanogaster.

Photo: left, non-mutant sib
right, rp

D. SPECIES - LINKAGE DATA

Report of D. Yardley
University of Georgia, Athens, Georgia

Linkage analysis of the α-amylase locus in pseudoobscura. Prakash and Lewontin (1968) placed the α-amylase locus (Amy-1) on the third chromosome. I have determined its map position by use of two stocks fixed for the Standard gene arrangement. Stock 86 is homozygous for the Amy-$1^{\cdot 84}$ allele and for the recessive eye color mutants orange (or) and purple (pr), which together produce white eye color. Stock 295 is fixed for the corresponding wild type alleles at the eye color loci and for Amy-$1^{1.00}$. Three replicate crosses of stock 86 females and stock 295 males, and three replicates of the reciprocal cross were made. F_1 females were then testcrossed to stock 86 males and their offspring scored for eye color and amylase. Amylase was scored electrophoretically by a method similar to that employed by Prakash and Lewontin (1968).

Examination of 1,240 flies gave the following results: single crossovers between or and Amy-1, 347; single crossovers between pr and Amy-1, 164; double crossovers, 62; and non-recombinants, 667. This places the Amy-1 locus at map position 33, 33 map units from or and 18 map units from pr. Our results are consistant with those of Tan (1936) in placing the or and pr loci about 50 map units apart. Tan (1937) placed Sc, whose map position is 29, in band 73 of the cytological map. The Amy-1 locus is probably somewhere within bands 73-74-75 and is thus included in the majority of third chromosome gene arrangements.

References: Prakash, S. and R. Lewontin 1968, P.N.A.S. 59:398-405; Tan, C.C. 1936, Genetics 21:796-807; Tan, C.C. 1937, Z.F. Zellforschung U. Mikr. Anatomie. Bd 26.

This work was supported by U.S.P.H.S. Grant 07180.

Kuzin, B.A., A.A. Aronstam, L.I. Korochkin. Institute of Cytology and Genetics, Siberian Department of Academy of Science, Novosibirsk, USSR. Studies on determination process of the expression of gene Est-6 in genitals of D. melanogaster males.

It has been shown that sexual dimorphism with respect to Esterase-6 (gene Est-6 -3,36.8), whose activity in males surpasses by twice that in females, is due to the high concentration of the enzyme in the male ejaculatory duct. Esterase-6 is observed in the duct of the male only 20 hours after emergence; in subsequent hours the activity of the enzyme rises, attaining the maximum 48 hours after emergence.

To determine the time of the appearance of cells capable of subsequent synthesis of Esterase-6, genital imaginal discs extracted from ♂♂ larvae of various age were transplanted to ♀♀ larvae just before pupation. Analysis of esterase activity in transplants has indicated that cells in which Esterase-6 is to be synthesized appear 8-10 hours after the beginning of third larval stage, i.e., long before differentiation starts.

The relation between the determination of the expression of the gene Est-6, its transcription and translation was assessed by studying the effect of the inhibitors of the synthesis of RNA (actinomycin D) and protein (cycloheximide) on the genitals of males of different ages. It has been demonstrated that the transcription and translation of the gene Est-6 occurs after metamorphosis during the first hours after the emergence of males.

Thus, there is a large interval between the appearance of cells capable of esterase synthesis and the time of expression of the structural gene controlling this enzyme. The data obtained suggest that cell determination may be due not to the accumulation of "long-living" templates (m-RNA), but rather with functional changes of DNP which may replicate during cell proliferation.

Clark, A.M. Flinders University of South Australia, Adelaide. Low temperature shock and the segregation of iso-second chromosomes in females.

Low temperature shock to aged females leads to a small but significant increase in the frequency of non-segregation of compound second chromosomes in female Drosophila melanogaster. Females of the genotype C(2L)RM, j^{63};C(2R)RM, px were exposed to the temperature shock, and then mated to C(2L)RM, b pr;C(2R)RM, vg males. Except for the period of temperature shock, all cultures were maintained at 25°C. The first brood interval was 24 hours, later broods being separated by 48 hours.

Treatment	Brood	% Disomic eggs	% Nullosomic eggs	Total progeny
Controls	1	0.43	0.34	1169
	2	0.49	0.38	3442
	3	0.37	0.40	3001
	4	0.41	0.27	3637
Newly emerged females held 24 hrs at 4°C	1	-	-	582
	2	0.19	0.19	1046
	3	0.35	0.07	1400
Females aged 48 hrs, then held 7 hrs at 4°C	1	1.29	0.79	2013
	2	0.91	0.69	4053
	3	0.53	0.38	3991
Females aged 7 days, then held for 7 days at 10°C	1	0.92	0.41	969
	2	0.68	0.39	1029
	3	0.91	0.18	552
	4	0.19	0.57	1050
	1	1.90	0.95	211
	2	0.78	0.13	767
	3	0.55	0.34	544
	4	0.48	0.24	415

Pappas, N. and L. Engstrom. Ball State University, Muncie, Indiana. Survey of ovariole numbers in wild-type D.m.

With the long-range aim of elucidating the genetics of gonad developmental homeostasis, we examined the ovariole numbers of 26 wild-type D.m. strains. It has been shown that great variations in ovariole numbers exist among Drosophila species (Kambysellis and Heed, 1971) and among French and Japanese strains of D.m. (Melou, 1961), but few reports exist regarding strains of D.m. readily available within the U.S.

We obtained 26 strains from various laboratories in the U.S. and grew them for several generations at 25°C under uncrowded conditions in half-pint milk bottles containing modified David's (1962) medium. From these stock cultures of each strain, at least 50 newly-emerged single pairs were selected at random, mated individually in 23 x 95 mm shell vials, and 50 females of each strain dissected 4-6 days later. After carefully teasing them apart, the germarial portions of the ovarioles in each ovary were counted.

Table 1 presents the data obtained. It can be seen that laboratory strains of D.m. possess a spectrum of ovariole numbers per individual female. The strains examined also possess varying degrees of genetic homogeneity based on the correlations of ovariole numbers between the two ovaries within females (Petit, 1963). Such information may be critical in developmental and reproductive studies.

The Margarita Island strain demonstrated a unique trait in that nearly 20% of all females possessed rudimentary ovaries (no maturing egg chambers) either unilaterally or bilaterally. Males of this strain likewise possessed rudimentary testes. This strain is presently under further genetic and developmental study.

Table 1. Mean total ovariole number and mean total log ovariole number (log of right plus log left ovaries' ovariole numbers) per female ± standard deviations. The sample size in each case is 50 females. The estimated correlation coefficients (r_s) between the ovariole numbers of the two ovaries within females are also presented (Engstrom and Brown, in preparation). The mean shown for the St. Margarita Is. strain are based on females all of which contain two normal ovaries; no correlation coefficient was calculated for this strain because of its unique gonads.

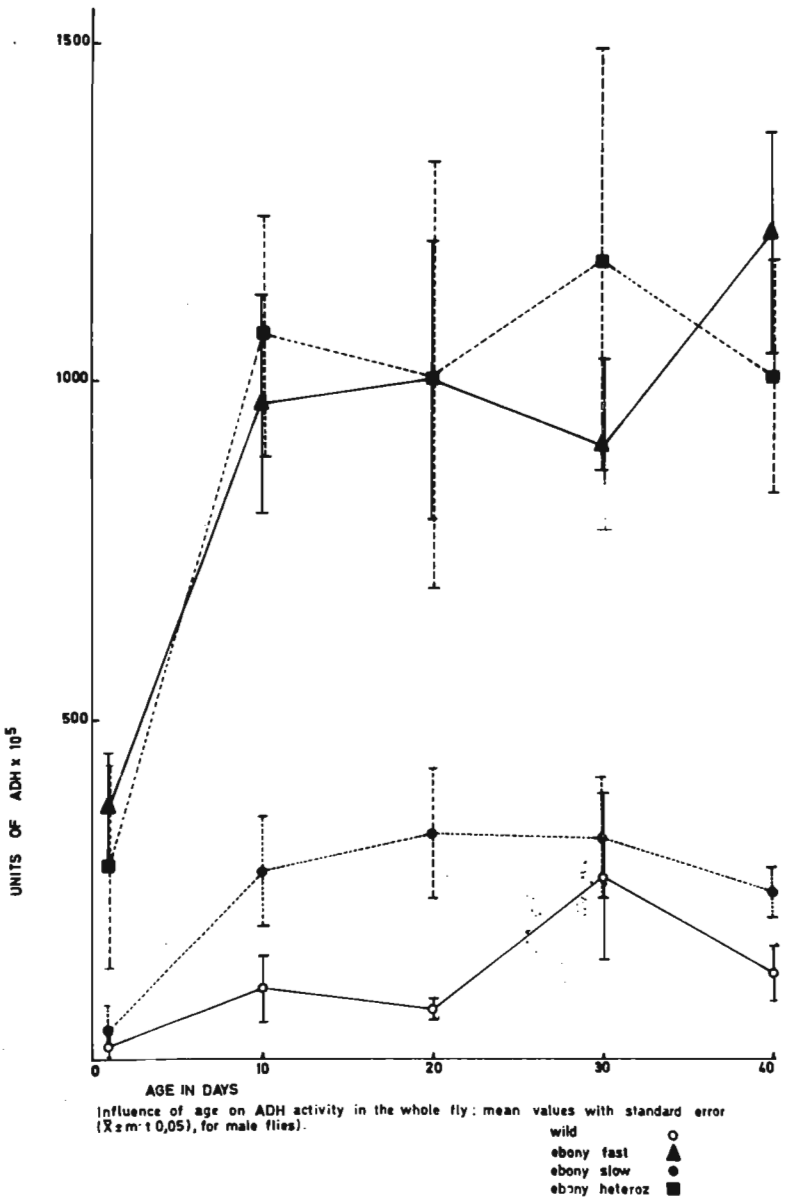
Strain	Mean total ovariole number ± S.D.	Mean total log ovariole number ± S.D.	r_s
Crimea	34.94 ± 2.32	2.48 ± .05	.2480
Hikone-R	29.39 ± 4.16	2.32 ± .14	.6765
Lausanne-S	44.16 ± 3.61	2.68 ± .07	.0619
Oregon-R	39.30 ± 3.46	2.58 ± .07	.1287
Samarkand	35.64 ± 2.57	2.49 ± .06	-.0069
Sato, Japan	26.45 ± 3.62	2.23 ± .11	.5055
Swedish-C	38.63 ± 4.10	2.56 ± .09	.0320
Waginengen	41.37 ± 3.20	2.62 ± .06	.0620
Curitiba	36.04 ± 2.39	2.50 ± .05	.1850
Formosa	33.73 ± 4.25	2.44 ± .11	.6008
Kalahari	29.61 ± 4.92	2.32 ± .17	.6050
Karsnas	43.30 ± 4.78	2.66 ± .09	.2842
Nyasa Lake	40.42 ± 8.43	2.55 ± .40	.2173
Wawawai	43.32 ± 4.59	2.66 ± .09	.4454
Witwatersrand	38.38 ± 3.03	2.56 ± .07	.0465
Culebra of P.R.	41.07 ± 3.85	2.61 ± .07	.2254
Hassel Is. of P.R.	32.68 ± 2.78	2.42 ± .08	.4190
St. Margarita Is., Venez.	36.76 ± 5.80	2.51 ± .15	---
Palimonitos of P.R.	36.40 ± 3.85	2.51 ± .09	.6822
St. Thomas	38.88 ± 3.27	2.57 ± .07	.3918
Surprise Key of P.R.	38.34 ± 3.31	2.55 ± .07	.0994
Bisgnano	45.09 ± 3.56	2.70 ± .06	.2947
Pavia	37.70 ± 4.41	2.54 ± .11	.4062
Roma	41.36 ± 3.63	2.62 ± .07	.3542
Sciolze	46.72 ± 5.23	2.72 ± .09	.3094
Umagazi River	39.28 ± 4.43	2.57 ± .10	.5012

References: David, J. 1962, DIS 36:128; Kambysellis, M.P. and W.B. Heed 1971, Amer. Nat. 105:31; Melou, J.P. 1961, Ann. Genet. 3:25; Petit, C. 1963, Ann. Genet. 6:29.

Libion-Mannaert, M., C. Deltombe-Lietaert, J. Delcour and A. Elens. Facultés Universitaires N.D. de la Paix, Namur, Belgium.

Ethanol utilization in a polymorphic ebony e^{11} strain.

A previous report from this laboratory has shown that our ebony e^{11} strain exhibits a very high constitutive level of Alcohol Dehydrogenase (ADH, E.C., 1.1.1.1.) as compared with a Canton Special wild control (Libion-Mannaert et al., 1972). As ADH isozymes in Drosophila are known (Johnson et al., 1964; Ursprung et al., 1965; Grell et al., 1965) to be under the control of two alleles (ADH^S and ADH^F), a further step in our studies was to investigate whether our stocks were polymorphic with respect to ADH alleles. Acrylamide electrophoresis of single fly homogenates (according to Ursprung et al., 1968) revealed the occurrence of only one pattern of bands in our Canton Special stock, corresponding to a homozygous ADH^S genotype, whereas in e^{11} three distinctive patterns were found to be attributable to ADH^F/ADH^F, ADH^F/ADH^S, ADH^S/ADH^S genotypes, the ADH^F and ADH^S alleles having frequencies of 0.71 and 0.29, respectively. Moreover, the agreement between the observed frequency of heterozygotes, and the expected value according to the Hardy-Weinberg law, indicates that our strain is in equilibrium. From this polymorphic strain it was then easy to derive homozygous substrains and to measure separately their ADH activity. It can be seen from Figure 1 that ADH activity in males is very different indeed at any age, from one genotype to another. (Similar results, not shown here, have been obtained with female flies.) Ebony ADH^F/ADH^F and ADH^F/ADH^S both exhibit a very high activity, whereas ebony ADH^S/ADH^S is characterized by an ADH level which is about the same as in Canton Special ADH^S/ADH^S. This confirms previous studies with wild strains showing that ADH^F genotypes exhibit a higher level of enzymatic activity (Day et al., 1974). Thus, the very high level of ADH activity previously found in our e^{11} laboratory stock obviously reflects the higher frequency of the ADH^F allele in that population.

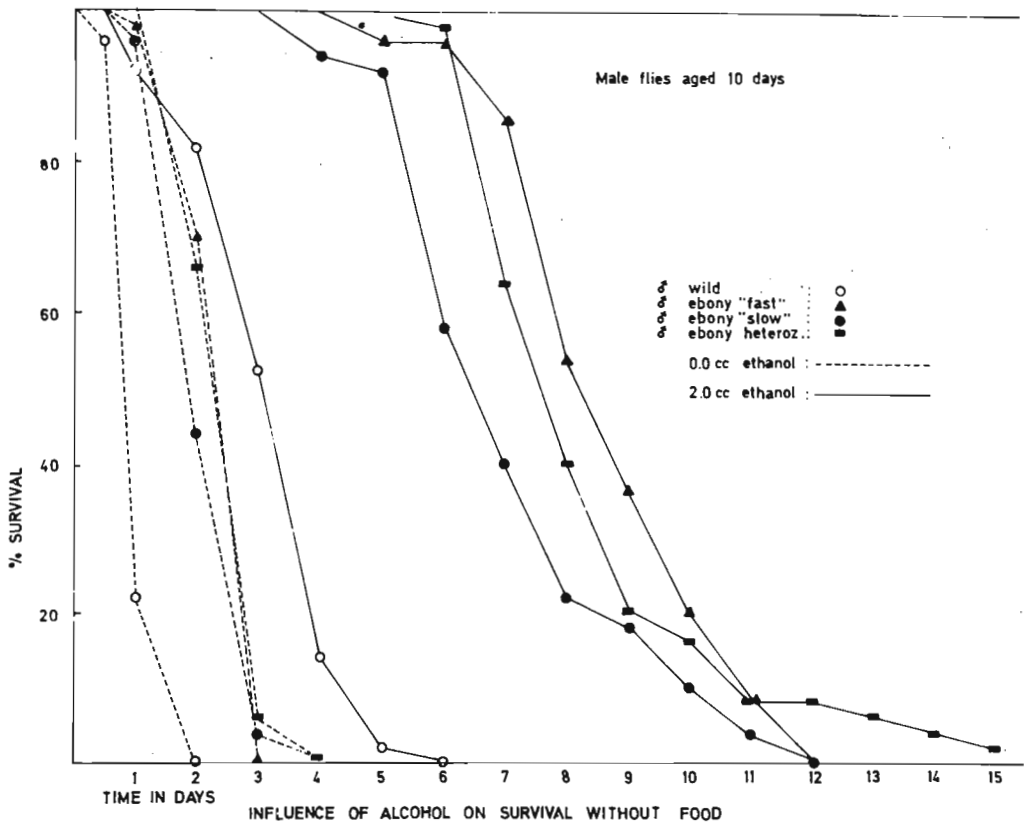


Influence of age on ADH activity in the whole fly: mean values with standard error ($\bar{X} \pm m \cdot t\ 0{,}05$), for male flies).

An interesting observation which might help explain why the ADH^F allele is so strongly favored under normal laboratory rearing conditions involving live yeast feeding, has been brought to our attention by Jacobs who showed that homozygous ebony flies fail to incorporate beta-alanine in their pupal cuticle and thus consequently retain a much higher concentration of beta-alanine in their haemolymph (Jacobs, 1966). As beta-alanine strongly inhibits $^{14}CO_2$

excretion from injected ^{14}C-labeled hexoses but not from ^{14}C-labeled glucose-6-phosphate (Jacobs, 1968), it seems that this metabolite can act as an inhibitor of carbohydrate metabolism at the phosphorylation step, thus preventing ebony mutants to get energy through glycolysis. Accordingly, ADH^F alleles, by providing ebony flies with an increased ADH activity, would be favored by selection as they make bearers more readily capable of getting energy from alcohol as a substitute for carbohydrate food.

In order to test that hypothesis, we decided to compare the survival time of Canton Special and e^{11} substrains under conditions where alcohol is the only source of energy available. Therefore, adult flies have been kept in vials containing a medium made exclusively of agar and water, to which various amounts of ethanol had been added. Figure 2 illustrates the



data obtained with 10 day-old males. It can be seen that in the absence of alcohol, there is no significant difference in survival time between the three e^{11} substrains which all survive a little longer than the Canton Special wild control. When alcohol is provided, although survival time of the control is only slightly increased, all three ebony substrains survive much longer, in the order $ADH^S/ADH^S < ADH^S/ADH^F < ADH^F/ADH^F$. We have repeated that experiment with male and female flies of various ages, and we observed the same trend - although in general differences between substrains are less drastic in young individuals, and the average survival time decreases with age. It can be concluded from these data that the special ability of ebony flies to use alcohol as a source of energy depends on their ADH content, which itself is under the control of ADH alleles, the homozygous ADH^F/ADH^F being in general superior in this respect to ADH^F/ADH^S heterozygotes, which in turn survive longer than the ADH^S/ADH^S homozygotes. Additional experiments are in progress to investigate further that phenomenon.

References: Day, T.H., P.C. Hillier and B. Clarke 1974, Biochem. Gen. 11:155; Grell, E.H., K.B. Jacobson and J.B. Murphy 1965, Sci. 149:80; Jacobs, M.E. 1966, Gen. 53:777; _____ 1968, J. Insect Physiol. 14:1259; Johnson, F.M. and C. Denniston 1964, Nature 204:906; Libion-Mannaert, M. and A. Elens 1972, DIS 49:77; Ursprung, H. and L. Carlin 1968, Ann. N.Y. Acad. Sci. 151:456; Ursprung, H. and J. Leone 1965, J. Exp. Zool. 160:147.

Kuroda, Y. National Institute of Genetics, Misima, Japan. Prolonged survival of cells from lethal embryos homozygous or hemizygous for dor in D. melanogaster in tissue culture.

Deep orange (dor) is an X-linked (1-0.3) recessive mutant of D. melanogaster. Eggs were collected from matings of dor/dor females and dor/Y males in the dor/ClB balanced stock and dechorionated by treatment with sodium hypochloride. Among 463 eggs obtained, 19 died before gastrulation, 179 died by abnormal gastrulation, 204 died after the stage of sac-like midgut, 61 died after muscular movement, and none were hatched.

Dor embryos which developed over gastrulation were torn into small fragments and cultured in T-5 flasks in medium K-17 supplemented with 0.1 mg/ml fetuin and 15% fetal bovine serum, as described previously (1,2).

Spindle-shaped muscle cells, 50 to 100 μm in length (Figure 1), flat polygonal epithelial cells, about 5 μm in diameter (Figure 2), and fibroblastic cells, 30 μm in length (Figure 3) were found to come out from tissue fragments and grow under in vitro conditions for a few

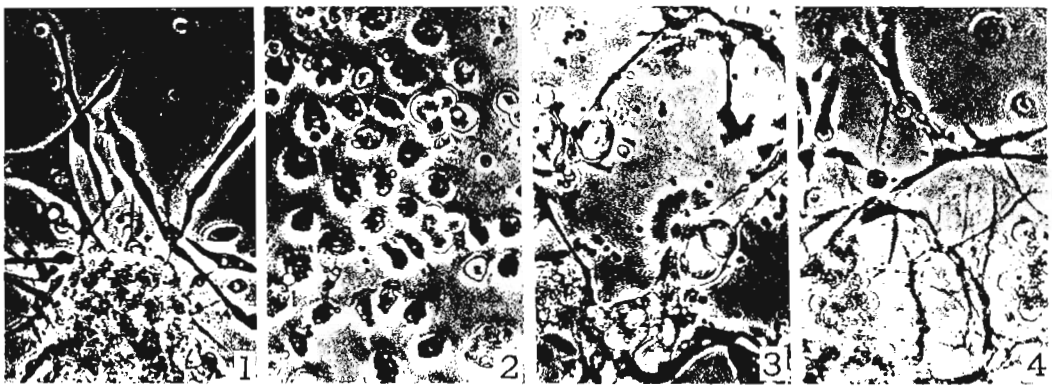


Figure 1. Muscle cells after 8 days of cultivation. x 320.
Figure 2. Epithelial cells after 3 days of cultivation. x 320.
Figure 3. Fibroblastic cells after 7 days of cultivation. x 320.
Figure 4. Nerve cells after 10 days of cultivation. x 320

weeks. Synchronous pulsation of muscle cells which were in contact with each other continued for more than five weeks. Some nerve fibers extended from nerve cells and formed a nerve network after 8 days of cultivation (Figure 4).

Most conspicuous differences in cultures of dor embryonic cells from those of wild-type embryonic cells were some defects in the syncytium formation of muscle cells, in the formation of baloon-like cellular spheres and in the growth of extremely small cells (assumed as imaginal disc cells), which have been previously found in cultures of wild-type embryonic cells (1).

The above results indicate that some cells from dor embryos were maintained in a functionally active state for a relatively extended period over the prospective lethal phase of the dor embryos.

References: 1. Kuroda, Y. 1974, Devel., Growth and Differentiation 16:55; 2. Kuroda, Y. 1974, J. Insect Physiol. 20:637.

Ebitani, N. North Shore College, Atsugi, Japan. The viability and esterase activities of electrophoretic variants in Drosophila virilis.

In Drosophila virilis, there are two esterase loci on the second chromosome, Est-α and Est-β. Ten alleles segregate for Est-α and five for Est-β locus (Ohba 1971; Ohba et al. 1974). Most of the esterase activity of electrophoretic variants are controlled by these two esterase loci (Ebitani 1974a).

As a part of the study in which the maintenance of esterase isozyme polymorphisms of D. virilis is experimentally analyzed, the effects of esterase activity on larva to adult viability were studied under competitive conditions in which three genotypes (00/00, 7B/00 and 7B/7B) having quite different esterase activities coexist at various frequencies. The 00/00 genotype has very low esterase activity in comparison with 7B/7B genotype. That of the heterozygotes (7B/00) is estimated to be intermediate between those of both homozygotes. Table gives the results of this study.

Relative larva to adult viability in relation to the frequencies of three genotypes.

Conditions	Series			Genotypes 00/00	7B/00	7B/7B	Significance
		Input freq.		0.04	0.32	0.64	
	I	Relative	♀	0.94 ± 0.25	1.00 ± 0.01		
		viability	♂	0	1.38 ± 0.16	0.87 ± 0.07	
		Input freq.		0.25	0.50	0.25	
A	II	Relative	♀	0.89 ± 0.08	1.04 ± 0.03		
		viability	♂	0.71 ± 0.16	1.11 ± 0.11	1.07 ± 0.19	
		Input freq.		0.64	0.32	0.04	
	III	Relative	♀	0.93 ± 0.04	1.13 ± 0.07		
		viability	♂	0.80 ± 0.08	1.46 ± 0.17	0.57 ± 0.41	*
		Input freq.		0.04	0.32	0.64	
	I	Relative	♀	0.60 ± 0.19	1.02 ± 0.01		
		viability	♂	0.19 ± 0.12	1.36 ± 0.15	0.87 ± 0.07	**
		Input freq.		0.25	0.50	0.25	
B	II	Relative	♀	1.12 ± 0.09	0.96 ± 0.03		
		viability	♂	0.92 ± 0.14	1.08 ± 0.09	0.92 ± 0.14	
		Input freq.		0.64	0.32	0.04	
	III	Relative	♀	0.97 ± 0.04	1.06 ± 0.07		
		viability	♂	0.79 ± 0.07	1.48 ± 0.14	0.59 ± 0.34	**

Condition A: 400 larvae per vial with 12 ml of 20% yeast medium
Condition B: 400 larvae per vial with 12 ml of 5% yeast medium
* Significant at the 0.05 probability level
** Significant at the 0.01 probability level

The heterozygotes seem to be slightly superior to both homozygotes but the viability did not differ between two homozygotes having different esterase activities. No evidence of frequency-dependent selection was recognized.

Alexandrov, I.D. and Z.V. Soluyanova. Research Institute of Medical Radiology, USSR Academy of Medical Sciences, Obninsk, U.S.S.R. Quantitative analysis of the complementation in white locus of D. melanogaster.

The negative complementation revealed by quantitative spectrophotometry of drosopterins was successfully used for the discrimination between w point mutations and deficiencies in part (w^-) or all (w^{258-45}) of the locus in question (Alexandrov, 1971). However, some of w point mutations (nonsuppressing z) do not obviously complement with the mutant w^{sp} at the phenotypic level: w point mutation/w^{sp} trans heterozygotes are basically similar to w^{sp}/w^{sp} but without mottled. According to Green (1959) such obviously noncomplementing w mutations represent a deficiency for a portion or all of the white locus. Thus, the data obtained by way of two tests are inconsistent relative to the nature of the same w mutations. The disagreement may depend on the rough qualitative test of complementation of the w with w^{sp} as opposed to the exact quantitative estimate of the negative complementation.

To test this possibility, the quantitative analysis of the drosopterins and ommochromes in the eyes of the visibly complementing and nonccomplementing trans heterozygotes was carried out. The double extraction of pigments was used according to Ephrussi and Herold (1944). After extraction, quantitative analysis of the drosopterins in the visible (480 mm) as well as in the ultraviolet (270 mm) regions of the spectrum and of the ommochromes in the visible (440 mm) region of the spectrum were made with a Spektromom 201 (Hungary). The data are expressed as the extinction (E) per 10 heads extracted per 1 ml of solvents.

According to these data (see the Table) all w mutations studied show the complementation in both visible and ultraviolet regions in contrast to the deficiencies. It is important that

The quantity of drosopterins and ommochromes in w^{sp}/w trans heterozygotes.

w^{sp}/w trans heterozygotes	Drosopterins 270 mm M*	Drosopterins 270 mm Conf. limits**	Drosopterins 480 mm M	Drosopterins 480 mm Conf. limits	Ommochromes 440 mm M	Ommochromes 440 mm Conf. limits	
w^{sp}/w^{258-45}	0.199	0.216 - 0.182	0.034	0.044 - 0.024	0.014	0.016 - 0.012	
w^{sp}/w^-	0.157	0.168 - 0.146	0.026	0.034 - 0.018	0.010	0.013 - 0.007	
w^{sp}/w^1	0.365	0.409 - 0.321	0.115	0.128 - 0.102	0.056	0.062 - 0.050	Suppress
w^{sp}/w^{10gA}	0.381	0.392 - 0.370	0.166	0.171 - 0.161	0.052	0.065 - 0.039	zeste
w^{sp}/w^{69gA}	0.325	0.354 - 0.296	0.079	0.096 - 0.062	0.031	0.041 - 0.021	
w^{sp}/w^{59gA}	0.290	0.301 - 0.279	0.054	0.060 - 0.048	0.032	0.039 - 0.025	Do not
w^{sp}/w^{57gA}	0.273	0.287 - 0.259	0.050	0.063 - 0.037	0.021	0.026 - 0.016	suppress
w^{sp}/w^{15gA}	0.260	0.285 - 0.235	0.068	0.093 - 0.043	0.033	0.036 - 0.030	zeste
w^{sp}/w^{13gA}	0.247	0.258 - 0.236	0.061	0.068 - 0.054	0.030	0.035 - 0.025	
w^{sp}/w^{sp}	0.193	0.206 - 0.180	0.042	0.048 - 0.036	0.011	0.016 - 0.006	

* Means of 4-6 replicas ** Conf. limits at $P_{0.05}$

in all cases there is complementation with respect to the quantity of both pigments simultaneously. It is evident too that the visibly complementing w mutations (w^1 and w^{10gA}) have more quantity of the pigments than other w mutations with questionable complementary phenotype. Thus, six irradiation-induced w mutations studied (w^{10gA} - w^{69gA}) are the complementing ones (positively with w^{sp} and negatively with w^+) and are therefore considered to be nondeficient. These results show that the quantities of pigments may serve as indirect assays of the biological activity of the white product modified by the mutations.

References: Alexandrov, I.D. 1971, DIS 46:71-72; Ephrussi, B. and J.L. Herold 1944, Genetics 29:148-175; Green, M.M. 1959, Z. indukt Abstamm. - u. Vererb. 90:375-384.

Salceda, V.M. and J. Guzmán. Colegio de Postgraduados ENA, Chapingo, México, and Instituto Nacional de Energía Nuclear, México City, México. A yearly record of Drosophila collections in Mexico City.

Starting in June 1969 and finishing in May 1970, a series of twice a week collections were done in the Mexico City area. The collections correspond to 26 localities scattered through the Distrito Federal, but due to the irregularity of collections in some localities, they were abandoned and only those places in which the trapping was continuous are recorded in the present report. The trapping was done in 1/4 liter bottles with slices of melon and a piece of cotton underneath; 3 to 4 bottles were put for 3 days in each locality and remained there until the next group of 3 to 4 bottles replaced them, then carried to the laboratory for further examination. The localities include even a garden, kitchen or garbage-place of the houses of the people from our laboratory who kindly collaborated in the collections. Once in the laboratory the flies were classified by species and sex; most of them were used for specific purposes (mainly D. melanogaster), the remaining species were classified and a stock was started when possible.

Once a complete season was obtained, the collecting continued in about 5 localities to the present time, mostly depending on the interest of the investigators. Data concerning genetic load, CO_2 sensitivity will be published elsewhere by the different authors working in the project; here we are concerned only with the relative abundance of Drosophila species and their distribution during the year.

Table 1. Total number of flies and their corresponding percents.

Species	# of flies	Percent
melanogaster	23801	66.76
Repleta group (3 species)	7461	20.92
pseuddobscura	1769	4.96
immigrans	1435	4.03
hydei	753	2.11
busckii	430	1.21

As can be seen in the Tables, 5 determined species plus undetermined members of the Repleta group were collected during the whole period of study (namely melanogaster, hydei, pseudoobscura, busckii and immigrans).

A total of 35649 flies were captured during the year. Occupying the first place in order of abundance was D. melanogaster with 23801 or 66.76%, then Repleta group (3 undetermined species) with 7461 or 20.92% and so on, as can be seen in Table 1.

Table 2 shows the number of flies caught according to sexual distribution; in all the cases males were more abundant than females, at least when we analyze the totals, since in some cases we can see that in a particular month and for a particular species there is a majority of females, even though this is not the general pattern.

Table 2. Relative frequency of Drosophila species in the period June 1969-May 1970, in the Mexico City area.

Species		June	July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	Mar.	Apr.	May	Total
melanogaster	♀	1019	1730	1783	2352	647	220	219	6	143	228	1962	695	23801
	♂	1182	2082	1958	2811	747	313	372	2	122	225	2157	826	
Repleta Group (undet. sp.)	♀	39	1	47	129	98	208	218	-	183	137	911	104	7461
	♂	244	7	90	171	190	950	246	-	458	444	2128	458	
pseudoobscura	♀	9	1	18	67	20	-	23	1	112	55	274	18	1769
	♂	20	3	39	108	82	37	28	1	205	100	499	49	
immigrans	♀	10	13	32	344	122	34	18	-	6	3	77	18	1435
	♂	9	11	22	263	155	51	26	-	2	4	179	36	
hydei	♀	19	31	41	91	39	10	12	-	12	11	32	12	753
	♂	8	28	32	94	65	40	16	-	36	16	82	26	
busckii	♀	4	7	16	65	58	19	2	-	1	-	2	13	430
	♂	1	2	8	104	83	23	5	1	-	-	7	9	
Total	♀	1100	1783	1937	3048	984	491	492	7	457	434	3258	860	14851
	♂	1464	2133	2149	3551	1322	1414	693	4	823	789	5052	1404	20798
Grand Totals		2564	3916	4086	6599	2306	1905	1185	11	1280	1213	8310	2264	35649

When we analyze the data concerning the distribution during the year, Table II shows that the most successful month is April, followed by September and August and in the last place January with only eleven flies. A peculiar pattern of distribution is shown in Figure 1 in which two peaks can be observed that could reflect the climatic condition of the area, since there are two periods of humidity (rain) alternating with two of dryness, and a reflection of the coldest month, January, that accumulates both effects, dryness and coldness, inhibiting the presence of flies. The peak that covers the months of June to October, seems to be a kind of plateau in which the highest percent of flies could be caught; the second plateau, even though it has the highest peak in collections - in April - averages fewer total flies caught.

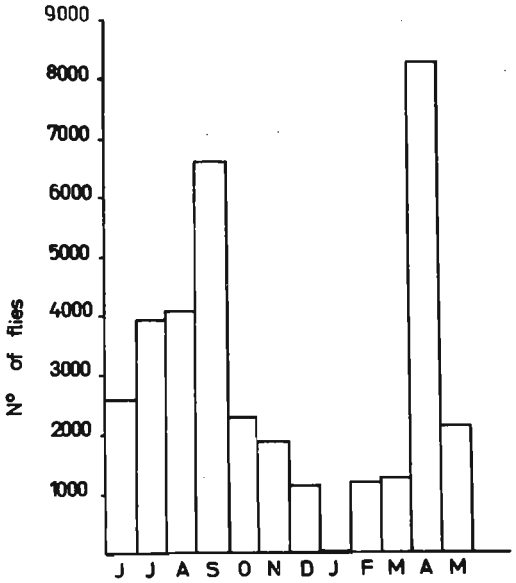


Fig.1.-Number of flies captured by month

When our interest is the abundance of a particular species, we observe that D. melanogaster and the Repleta group species are the most abundant during the whole year; here we can note that melanogaster follows the same pattern of distribution as the total of flies with the highest percent occurring from June to September in contrast with the species of the repleta group in which the highest plateau corresponds to the months of February to May with a very peculiar peak in November that, curiously, is the only month in which melanogaster does not occupy the first place, and concerning the Repleta group is the second most abundant month. The other species, except pseudoobscura, follow the same pattern as melanogaster.

When our attention is oriented to those collecting places that are best, we see, in Table 3, those localities that are most successful for different species caught. The temporal distribution in all the localities is the same as that of the pattern of Figure 1.

Table 3. Species distribution by locality.

Species	Locality number*
melanogaster	1,2,3,4,5,6,7,8,9,10,11,13,19,20,21,23,25,26,27,30,31,34,35,36.
Repleta group (3 species)	1,2,5,6,8,9,10,11,19,20,21,26,34.
pseudoobscura	1,2,3,4,6,9,19,20,21,30,31.
immigrans	1,2,3,6,8,9,11,19,20,21,25,26,27,30,31,34,37.
hydei	1,2,3,5,6,9,10,19,20,21,23,30,31,34,37.
busckii	1,2,3,5,6,9, 10,19,20,21,24,25,26,27,30,34.

* The localities' numbers correspond to those cited by Felix, R, et al, DIS 47:105-109

Hedrick, P.W. University of Kansas, Lawrence, Kansas. X-linked selection.

The behavior of X-linked genes is quite different from that of autosomal genes when the gene frequencies in the two sexes are different. Instead of reaching an equilibrium in two generations, the gene frequencies in the males and females for an X-linked gene oscillate around the mean gene frequency for many generations. Will these oscillations still be present if selection is operating at an X-linked locus where there are different gene frequencies in the two sexes? And will selection be as effective as when there is no initial unbalance in gene frequencies?

In order to investigate these questions, I set up two experiments to demonstrate the effect of unequal gene frequencies in males and females on the rate and pattern of elimination of a mutant (yw), X-linked chromosome. The initial mean gene frequency was .833 in both these experiments. In experiment 1, .833 was also the initial frequency in both sexes but in

experiment 2 all the wildtype chromosomes were in the males so that the gene frequency in the males was .5 and in the females, 1.0. The closed circles in Figures 1 and 2 give the gene frequency over generations for these experiments. The values given are the

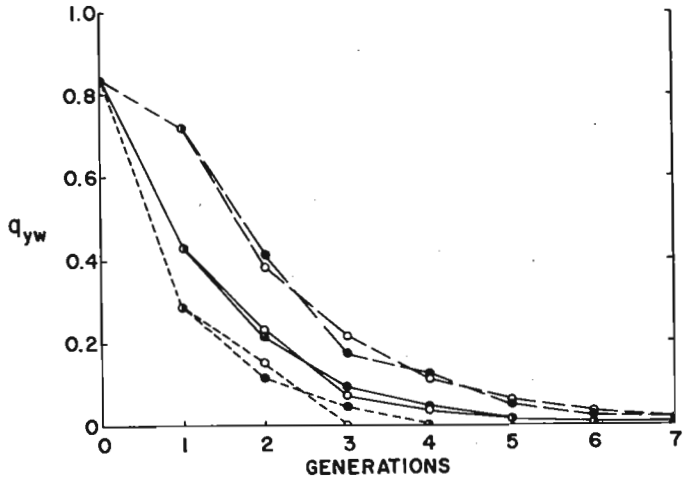


Figure 1. The average gene frequency of yw over generations when the initial gene frequency in both males and females is .833. The solid, broken, and dotted lines indicate the mean, male, and female gene frequencies, respectively. Closed circles indicate the observed values (average of four replicates).

means of four replicates for each experiment. There was little variation in response between the replicates. The open circles are simulated values which will be reported elsewhere.

In both experiments the mutant

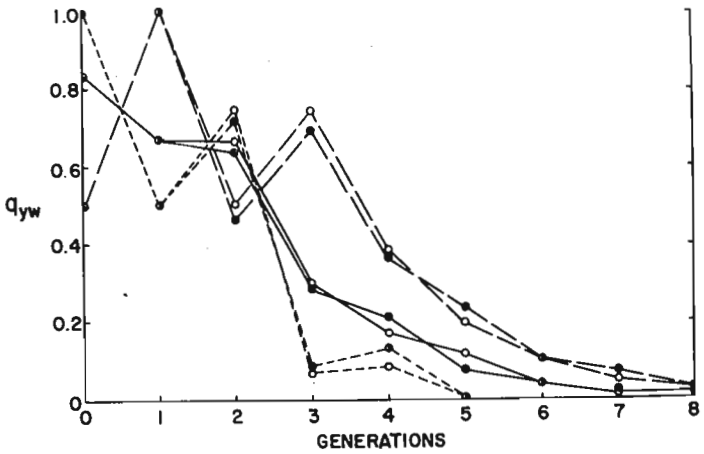


Figure 2. The average gene frequency of yw over generations when the initial gene frequency in the males is .5 and 1.0 in the females. The solid broken and dotted lines indicate the mean, male, and female gene frequencies, respectively. Closed circles indicate the observed values (average of four replicates).

chromosome was eliminated quickly so that after seven and eight generations, respectively, it was virtually absent in all the replicates. The pattern of elimination between the two experiments is quite different, however. In the first experiment, elimination of the mutant proceeded very much like an autosomal gene if only the mean gene frequency (solid line) is considered. But when the gene frequencies in the males (broken line) and females (dotted line) are considered separately, the gene frequency estimate of the mutant in the females is significantly lower than the frequency of the males in all the generations. This differential is due both to the strong mating advantage that wildtype males have over yw males and to the one-generation lag in male gene frequencies for an X-linked gene.

The elimination of the mutant was slower in experiment 2, with the mean gene frequency of the mutant always significantly higher than for the same generation in experiment 1. While the gene frequency declined every generation, in generation 2 the change in experiment 2 was particularly small (-.032). For the same generation in the first experiment the gene frequency declined nearly seven times as much (-.213). The difference is a result of the absence of wildtype males and the consequent absence of mating selection in this generation for experiment 2.

When the frequencies in the males and females are examined in experiment 2, they are found to oscillate for the first three generations around the mean frequency. Males had the highest frequency in generations 1, 3 and all succeeding generations and females the highest in generations 0 and 2. In other words, the frequencies oscillated but the oscillations were quickly damped. After the oscillations had ceased, the mutant was eliminated in much the

fashion of experiment 1. During this latter period, the gene frequency estimate in the females was significantly lower than in the males as in experiment 1.

From these experiments it is apparent that even with selection, oscillations in gene frequency for an X-linked gene can occur for several generations. Furthermore, in this experiment these oscillations had the effect of retarding the eliminination of the mutant chromosome.

Bock, I.R. and J.S.F. Barker. University of Western Australia, Perth; University of Sydney, Sydney. Further Drosophila collections from Indonesia.

Barker and Bock (DIS 50:163) reported the species analysis of a Drosophila collection from Sulawesi, Indonesia, made in February 1973.

The opportunity presented itself in February, 1974, for one of us (J.S.F.B.) to make several further Drosophila collections in Indonesia. The following three separate localities were sampled: Bogor (Bogor Botanic Gardens - sweep over rotting star-fruit); Malang (East Java - sweep over mixed fruit bait); and Ujung Pandang (South Sulawesi - sweep over mixed fruit bait); the site and method of collection were the same as in the above reference.

Some of the material collected was returned to the laboratory live and analysed after isofemale cultures were established from wild-caught females; some material was preserved in alcohol immediately after catching. Analysis of the collections is tabulated below:

	Locality*				
	M	UP	M	UP	B
	Isofemale cultures		Alcohol preserved		
Drosophila (Sophophora) melanogaster sp. gp.:					
Females	-	-	131	36	10
	♂♂ only				
D. ananassae Doleschall	12	0	2	2	3
D. atripex Bock & Wheeler	0	0	0	1	0
D. bipectinata Duda	10	22	28	2	0
D. engracilis Bock & Wheeler	0	0	2	0	0
D. kikkawai Burla	0	0	3	0	0
D. parabipectinata Bock	0	23	2	1	0
D. pseudoananassae Bock	0	0	2	0	0
Suzukii subgp. sp. nov.	0	0	1	0	0
(infertile)	6	5	-	-	-
	♂♂ + ♀♀				
Drosophila (Drosophila) immigrans sp. gp.:					
D. hypocausta Osten-Sacken	1	1	8	0	4
D. sulfurigaster Duda	0	0	1	0	31
sp. undet.	0	0	1	0	0
Drosophila (Scaptodrosophila):					
sp. 1	0	0	1	0	1
sp. 2	0	0	0	0	3
sp. 3	0	0	0	0	4
Liodrosophila sp.:	0	0	0	0	2

* M = Malang, UP = Ujung Pandang, B = Bogor

The dominant spp. in the collections were melanogaster group (particularly ananassae subgroup: ananassae, atripex, bipectinata, parabipectinata, pseudoananassae), and two spp. of the immigrans group.

A few individuals of a third subgenus, Scaptodrosophila (= Paradrosophila = Pholadoris) were also found. From recent experience with members of this latter subgenus in Australia it appears that many, if not most, of the spp. are not attracted to fruit baits and can only

be collected by the laborious process of making a general sweep and separating the Drosophila caught from everything else. Thus there may be many more spp. of the subgenus Scaptodrosophila in the regions sampled in these collections than were actually taken by baiting.

The high frequency of males in the melanogaster group in the 1973 Ujung Pandang collection (33/38) was not repeated in the 1974 collection, and in fact the frequency of males was very low (6/42 in the preserved material; males not recorded in the live material as many were mired in the medium during transit). There were some differences in the composition of the fruit bait in the two years, but more intensive collecting would be necessary to assess any significance of the apparent difference in sex ratio.

Berg, R.L. 9 Colborne Rd., Apt. 11, Brighton, Massachusetts. Concentration, mode of inheritance, rate of inheritance, rate of occurrence of abnormal abdomen (aa) in three populations of D.m. in 1973.

In October 1973 flies were collected in populations of Magarach (near Nikita Botanical Gardens, the Crimea) and of Dilizhan and Erevan (Transcaucasus). A simultaneous rise in the rate of abdominal segmentation abnormalities was observed in all of them in 1968. In 1969, 1971, 1972 the aa phenotype was abundant in all of them (Berg 1972, 1973). In 1973 the aa phenotype was as abundant as in 1972 (Table 1). Fifty aberrant males were crossed individually with Muller-5 females to study the mode of inheri-

Table 1. Concentration of abnormal abdomen phenotype in geographically isolated population of Drosophila melanogaster in 1973.

Population		Males			Females		
		n	number	%	n	number	%
Crimea	(Nikita Botanical Gardens, Magarach)	676	176	26.0	650	296	45.5
Transcaucasus	(Dilizhan)	270	58	21.4	349	239	68.5
	(Erevan)	1256	228	18.1	997	437	43.8
	Totals	2202	462	21.0	1996	972	48.7

tance of their phenotypical deviations and the rate of occurrence of sex linked mutations in their spermatogenesis and in the gametogenesis of their offspring raised under laboratory conditions. 38 males manifested the character aa, 12 males manifested other phenotypical abnormalities (Table 2).

Table 2. Mode of inheritance of abnormal abdomen (aa) in the progeny of wild aberrant males.

Population	Phenotype of the wild aberrant male	Mode of inheritance of aa				Total
		Sex Linked	Autosomal	Sex linked & autosomal (2 alleles)	No segregation	
Magarach	aa	0	2	2	1	5
	other abnormalities	0	2	0	2	4
	total	0	4	2	3	9
Dilizhan	aa	0	4	0	3	7
	other abnormalities	0	1	0	1	2
	total	0	5	0	4	9
Erevan	aa	1	14[x)]	5	6	26
	other abnormalities	0	1	0	5	6
	total	1	15	5	11	32
Totals	aa	1	20[x)]	7	10	38
	other abnormalities	0	4	0	8	12
	total	1	24[x)]	7	18	50

x) One of these males is heterozygous for two autosomal alleles

In the progeny of ten aa males no segregation was observed. Somatic mutations, as well as rare combinations of mutant alleles with a very low penetrance, are probable. The 28 aa males proved to be bearers of 36 hereditary abnormalities of the abdominal tergites. One of these 28 males was heterozygous for two different autosomal mutations - one characterized by high, the other by low penetrance in homozygotes. In the progeny of 19 males segregation for the autosomal aa allele was observed. One male manifested a sex linked mutation. Seven males had two mutant aa alleles - one sex linked and one autosomal. In the progeny of 12 males bearing other phenotypical abnormalities, 4 proved to be heterozygous for an autosomal aa mutant gene. The concentration of sex linked aa alleles in the populations studied is not less than 8:2202 or 0.363 percent. The actual value exceeds this minimum more than tenfold. Only 38 aa males out of 462, i.e., less than one tenth, were studied to determine among them the number of males bearing sex linked aa alleles. The frequency of wild males manifesting sex linked aa alleles in their phenotype is roughly 3-4 percent. The rate of occurrence of mutations proved to be very high (Table 3). 35 lethal mutations (2.04 percent), 13 subvital mutations with no visible effect (0.76 percent), 35 visible mutations (2.04 percent) were detected among 1713 X-chromosomes of wild males. Out of 35 visible mutations 12 were aa (6 occurred in Magarach males and 6 in Ararat males). Three out of these 12 mutations manifested

Table 3. Rate of occurrence of sex linked mutations in the spermatogenesis of wild males and in the gametogenesis of their offspring raised under laboratory conditions.

Population	n	Mutation Rate: lethals no.	lethals %	subvitals no.	subvitals %	visible mutations no.	visible mutations %	total no.	total %	among visible mutations aa mutations no.	among visible mutations aa mutations %
Magarach	1222	24	1.96	5	0.41	24	1.97	53	4.34[x] (8.43)	6	0.49
Dilizhan	238	9	3.78	5	2.18	3	1.31	17	7.28	0	0
Erevan	253	2	0.79	3	1.19	8	3.16	13	5.14	6	2.37
Totals	1713	35	2.04	13	0.76	35	2.04	83	4.85[x] (7.76)	12	0.70

x) This percentage does not include a cluster of reverse mutations arisen at a most early stage of gametogenesis of one of the wild Magarach males. In 114 out of 178 F_2 cultures males had poor viability and abnormal wing position (raised wing), 14 F_2 cultures contained no males, 50 F_2 cultures contained the normal number of males manifesting normal wing position. Percentages including these 50 mutant gametes are given in parenthesis.

aa among several pleiotropic effects. Two mutant alleles manifested themselves only in the presence of autosomal aa alleles. The average rate of occurrence of sex linked aa mutations is thus 12:1713 or 0.70 percent. In the Ararat population it is 6:253 or 2.37 percent. Some of the aa mutations arisen during the experiment were most likely missed due to low penetrance of most of the aa alleles. Besides, 8 out of 50 P males of the M-5 experiment had sex linked aa alleles in their X-chromosomes masking new mutations. The value 0.70 percent is by no means exaggerated. Besides the obvious and most important role of natural selection in the maintenance of heterogeneity at the aa loci, mutability contributes greatly to the maintenance of the polymorphism. The role of mutation pressure is intensified during the periods of the temporal rise of mutation rate. It is highly probable that in 1967 Drosophila populations entered into the period of raised mutation rate which lasts till now and the aa loci are involved in this mutability rhythm.

References: Berg, R.L. 1972, DIS 48:67-69; Berg, R.L. 1973, DIS 50:92.

Budnik, M. University of Chile, Santiago.

"Hybrid vigor" of interspecific hybrid between D. pavani and D. gaucha.

There are numerous instances in which hybrids between species are larger, faster growing, or otherwise exceeding the parental species in some quality. This kind of "luxuriance" or "hybrid vigor" cannot be related to true "heterosis" because interspecific hybrids fail to reproduce.

An interesting case of "hybrid vigor" has been recently observed in crosses between Drosophila pavani and its sibling species D. gaucha. D. pavani occur mainly in Central Chile but it overlaps the distributional area of D. gaucha in a small area near San Luis (Argentina). D. gaucha have been found in Southern Brasil, Uruguay, Argentina and Bolivia. Both species interbreed freely in the laboratory giving an abundant hybrid offspring that is completely sterile (Brncic and Koref-Santibañez, 1957).

In experiments on competitive ability of preadult hybrids between D. pavani and D. gaucha under a limited supply of food and space, it was found that the egg-to-adult survival ratio of the hybrids is higher than the corresponding parental species (Brncic and Budnik, 1974).

For the analysis of preadult viability, two experimental series were set up. The first consisted in a series of small vials containing 10 cc of basic cornmeal-agar food medium. Into each vial were transferred either 100 eggs of D. pavani or 100 eggs of D. gaucha or 100 eggs of D. gaucha females inseminated by D. pavani males. In each group, 4000 eggs were sown. The second experimental series was set up similarly to the first, but transferring 200 eggs per vial. In the experiments referred to here, one stock of D. pavani was used which originated from flies collected in Central Chile (Bellavista), and a X-linked "yellow" mutant of D. gaucha that was selected and established from Brasil (Campos do Jordan).

Table 1 shows a higher egg-to-adult survival ratio of hybrids between D. pavani and D. gaucha when compared with the corresponding parental species. This observed superiority seems to be density dependent. At the higher density assayed (200 eggs/vial) the differences in viability are more significant.

Table 1. Differences in egg-to-adult survival of D. pavani, D. gaucha and their hybrids at two densities.

	100 eggs per vial			200 eggs per vial		
Crosses	Total No. of eggs	No. of adults emerged	χ^2	Total No. of eggs	No. of adults emerged	χ^2
pavani/gaucha	4000	2762		4000	2083	
pavani/pavani	4000	2665	5.40*	4000	1393	242.20**
gaucha/gaucha	4000	2532	29.54**	4000	1682	80.66**

* P. (1 d.f.) = 0.02
** P. (1 d.f.) < 0.0001

The superiority in survival of the hybrids under crowding conditions may be interpreted as the expression of some kind of "hybrid vigor" in relation to the use of food supply. According to Brncic and Budnik (1974) this superiority is not expressed when the hybrids compete for the same food with the parental species. In the presence of D. pavani or D. gaucha preadults, there is a break down of the egg-to-adult survival ratio of the hybrids. This apparent contradiction may be due to the fact that under competitive conditions with the parental species, there are in play other factors besides shortage of food, such as "interference" based in the accumulation of waste metabolic products of the latter species that could partially inhibit the growth of the hybrids.

(Research financed by grants from the University of Chile (Grant No. 159), and the Multinational Genetic Program of the O.A.S.).

References: Brncic, D. and S. Koref-Santibañez 1957, Evolution 11:300-310; Brncic, D. and M. Budnik 1974, Ecology 55:662-666.

Anastasia-Sawicki, J. Cornell University, Ithaca, New York. Ultrastructural histochemical localization of acid phosphatase in salivary glands of D. melanogaster.

Optimal reaction conditions for the histochemical localization of acid phosphatase-1 in D. melanogaster using the Gomori method[1] have been determined. Salivary gland tissue from third instar larvae was used in defining these conditions for three reasons: 1) the glands are large and relatively easy to remove and dissect, 2) the tissue is composed of primarily one cell type, 3) the glands undergo histolysis during the pupal stage; it is, therefore, reasonable to expect large numbers of lysosomes in these cells. Acid phosphatase has been localized to lysosomes in many other organisms[2].

A systematic analysis in which different parameters of the Gomori reaction were varied was made. Optimal reaction conditions, defined as conditions under which essentially every lysosome stains for acid phosphatase (Figure 1), are obtained when 0.058 M Na β-glycerophosphate, pH 5, is used as substrate. The optimal incubation time for the reaction is 30 minutes at 37°C. Under these conditions, diffusion artifacts are minimal and specificity of the reaction for acid phosphatase-1 is maintained.

Several substrates at different concentrations and for different incubation times were tested in these studies. Substrates that were used are 2' and 3' AMP, α-naphthol phosphate, and Na β-glycerophosphate. The substrate concentration was varied in each case from 0.001 M to 0.058 M. The incubation time was also varied from one second to one hour for each substrate.

For each set of conditions, salivary glands were first dissected and removed in standard Drosophila Ringer's solution and fixed in Karnovsky's fixative[3] at 4°C for 1-1/2 hours. The tissue was then transferred to freshly prepared Gomori medium and incubated at 37°C for the appropriate time. Following a 3 minute wash in 0.1 M cacodylate HCl buffer, pH 7.2, the tissue was postfixed in 2% aqueous OsO_4 for 2 hours at room temperature. The tissue was washed in distilled H_2O for 25 minutes and then dehydrated in a graded acetone series. The tissue was embedded in Epon 812 which was polymerized by incubating at 60°C for 36 hours. Thin sections, having a silver or gold interference color, were prepared using a Reichert Om U2 microtome and glass knives. The sections were floated onto Formvar coated 300 mesh copper grids. All sections were stained in 2% uranyl acetate for 1 hour. Some were stained a second time with lead citrate for 5 minutes. Sections were viewed on a Philips 300 electron microscope operating at 80 kV.

Throughout this study, three different controls were used. In one, tissues were incubated in a medium lacking substrate. The results from this control were defined as the background level of reaction product deposition. Another control relied on the inhibition of acid phosphatase by 0.01 M sodium fluoride. The third control employed a homozygous null mutant for acid phosphatase-1. In spectrophotometric assays, this mutant displays less than 10% wild-type total acid phosphatase activity and no visible band is present in the acid phosphatase-1 zone after staining either starch or acrylamide gels[4].

It should be emphasized that reaction conditions which are optimal for salivary gland tissue may not necessarily be the optimal conditions for other tissues due to differences in cellular permeability and cytophasmic constitution.

References: 1) Gomori, G. 1952, Microscopic Histochemistry: Principles and Practice, U. of Chicago Press, Chicago, Ill.; 2) Dingle, J.T. and H.B. Fell, eds. 1969, Lysosomes in Biology and Pathology, Vol. 1, North-Holland Pub. Co., London; 3) Karnovsky, M.J.A. 1965, J. Cell Biol. 27:137A; 4) Bell, J. and R. MacIntyre 1973, Biochem. Genetics 10:39.

This research was supported by NIH Training Grant #GM01035-II.

Figure 1. (on following page) Third instar salivary gland tissue, wild-type for acid phosphatase-1, following incubation for 30 minutes at 37°C in Gomori medium with 0.058 M Na β-glycerophosphate as substrate. (X 31,800).

Intensely black material is lead phosphate resulting from acid phosphatase-1 activity. Note its deposition, as indicated by the arrows, in lysosomes (L). No activity is seen in mitochondria (M), the nucleus (N), and secretory granules (S).

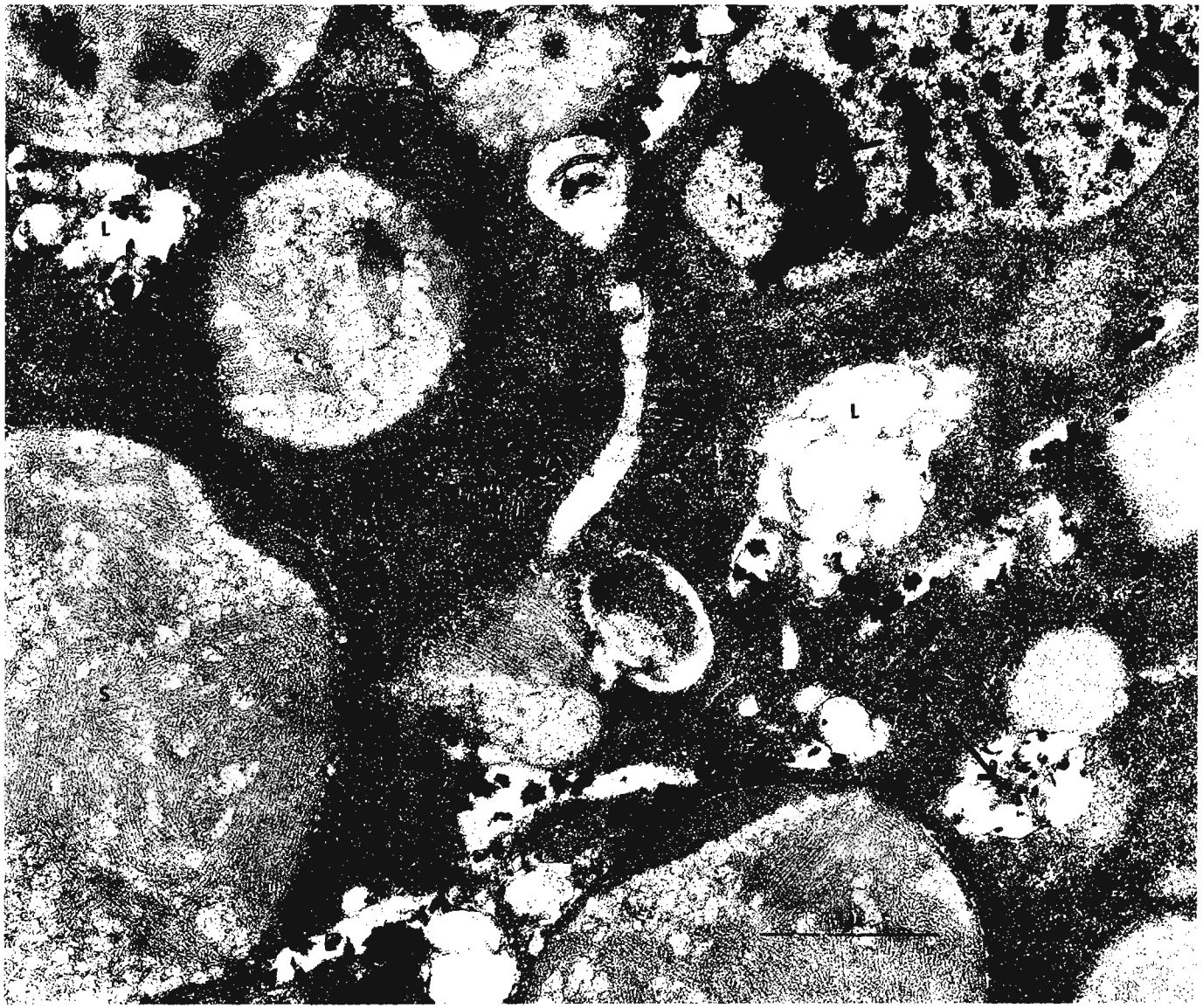


Figure 1: J. Anastasia-Sawicki "Ultrastructural histochemical localization of acid phosphatase in salivary glands of D. melanogaster". See legend on preceeding page.

Barker, J.F. University of the Witwatersrand, Johannesburg, South Africa.
Species of the Zaprionus tuberculatus group and their distribution.

Many single female lines of Z. tuberculatus were established from eight populations in Nigeria in 1970. In 1970-71 esterase polymorphism was studied in these lines in Professor Forbes Robertson's department at Aberdeen, Scotland. It was found that individuals derived from Northern Nigerian populations had a different pattern of esterase isozymes to individuals derived from the most southerly Nigerian populations. Breeding tests showed that northern individuals would not interbreed with southerly individuals, so there appears to be two biological species within Z. tuberculatus. Tests carried out here in South Africa during the last two years using Nigerian derived stocks and a few single female lines that the writer has established from parts of South Africa, have so far shown that the South African forms will interbreed with the Northern but not the Southern Nigerian forms. However, only a small part of South Africa has so far been sampled. In Nigeria, it appears that the southern form may be largely confined to areas which are or were, tropical rainforest. In 1971, samples of flies were sent to Mr. E. Basden at Edinburgh. He was unable to find any differences between the two Nigerian forms in genitalia and other conventional taxonomic characters.

It was also shown at Aberdeen that there are differences between the Nigerian populations with respect to fly size and length of time between egg laying and adult emergence under standard conditions. However, these differences were not between populations of the northerly "species" and the southerly "species". So the difference in the esterases remains the only known phenotypic difference between the two forms.

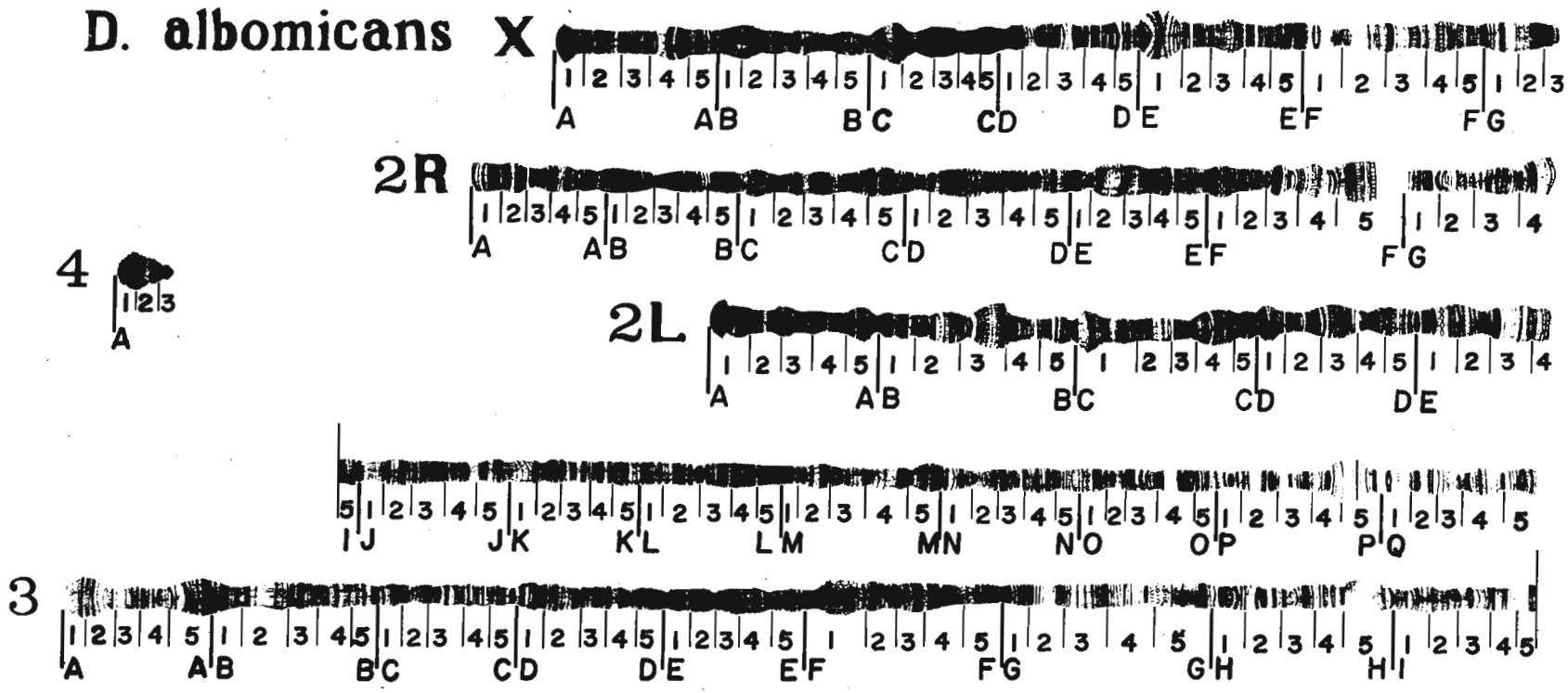
Lin, F-J.[1], H-C. Tseng[2] and T-C. Wang[1]. 1. Academia Sinica, Nankang, Taipei, Taiwan; 2. University of Nebraska, Lincoln.
Standard map of the salivary gland chromosomes of Drosophila albomicans Duda.

The standard map of the salivary chromosomes of D. albomicans has been made by us. D. albomicans is one of the dominant species in Taiwan. There are 113 stocks of this species kept in our laboratory collected from various localities in Taiwan. We chose a stock from Jui-suei in eastern Taiwan, for the frequency of its chromosome inversions is very low, as our working material.

The salivary gland chromosome complement of D. albomicans consists of four arms and one dot radiating from a conspicuous chromocenter. The longest arm represents the third chromosome and the dot which is made from dot plus heterochromatin represents the fourth chromosome. The remaining three arms are the X-chromosome, the chromosome 2R and chromosome 2L. The X-chromosome can be easily recognized by comparing the pairing of sex chromosome from the F_1 hybrid of interspecific crosses with D. sulfurigaster and its characteristic doubled length. The chromosome 2R has a notable feature of a 'loop', which is not an inversion, near the base of the arm. When a salivary slide is squashed hard enough to stretch the chromosome, the 'loop' no longer appears in some cells but as the proximal end of the 2R (see also Wilson et al. 1969). Besides, the chromosome 2R is nearly as long as the X-chromosome. The remaining one is the chromosome 2L. The chromosome 2L is usually marked by a conspicuous band near the base. For chromosome variations of D. albomicans see Wakahama et al. (1968, 1971) and Mather and Thongmeearkom (1972a, 1972b and 1973 as Taxon B of nasuta complex).

Each arm of the chromosome, from the terminal end to the base, is divided into several sections by alphabetical order and each alphabet-cited section is then subdivided into five subsections according to points where chromosome breakage easily occurred to form inversions, approximately equal numbers of chromosome bandings, and easily recognized bands or puffs as shown in the figure (next page).

References: Mather, W.B. and P. Thongmeearkom 1972a, DIS 48:40; 1972b, DIS 49:109; and 1973, DIS 50:60; Wakahama, K., O. Kitagawa and C. Kastritsis 1968, Proc. XII Int. Cong. Genet. 1:17.3.5; Wakahama, K., O. Kitagawa and O. Yamaguchi 1971, DIS 46:144; Wilson, F.D. M.R. Wheeler, M. Harget and M. Kambysellis 1969, Univ. Tex. Publ. 6918:207-253.



Standard map of the salivary gland chromosomes of Drosophila albomicans Duda.
(See note of F-J. Lin, H-C. Tseng and T-C. Wang on preceding page.)

Moiseenko, E.V. and V.T. Kakpakov.
Kurchatov's Institute of Atomic Energy, Moscow, USSR. The absence of hypoxanthine-guanine phosphoribosyltransferase in extracts of Drosophila melanogaster flies and established embryonic diploid cell line.

The lack of the corresponding phosphoribosyltransferases results in a resistance of the mutant mammalian cells to purine base analogues. The possibility of obtaining such mutants in D. melanogaster cell lines was studied. However, the examination showed that diploid cell line 67j25D (Kakpakov et al., 1969) is resistant to a high concentration (100 µg/ml) of 6-mercaptopurine (6MP) and 8-azaguanine (8AG) while a low concentration of these analogues (1-10 µg/ml) inhibits the growth of mammalian cells. At the same time the Drosophila cells are sensitive to adenine analogues: 8-azaadenine (8AA) and 2,6-diaminopurine (2,6DAP) (Table 1).

Table 1. Effects of purine analogues on growth of Drosophila cell line 67j25D.

Analogues	µg/ml	Inoculum x10^6 cells/ml	Yields of cells x10^6 cells/ml (days)						
			3	4	6	7	9	10	11
	0	0.2	-	-	20.0	-	-	27.0	-
6MP	10	0.2	-	-	20.0	-	-	23.0	-
	100	0.2	-	-	12.0	-	-	20.0	-
	0	0.2	0.5	-	3.5	-	-	8.5	-
8AG	10	0.2	0.5	-	4.0	-	-	9.0	-
	100	0.2	0.4	-	3.0	-	-	6.6	-
	0	0.1	-	1.6	-	14.0	-	-	25.0
8AA	10	0.1	-	0.7	-	2.0	-	-	11.0
	100	0.1	-	0.5	-	0.7	-	-	1.0
	0	0.3	3.0	-	-	10.5	12.8	-	-
2,6DAP	10	0.3	0.9	-	-	1.6	1.7	-	-
	100	0.3	0.4	-	-	0.2	0.3	-	-

In order to explain the reason for different sensitivities of Drosophila cells to the guanine and adenine analogues, assays of activities of purine pyrophosphorylases were carried out.

It is well known that the sensitivity of mammalian cells to purine base analogues is determined by the presence of the hypoxanthine-guanine phosphoribosyltransferase (HGPRT) and adenine phosphoribosyltransferase (APRT) which are responsible for the interconversion of purine base analogues to the corresponding nucleotides.

The HGPRT and APRT activities were tested according to the methods of Fujimoto and Seegmiller (1970) and Harris and Cook (1969). The results of measurement of the HGPRT and APRT activities in extracts of the three established cell lines of Drosophila melanogaster and one mosquito cell line are shown in Table 2. The cell line 67j25D (Kakpakov et al., 1969) and the

Table 2. Activities of HGPRT and APRT (counts/100 sec/µg protein).

	Drosophila melanogaster							Mosquito cell line	Chinese hamster
	Cell lines			Fly stocks					
Enzyme	67j25D	Kc	Cl	Swedish	Ore-R-C	Ore-R-S	br pn	Mos 20 A	cell line
HGPRT	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	< 0.5	250
APRT	390	380	390	135	258	157	142	370	1000

line Kc and Cl established by Dr. G. Echalier (Echalier and Ohanessian, 1970) were used. The absence of HGPRT activity in comparison to the mammalian cell line but normal level of APRT activity in all the cell lines was observed. In accordance to this result no incorporation of hypoxanthine-8-^{14}C in nucleic acids of the Drosophila cell cultures was detected (12 count per 100 sec E_{260} for hypoxanthine-8-^{14}C as compared to 20050 count per 100 sec E_{260} for adenine-8-^{14}C). Thus the insensitivity of Drosophila cell lines to 6MP and 8AG is a result of the absence of HGPRT in these cells. On the contrary, the sensitivity to the adenine

analogue correlates well with normal level of the APRT. In this connection the results of Echalier (1971) reported the isolation of 8AG-resistant mutant in Drosophila melanogaster cell line (Kc) are surprising. Determination of activities of pyrophosphorylases in extracts of flies from several stocks of Drosophila melanogaster reveals the natural resistance of D. melanogaster cells to 6MP and 8AG indicating the absence of HGPRT (Table 2).

Our results show the inability to use resistance to 6-mercaptopurine and 8-azaguanine as a genetic marker in Drosophila genetics.

References: Kakpakov, V.T., V.A. Gvozdev, T.P. Platova and L.G. Polukarova 1969, Genetika (Russ) 5:67-75; Fujimoto, W.Y. and J.E. Seegmiller 1970, Proc. Natl. Acad. Sci. USA 65:577-584; Echalier, G. and A. Ohanessian 1970, In Vitro 6:162-172; Echalier, G. 1971 Curr. Top. Microbiol. Immunol. 55:220-227; Harris, H. and P.R. Cook 1969, J. Cell Sci. 5: 121-133; Varma, M.G.R. and M. Pudney 1969, J. Med. Entomol. 6:432-439.

Pinsker*, W. and H. Hampel. Institut für allgemeine Biologie, Vienna, Austria.

The importance of visual stimuli in the mating behavior of D. ambigua.

The mating behavior of D. ambigua was investigated with reference to its dependence on visual stimuli. For that purpose the mating frequency was tested in light and darkness.

Three strains were used for these experiments: a wild-type strain and the mutant strains "white" (w) and "yellow" (y). In the white strain visual acuity is reduced by the lack of eye pigment, and therefore these flies cannot receive visual courtship signals. In the yellow strain the color of the body has changed from black to yellow. A homologous mutation is known already in other species of Drosophila in which males of this phenotype are discriminated against by wild type females.

For measuring the mating success single pairs were set up in 15 ccm plastic tubes for 24 hours, and afterwards the storage organs of the females were examined for sperm. In this way homogamic as well as heterogamic matings were studied, 200 pairs respectively.

The percentages of inseminated females are shown in the table.

		+ ♂♂	w ♂♂	y ♂♂		+ ♂♂	y ♂♂		y ♂♂	
Light		86.5	31.5	54.0		95.5	38.5		85.5	%
	+ ♀♀				w ♀♀			y ♀♀		
Dark		19.5	25.0	3.0		----	----		13.5	%

The figures prove that wild type D. ambigua copulates less frequently in the dark. The same reduction of mating frequency has been found in the yellow strain (in homogamic matings). On the other hand white males paired with wild-type females do not distinguish between light and dark mating conditions ($\chi^2 = 1.45$ n.s.). Owing to their reduced vision their insemination rate equals the level of wild-type males in complete darkness.

From these results may be concluded that the courtship behavior of D. ambigua males depends on visual stimuli from the courted individual.

Visual courtship signals given by the males are of no importance to the females as the experiment with white females and wild type males reveals. The lack of visual acuity in the white females does not reduce their mating activity in the least.

Another finding of these investigations is the fact that normal females discriminate against yellow males significantly ($\chi^2 = 49.0$, $p < 0.001$).

Accordingly it was of interest to find out whether this sexual disadvantage of the yellow males was caused by their different color of body. If this were the case, yellow males would not be repelled by normal females in the dark, nor by the weak eyed white females in light either.

The results of the experiments, however, are contrary to that conclusion: wild-type females discriminate against yellow males to a higher degree in the dark ($\chi^2 = 12.5$, $p < 0.001$) and so do the white females in light, compared with normal females ($\chi^2 = 5.35$, $p < 0.025$).

The reason for this discrimination of yellow males cannot be attributed to the color of the body but must be found in other pleiotropic effects of this mutation, which will dominate when visible characters recede.

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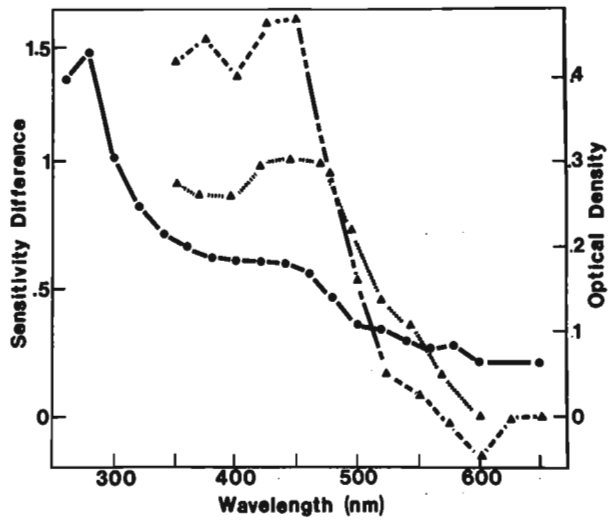
Stark, W.S. The Johns Hopkins University, Baltimore, Maryland. Spectral absorption characteristics of sepiapterin measured in situ and in vivo.

Recent studies have determined the absorption spectra of Drosophila eye color pigments using microspectrophotometric (MSP) measurements of pigment absorption in situ (Strother and Superdock, 1972; Stark, 1973), and electroretinographic (ERG) studies of the in vivo effects of pigments on the visual spectral sensitivity (Stark, 1973; Stark and Wasserman, 1974). These studies have resulted in several conclusions which could not have been predicted on the basis of previously published absorption data of the pigment extracts, namely: (1) that the red drosopterins absorb at longer wavelengths in the eye (Stark, 1973) than in vitro (c.f. Ephrussi and Herold, 1944; Maas, 1948), probably because of binding of the chromophore to pigment granule proteins; (2) that the darkening of cn and bw eyes with age does not cause functional increases in the pigments' visual screening effect; and (3) only the oxidized form of xanthomatin functions to screen light in bw eyes even though the reduced form is also present in the eye.

The present study was undertaken to obtain in vivo and in situ absorption spectra of the yellow pterin which is present in sepia (se) eyes (sepiapterin). For this purpose, a homozygous cn;se (having only sepiapterin) stock was derived and compared with cn bw (having no eye color pigments) in ERG studies. The ERG-based in vivo absorption spectra of sepiapterin in newly emerged flies were obtained by subtracting cn;se from cn bw spectral sensitivities obtained using methods similar to those described elsewhere (Stark, 1973). MSP-based in situ absorption spectra were obtained from frozen sections through newly emerged cn;se eyes mounted on quartz slides with quartz coverslips in pH 7.4 phosphate buffered 15% glycerin using a Zeiss UMSPI. Optical density was computed as the log of the light transmitted through the brain (to control for light scatter) over the light transmitted through the eye color pigments between ommatidia and near the basement membrane.



These absorption spectra are plotted in the accompanying figure: newly emerged ERG-based, hollow triangles and dashed line; 7-day post-eclosion ERG-based, solid triangles and dotted line; newly emerged MSP-based, dots and solid lines. Note that the ERG data are plotted against the "sensitivity difference" ordinate while the MSP data are plotted against the "optical density" ordinate. The amplitude of the MSP-based data is arbitrary relative to the amplitude of the ERG-based functions. The ERG-based function from newly emerged cn;se peaks at 425-450 nm with a high absorption at shorter wavelengths and a rapid drop-off at longer wavelengths. This is in sharp contrast with the red Drosophila pterins (unique to Drosophila) (drosopterins) which have ERG- and MSP-based absorption maximum at 500 nm (Stark, 1973). The ERG-based function from 7-day post-emergence cn;se shows a decreased effect of the pigment on sensitivity for short wavelengths and an increased relative absorption at longer wavelengths. This bathochromic shift between the sepiapterin from newly emerged and 7-day post-eclosion flies suggests that the pigment changes its binding, reacts, or is partly replaced by a different pigment in the first week of adult life. This difference may also account in part for the difference in eye color appearance in the first week of adult life. The MSP data confirm the relatively low absorption in the long wavelength region for sepiapterin from newly-emerged cn;se and also show an absorption peak around 280 nm. The relative maximum regions of these MSP data agree with previously published MSP data concerning sepiapterin (Strother and Superdock, 1972). These data show that the yellow sepiapterin has shorter wavelength absorption than the red drosopterins of cn or the brown ommatins of bw (c.f. Stark, 1973). The pterins of se appear to have an absorption spectrum similar to the pterins present in the eye of other diptera like the blowfly Calliphora (Burkhardt, 1962).

References: Burkhardt, D. 1962, Symp. Soc. Exptl. Biol. 16:86-109; Ephrussi, B. and J.H. Herold 1944, Genetics 29:148-175; Maas, W.K. 1948, Genetics 33:177-190; Stark, W.S. 1973, J. Insect Physiol. 91:427-441; Strother, G.K. and D.A. Superdock 1972, Vision Res. 12: 1545-1547.

Supported in part by an award from The Johns Hopkins University Biomedical Sciences Support Grant and in part by The Johns Hopkins University Department of Psychology.

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Selection for Adh-variants in D.m.

Eight populations of Drosophila melanogaster were set up to study the effects of different alcohols on the allele frequencies at the alcoholdehydrogenase locus. The populations were started with 75 pairs of F_1 flies derived from crosses between homozygous S-lines and homozygous F-lines from the Groningen population (details in Bijlsma-Meeles and Van Delden, 1974). Each population was kept on a particular food medium at 25°C and consisted of five bottles. Each generation the newly emerged flies from the five bottles were thoroughly mixed and divided into approximately equal numbers over five bottles with fresh medium, except for a sample of about 75 pairs of flies which were available for genotype determination by electrophoresis. One control population was kept on standard medium, consisting of 1000 ml water, 19 g agar, 54 g sucrose, 32 g yeast, 13 ml nipagin solution (10 g nipagin in 100 ml ethanol 96%). The other populations were kept on standard food supplemented with one of the following alcohols (concentrations given as volume percentages): methanol 2.5%, ethanol 15%, propanol 2.5%, isopropanol 2.5%, butanol 2.5%, hexanol 0.5%, glycerol 15%. Electrophoresis was carried out on whole fly homogenates on 5.5% polyacrylamide gels for 2.5 h at 300 V and 40 mA, in 0.041 M veronal-HCl buffer, supplemented with 0.001 M EDTA, pH 8.4. After electrophoresis the gels were transferred to a staining solution, consisting of 25 ml 0.041 M veronal-HCl buffer, supplemented with 0.001 M EDTA, pH 8.4, 7.5 mg MTT, 10.0 mg NAD, 0.5 mg PMS and 0.1 ml isopropanol. Alleles were described according to the nomenclature of Grell c.s., 1965.

Genotype frequencies were determined in generations 2, 5 and 10. The resulting allele frequencies are shown in Figure 1. An increase in the frequency of the F-allele is found on all alcohol media, except for glycerol. The sharpest rise is observed on hexanol, where the F-allele is nearly fixed (p_F = 0.98 in generation 10). Because the concentrations of the alcohols applied in this experiment were such that no drastic reduction in population size occurred, genetic drift cannot be held responsible for the changes in allele frequency of the observed magnitude. As it is known (Gibson, 1970; Vigue and Johnson, 1973; Van Dijk, unpublished) that extracts of FF homozygotes have a higher specific activity in vitro compared to both other genotypes, the rise in F-frequency is probably the result of a better conversion of the toxic alcohols by the FF homozygotes, resulting in a higher survival of this genotype.

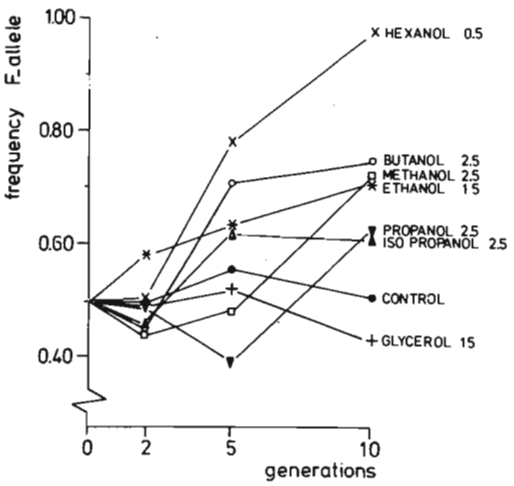

Figure 1. Change in frequency of the Adh^F-allele on different alcohols.

These determinate changes in allele frequency, brought about by the presence of proper substrates for the alcoholdehydrogenase enzyme, unambiguously prove the occurrence of selection at this locus. The results are not in conformity with the hypothesis that allozyme variants are selectively neutral.

References: Bijlsma-Meeles, E. and W. van Delden, 1974, Nature 247:369-371; Gibson, J. 1970, Nature 227:959-960; Grell, E.H. c.s. 1965, Science 149:80-82; Vigue, C.L. and F.M. Johnson 1973, Biochem. Genet. 9:213-227.

Gallo, A.J.* and V.M. Salceda. Colegio de Postgraduados ENA, Chapingo, Mexico.
Drosophila collections from four states in Mexico.

From August to October 1973 a series of collections were done in four states of Mexico (Mexico, Michoacán, Veracruz and Sinaloa); a total of eight localities were sampled. The sampling was done using fermented bananas as bait, but in the localities of Veracruz and Uruapan, sweeping of the net was also practiced; in these cases, the places for collecting were market place and wine distillery. From each locality, three to four places were sampled in the outskirts of the city, such as orchids or pine forest. The altitude of the localities ranges from sea level to 2250 m. A total of 8468 flies were captured and carried to the laboratory in Chapingo, Mexico, where they were classified by species group, species and sex. Table I shows the total numbers of flies captured and their percentages. Table II shows the totals of males and females of each species captured and their corresponding localities.

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Table I. Total number of flies and their corresponding percents.

	hydei	aldrichi	arizonensis	nigricuria	nigrohydei	leonis	longicornis	melanogaster	simulans	ananassae	unipunctata	bipunctata
Total no. of flies	289	65	341	30	12	13	276	1417	963	3904	3	2
Percents	3.41	0.76	4.02	0.35	0.14	0.15	3.26	16.73	11.37	46.10	0.03	0.02

	Grupo tripunctata Undetermined sp.	sturtevanti	emarginata	cardini	cardinoides	willistoni	nebulosa	pseudoobscura	virilis	latifascideformis	immigrans	busckii
Total no. of flies	23	49	9	25	82	276	2	145	1	56	197	28
Percents	0.27	0.57	0.10	0.29	0.96	3.26	0.02	1.71	0.01	0.60	2.32	3.40

Table II on next page

QUOTABILITY OF NOTES

Alexandrov, I.D. 44:78, 44:114, 46:69, 46:71, 46:72, 48:88, 48:133, 51:105
Alexandrov, I.D. & Z.V. Soluyanova 51:32
Arajärvi, P. & A. Hannah-Alava 44:73
Hannah-Alava, A. 44:110
Lakhotia, S.C. 48:110
Lakhotia, S.C. & A.S. Mukherjee 45:108, 46:65
Mather, W.B., M. Clyde & D. Lambert 51:125
Mather, W.B., P. Thongmeearkom, M. Clyde & D. Lambert 51:86
Puro, J. & P. Arajärvi 43:90
Puro, J., T. Mygrén & M. Nuutila 50:108
Steiner, W.W.M. 51:129
Steiner, W.W.M., W.E. Johnson & H.L. Carson 50:100

For previous listings see DIS 38, 42, 43, 44, 45, 47, 48, 49 and 50.

		Veracruz, Ver. sea level 4-6 August		Jalapa, Ver. 1420 m 4-6 August		Zitacuaro, Mich. 1781 m 25-27 August		Morelia, Mich. 1941 m 25-27 August		Patzcuaro, Mich. 2174 m 25-27 August		Uruapan, Mich. 1634 m 25-27 August		Chapingo, Méx. 2250 m 15-17 October		Culiacan, Sin. 53 m 7-11 August		Culiacan, Sin. 53 m 19-22 October	
		M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
repleta	hydei	25	28	-	1	-	-	-	-	-	2	149	62	-	-	-	-	1	1
	aldrichi	35	30	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
	arizonensis	-	-	-	-	-	-	2	2	-	-	-	-	-	-	172	165	-	-
	nigricuria	-	-	-	-	-	-	-	-	-	-	-	-	12	18	-	-	-	-
	nigrohydei	-	-	-	-	-	-	-	-	-	-	-	-	3	9	-	-	-	-
	leonis	-	-	-	-	-	-	-	-	-	-	-	-	7	6	-	-	-	-
	longicornis	-	-	-	-	-	-	-	-	-	-	-	-	186	90	-	-	-	-
melanogaster	melanogaster	316	265	14	4	4	-	-	-	9	2	360	242	21	13	31	39	69	28
	simulans	95	88	77	99	6	5	8	28	66	24	108	43	213	82	10	6	1	4
	ananassae	424	315	-	-	-	-	-	-	-	-	1	-	-	-	-	-	1774	1390
tripunctata	unipunctata	-	-	-	1	-	-	-	-	-	-	1	1	-	-	-	-	-	-
	bipunctata	-	-	-	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-
	Undetermined sp.	-	-	-	-	-	-	-	-	-	-	16	7	-	-	-	-	-	-
saltans	sturtevanti	9	40	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
	emarginata	-	-	3	6	-	-	-	-	-	-	-	-	-	-	-	-	-	-
cardini	cardini	-	1	-	-	-	-	-	-	-	-	9	12	-	-	-	1	1	1
	cardinoides	-	-	-	-	-	-	-	-	-	-	24	33	1	1	-	-	-	-
willistoni	willistoni	-	-	3	2	1	-	-	-	-	-	121	65	-	1	-	-	-	-
	nebulosa	1	1	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
obscura	pseudoobscura	-	-	-	-	-	-	-	-	2	-	8	4	75	56	-	-	-	-
virilis	virilis	-	-	-	-	-	-	-	-	-	-	1	-	-	-	-	-	-	-
victoria	latifascideformis	34	15	-	-	-	-	-	-	-	-	3	-	-	-	-	-	2	2
immigrans	immigrans	-	-	3	3	-	-	-	1	2	1	107	24	-	-	-	-	-	-
	busckii	-	-	-	-	-	-	-	-	4	3	42	38	3	1	-	-	-	-

Table II. Number of flies per species, sex and locality.

Trippa, G., A. Loverre and P. Torda.
Università di Roma, Italy. A third chromosome that modifies the Segregation Distortion phenomenon in D. melanogaster.

For some time now, factors that interact with the SD gene on the Segregation Distortion phenomenon have been known (Sandler and Hiraizumi, 1960 and 1961; Sandler, 1962; Sandler and Rosenfeld, 1962; Kataoka, 1967; Hartl, 1969; for a fairly precise review, see G.L. Gabor Miklos, 1972).

All the cases described so far relate to factors localized either on the X chromosome or on the second chromosome.

The present note concerns the discovery of a third chromosome in the natural population of Ranna (Sicily, Italy), the III^{Ra} chromosome, the effect of which, both in heterozygosis and in homozygosis, is to modify the k values shown by SD/SD^{+} heterozygous males. Its action is such that in the cases in which the initial value of k is low (0.66-0.85) it rises (1.00); but when it is high (0.99) it falls (0.88), as shown in Table 1.

Table 1. The effect of the III^{Ra} chromosome on the k values shown by males heterozygous for a second chromosome marked with bw and different SD chromosomes extracted from different Italian natural populations.

SD chromosome	Original k values*	Number of tested males	Total progeny examined	k values ± S.E.
Ca-148	.73	12	1191	1.00
Ca-230	.82	17	2176	1.00
Ra-88	.75	15	1773	1.00
Ra-127	.73	6	327	.97±.01
Ot-1	.80	14	1331	.99±.00
Co-17	.78	10	1070	1.00
Co-64	.71	14	1359	1.00
Co-100	.66	10	714	1.00
Co-IV	.70	12	865	1.00
Ar-38	.73	13	1142	1.00
Ar-249	.74	13	1083	1.00
Pe-6	.76	15	1037	1.00
Pe-122	.76	12	1394	1.00
Sa-262	.85	10	1061	1.00
Ro-1	.99	18	1878	.88±.02

Ca=Castellaneta; Ra=Ranna; Ot=Otranto; Co=Corato; Ar=Archi; Pe=Pedalino; Sa=Salemi; Ro=Roma

* The S.E. for all these values is in the order of 0.02

Moreover, in order to assess whether the action of the III^{Ra} chromosome works on the SD gene and induces a permanent and hereditary change in it, as has previously been observed with the Muller-5 chromosome by Sandler and Rosenfeld (1962), F_1 SD/bw;st/st males derived from $SD/bw;III^{Ra}/st$ males were crossed with bw;st virgin females and the values of k were calculated on the offspring. The values obtained by using six of fifteen SD chromosomes indicate that the mechanism of action of the III^{Ra} chromosome does not imply a permanent and inheritable modification on the SD gene present in the same genotype of the parent male.

A work in preparation discusses the possible importance of third chromosomes of this type in maintaining the SD chromosomes in natural populations.

References: Gabor Miklos, G.L. 1972, Genetics 70:405; Hartl, D.L. 1969, PNAS 63:782; Kataoka, Y. 1967, Japan J. Genetics 42:327; Sandler, L. 1962, Am. Naturalist 96:161; Sandler, L. and Y. Hiraizumi 1960, Genetics 45:1269; Sandler, L. and Y. Hiraizumi 1961, Genetics 46:585; Sandler, L. and A. Rosenfeld 1962, Can. J. Genet. Cytol. 4:453.

Chung, Y.J. Ewha Womans University, Seoul, Korea. The presence of Segregation-Distorter in a natural population of D. melanogaster in Korea.

The phenomenon of Segregation-Distortion (SD) has now been isolated from a natural population of D. melanogaster in Seoul, Korea. Males from wild and mass cultured populations of melanogaster were analyzed with the cn bw marker chromosomes. Among 95 males collected from Bulkwang-dong area in Seoul, September 1973, one male exhibited the k value of above 0.995, so this chromosome was designated as SD-K29. In order to test this abnormal chromosome for identity with the original SD-bearing chromosome (SD-72, SD^{NH}-2), various genetic experiments were performed and some of them are now in progress. Up to now, this SD has been stably maintained for about 20 generations by the recurrent backcrosses to the cn bw females. During the recurrent backcrosses, some stocks regularly exhibit distortion value (k) of 1.00 for several generations.

Reciprocal cross of SD-K29 females to cn bw males yields SD and cn bw progeny in approximately equal proportions as seen in the table below:

Phenotypes of progeny				Total	k values	% of recombination
SD	cn bw	bw	cn			
10,823	10,636	39	31	21,529	0.503	0.3251

This result indicates that the Segregation-Distortion is not expressed in the heterozygous females (mean k value of 0.503). And it is clear from the low recombination frequency that at least one inversion probably coupling with SD system is involved. In order to see if the SD-K29 chromosomes may carry a lethal gene or not, viabilities of heterozygous males for this SD-bearing chromosome which have been maintained for several generations were estimated by Cy/bw^{VI} balanced inversions. From the 24 tested SD-bearing second chromosomes, Cy flies only emerged in the F_3 generation (Cy:6,418; non-Cy:0). The results showed that the SD-K29 chromosome carries the lethal gene. This lethal carrying SD-K29 chromosome has been maintained by the Cy-balanced system.

The SD/Cy males were individually mated to cn bw females in order to see if the SD effect is suppressed by the Cy inversions as it is in the original SD strains and the mean k value was found to be 0.46, indicating that the SD action is inhibited by the Cy inversion. Since homozygous SD yields progeny in 1:1 ratios, important test for identity is undertaken to see the sensitivity (or insensitivity) of one to the other. Heterozygote between these SD-K29 and a Madison SD (SD-72) or a Odate SD (SD^{NH}-2) exhibits no distortion.

The frequency of SD genes in the Seoul population of D. melanogaster was about 0.47%. Further genetic study is now in progress.

Goldstein, R.B.[1], D.D. Miller[1] and R.A. Patty[2]. (1) University of Nebraska, Lincoln, and (2) Wake Forest University, Winston-Salem, North Carolina. Correction of some previously reported data concerning D. athabasca courtship sounds.

In a previous report of data collected from sonagrams of D. athabasca male courtship sounds (Patty et al., DIS 50:67, 1973) we reported that the intrapulse frequency for the "eastern A" semispecies averaged about 204 Hz. Further observations of sonagrams and of oscilloscopic patterns reveal that the dominant intrapulse frequency averages about the same as those reported for the other two semispecies. Although the sonagrams still tend to indicate a lower fundamental frequency (ca. 316 Hz) it is apparent that our earlier report was in error.

Additionally, improved recording techniques have removed much of the background noise, which had troubled our earlier determinations, revealing pulses (sensu Ewing, A.W. and H.C. Bennet-Clark, Behaviour 31:291, 1968) on the "wide band" sonagrams as well as on the oscillioscopic patterns. The "pulses" reported by us in our earlier note (Patty et al., 1973) are more precisely termed bursts (sensu Ewing and Bennet-Clark, 1968).

The improved clarity and increased number of recordings have also made us aware of the fact that all three semispecies may be easily distinguished from one another aurally, in opposition to our previous comment, that "eastern A" males were distinguishable from "eastern B" and "western-northern" males, but males of the latter two semispecies were not easily distinguishable from one another without the use of sonagrams.

Zacharopoulou, A. University of Patras, Greece. New types of inversions found in a wild D.m. population of Southern Greece.

During the Spring 1974, the analysis of a sample from a wild Greek Drosophila melanogaster population of the Peloponnese (Southern Greek Penninsula) revealed the presence of three new inversions. One of them was found on the X-chromosome and the other two on the third chromosome.

The break points were identified on the basis of salivary gland chromosome maps provided by Bridges and Brehme (1944). In (I) IA:3D (Figure 1) is the first wild X-chromosome inversion ever recorded in Drosophila melanogaster. In (3LR) 67E:98F (Figure 2) and In (3LR) 71F:90D (Figure 3) are the first pericentric inversions ever found on the third chromosome of Drosophila melanogaster.

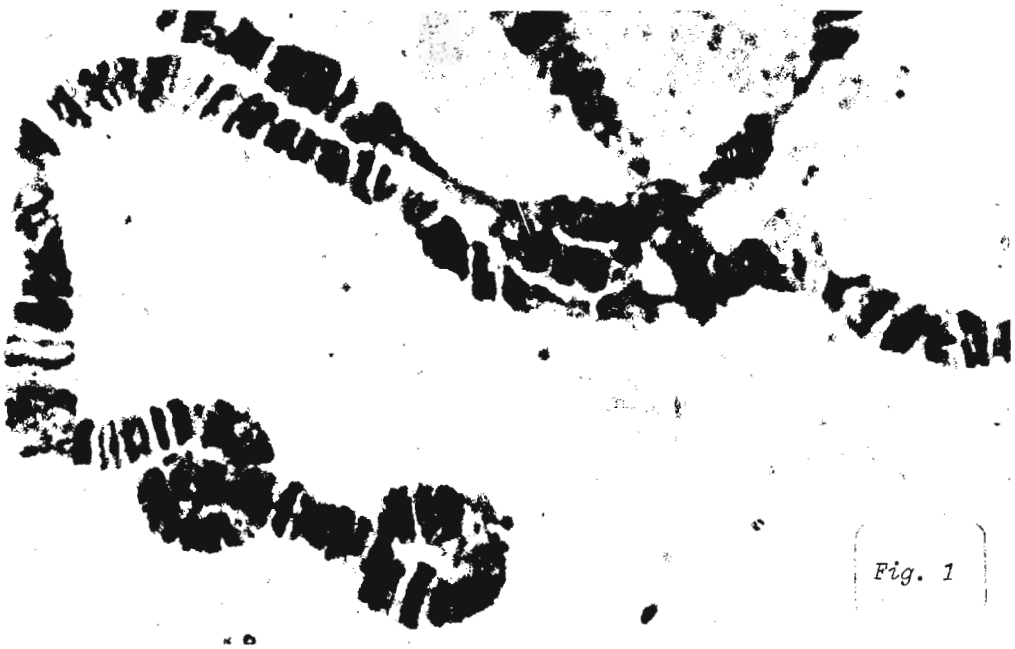

Fig. 1

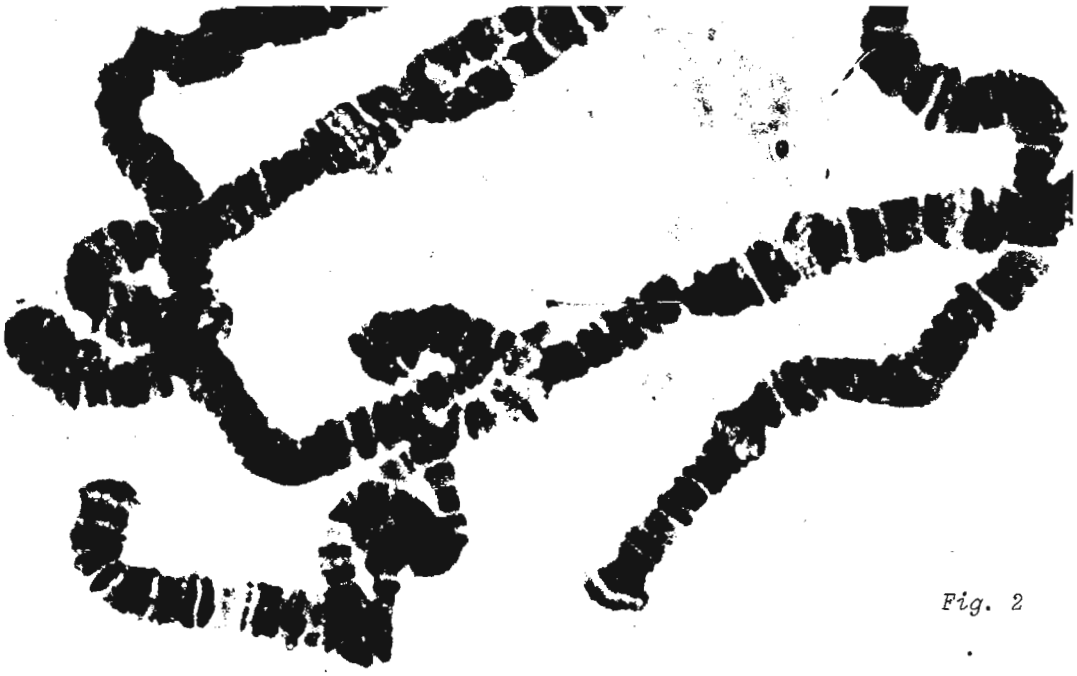

Fig. 2

Figure 3 next page

See note by A. Zacharopoulou on preceding page on new types of inversions found in a wild D.m. population of Southern Greece.

Oksala, T.A. University of Turku, Finland. Autosomal nondisjunction produced by an extra Y chromosome in Drosophila melanogaster females.

As early as 1958, the present author (Oksala, 1958) showed that the number of offspring resulting from the cross $w^{m4}/w^{m4}/Y;In(2L+2R)Cy/+$ female x $w^{m4}/Y;+/+$ male was only about half of that from the corresponding cross in which the female did not have an extra Y. The result was explained by assuming that the Y chromosome gave rise to about 50% nondisjunction of the two second chromosomes when they were heterozygous for the two Cy inversions.

In order to demonstrate the assumed nondisjunction ad oculos the following experiment was performed recently in which a second chromosome compound stock was utilized: $w^{m4}/w^{m4}/Y;In(2L+2R)Cy/+$ female x +/Y;C(2L)b;C(2R)vg male. A total of 30 females were mated to about 100 males divided between three culture bottles each containing 10 females and many males. The females were allowed to lay eggs for 15 days during which time they were moved twice into fresh bottles. All offspring consisted, of course, of nondisjunctional flies with phenotypes Cy or b vg as follows:

Cy ♀♀ 270, all with wild eyes
Cy ♂♂ 252, all with mottled eyes
b vg ♀♀ 166, all with wild eyes
b vg ♂♂ 190, all with wild eyes

The result shows that the Y chromosome is effective indeed in causing nondisjunction of the two structurally heterozygous second chromosomes and the original assumption was thus confirmed. In the corresponding cross, in which females without an extra Y chromosome were used, no offspring developed.

References: Oksala, T.A. 1958, Cold Spring Harbor Symp. Quant. Biol. XXIII.

Leigh, B., D.R. Parker* and F.H. Sobels. University of Leiden, The Netherlands. Radiation induced detachment of C(2R)RM chromosomes in immature oocytes.

Within 1-4 hours after hatching, C(2L)RM,j; C(2R)RM,px females were given an exposure of 2kR or 3kR. The irradiated females were aged for one day and then mated to C(2L)RM,dp;F(2R),bw males. Five groups of females were irradiated, the total numbers of progeny being so low that they are all pooled in the second line (I) of Table 1. The progeny from two groups of control females are given in the first line of Table 1.

Spontaneous paternal non-disjunction produces four kinds of gamete; 1) C(2L),dp/0, 2) F(2R),bw/F(2R),bw, 3) C(2L),dp/F(2R),bw/F(2R),bw, and 4) nullo-2. The first two classes

Table 1. Progeny from two groups of control (C) and five groups of irradiated (I) C(2L),j;C(2R),px females mated to C(2L),dp;F(2R),bw males.

	dp px	j bw	dp bw	j px	dp +	j +
C	5	6	-	-	-	-
I	13	12	2	1	18	4

form viable zygotes with regular C(2R),px and C(2L),j gametes, respectively. dp px and j bw were found in both the control and the irradiated series. They occur in a ratio of 1:1. The diplo-2 paternal gametes only form viable zygotes with the corresponding maternal nullo-2 or diplo-2 gametes, dp bw and j px progeny were rare.

Regular paternal gametes were C(2L),dp/F(2R),bw and 0/F(2R),bw, these being able to form viable zygotes with maternal gametes which carried 0/F(2R),px and C(2L),j/F(2R),px, respectively. The maternal F(2R) chromosomes are obtained by detachment of C(2R),px. The data in the last two columns of Table 1 indicate that these detachments are indeed obtained. It is interesting to note that new F(2R) chromosomes are recovered preferentially, but not exclusively, with the paternal C(2L).

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Suchowersky, O., M. Kiess and T.C. Kaufman. University of British Columbia, Vancouver, B.C., Canada. Recovery and partial characterization of X and Y linked temperature-sensitive male sterility on an attached $\overline{XY}$ chromosome in Drosophila melanogaster.

Eighty-six temperature-sensitive (ts) male sterile mutations have been induced by EMS treatment (0.0125 M, feeding) on three differentially marked attached $\overline{XY}$ chromosomes (Table 1). The attachment used was that synthesized by Lindsley and Novitski, 1959) and has Y^S and Y^L attached to opposite ends of the X (X is also inverted, In(1)EN). The rationale for using this chromosome was that after recovery and characterization of the ts lesions, the Y-linked steriles would be more amenable to genetic analysis, e.g., the construction of a double mutant.

Sixty-one of the male sterile mutations are heat-sensitive (hs), that is, they are sterile when raised at 28° but fertile at 22° and 17°C. The remaining twenty-five are cold-sensitive (cs), i.e., sterile at 17° but fertile at 22° and 28°C.

When a normal Y chromosome is added to the genome of a male sterile mutant at the non-permissive temperature to produce an $\overline{XY}$*/Y genotype, a restoration of fertility indicates a Y-linked mutation. By the use of this criteria, 32 of the hs male steriles were found to be Y-linked, while 29 were classified as putative X-linked mutations. It is, of course, possible

Table 1. Results of screen and retests for EMS-induced temperature-sensitive male sterility in $Y^SX.Y^L$, In(1)EN.

Stock	No. of chromosomes tested	Retests for temperature-sensitivity								
		I		II			III		IV	
		hs	cs	hs	cs		hs	cs	hs	cs
y v f B.y^+	1,620	1,058	467	132	16	X	29	2	29	2
						Y	63	14	32	6
						Total	92	16	61	8
y.y^+	2,808	-	79	-	25	X	-	6	-	-
						Y	-	10	-	-
y B	317	-	10	-	8	X	-	0	-	-
						Y	-	3	-	-

that the putative X-linked steriles are actually Y-linked and dominant and tests are currently under way to differentiate between these two possibilities. Similarly, 19 of the cs mutations were designated as Y-linked and eight as being on the X.

Further analysis of the Y-linked steriles by complementation with Y^S and Y^L chromosome fragments, as well as Y chromosomes carrying deficiencies in the seven known fertility factors (Brosseau, 1960) is now in progress. Preliminary results indicate that hs steriles occur more frequently in the long arm, while cs steriles are more frequent in the short. Also, over half of the Y-linked steriles fail to compliment with more than one of the fertility factors, i.e., they appear to be deficiencies. Furthermore, in at least three cases, genes in both arms of the Y seem to be affected, all the more remarkable as they are separated by an entire X chromosome. These results could be explained by the recovery of multiple site ts mutations. However, judging from the frequency of single site ts's, this seems highly unlikely. Alternatively, the result suggests the presence of regulatory genes on the Y and/or X chromosomes which control the activity of several Y loci.

Another interesting and, we think, novel class of mutation has been recovered. This class (there are 7 hs and 4 cs) is characterized by being sterile in the $\overline{XY}$/O condition at the non-permissive temperature. However, when any Y fragment (Y^S or Y^L), Y deficiency (ks2, kl1, kl2, kl3,4 or kl4,5) or sc^8Y is added to the genome ($\overline{XY}$*/Y), the mutation becomes a pupal lethal rather than a sterile at the non-permissive temperature.

In summary, from the initial complementation results and results given in the table, several generalizations can be made: (1) heat-sensitive male sterile mutations are approximately four times as common as cold-sensitive sterile mutations; (2) cold-sensitive sterile mutations tend to localize to Y^S, whereas heat-sensitive steriles are predominantly recovered on Y^L; (3) Y-linked steriles seem to have a high rate of spontaneous recovery of fertility at the non-permissive temperature. This is especially apparent between the third and fourth retests (Table 1) (a period of about 6 months), where almost half of the hs Y-linked steriles

regained fertility. A similar proportion of cs steriles were also lost in this manner. In comparison, the X-linked steriles are much more stable as none regained fertility during the same interval. The apparent instability of the Y-linked mutations could be due to some unique property of loci on the Y chromosome. However, when non-temperature-sensitive Y-linked steriles, induced on an attached $\overline{XY}$ chromosome, are examined, no spontaneous recovery of fertility is observed. Therefore, this phenomenon appears to be a specific property of Y-linked temperature-sensitive mutations. These preliminary results further suggest that the Y chromosome is more complex than the results of Brosseau (1960) would indicate.

Through the use of both cs and hs mutations in different arms of the Y chromosomes, and the synthesis of double hs-cs mutations by means of recombination, it may now be possible to further analyze and order the sequence of gene activity during gametogenesis in the male.

* XY* indicates a ts-bearing chromosome.

References: Brosseau, G.E. 1960, Genetics 45:257-274; Lindsley, D.L. and E. Novitski 1959, Genetics 44:187-196.

(Supported by grants from NRC of Canada and NCI of Canada to D.T. Suzuki.)

Jaenike, J. Princeton University, New Jersey. Estimation of Drosophila population sizes.

Dave's Island, in the Gulf of Maine, is less than one acre and lies about two miles south of Deer Isle, Maine. Twelve banana baits were set up throughout the island, and from them Drosophila were collected from August 15-19 1973. The baits were renewed every other day. The table shows the results. On August 20, all flies were released back onto the island.

August	15	16	17	18	19
algonquin males	6	15	4	2	1
athabasca males	16	16	7	2	3
alg-atha females	25	19	11	5	4
transversa	127	117	160	99	188
putrida	6	12	3	11	9

D. transversa and putrida showed no decrease in capture rate with time, probably because they were emerging in great numbers from mushrooms at that time.

The numbers of D. athabasca and algonquin caught did decline. The results are plotted in Figure 1 on a logarithmic scale, because if each fly has a constant chance of being caught, then the number of flies caught each day should decrease exponentially. Regressions are drawn by eye. Summing the number of individuals expected to be caught on each day over all days gives an estimate of the population size. The estimates obtained are: athabasca males, 47; algonquin males, 32; alg-atha females, 68. Parceling out the females in proportion to the number of males gives: athabasca, 87; algonquin, 60.

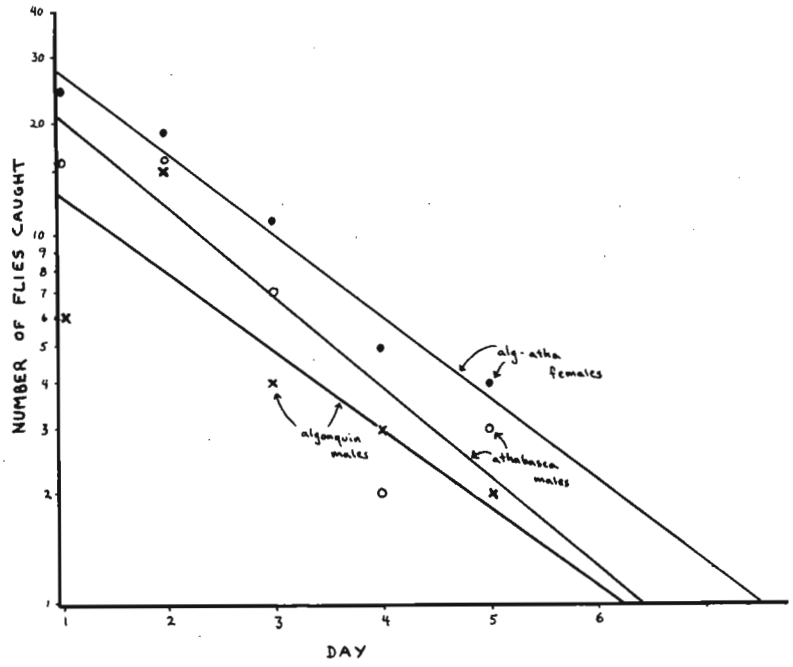


Figure 1. Numbers of Drosophila caught on successive days.

Typically, in Maine, D. affinis subgroup populations increase in density about 5-fold from May to mid-July, at which point they level off. These populations on Dave's Island must indeed be very small.

Aronshtam, A.A., B.A. Kuzin and L.I. Korochkin. Institute of Cytology and Genetics, Novosibirsk, USSR. Some regularities of the phenotypical expression of alleles of gene Est-6 in hybrid D. melanogaster x D. simulans.

The activity of the esterase-6 isozyme which is controlled by alleles of D. melanogaster is inhibited specifically in hybrid males. Both alleles are expressed equally in hybrid females. The differential expression of alleles of gene Est-6 in hybrid males occurs after emergence in all tissues but especially in ductus ejaculatorius - d.e. (L.I. Korochkin, A.A. Aronshtam, N.M. Matveeva, Biochem. Genet. in press).

It was observed on electropherograms of proteins from mass extracts of d.e. that the region of esterase-6 is expressed by two fractions after protein staining in D. melanogaster

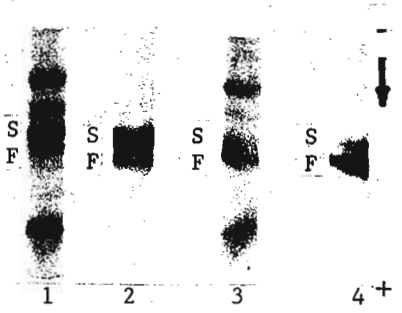


Figure 1

Electropherograms of proteins (1, 3) and enzymograms of allozymes of esterase-6 of hybrid males (2, 4) from reciprocal crosses.

Figure 2

Immunoelectrodiffusion of allozymes of esterase-6 of hybrid males after disc-electrophoresis.

males heterozygotes for the gene Est-6 (see report in this volume A. Aronshtam et al.). It can be seen that these fractions stain equally and correspond to "slow" and "fast" isozymes of esterase-6. In hybrid males density of staining of protein fraction corresponding allozyme D. simulans is denser (Figure 1). It was found by immunoelectrodiffusion technique, that the differential expression of Est-6 alleles is determined by a difference in the amount of enzyme (Figure 2). Using labelled precursors we found that these differences are based on the dif-

Table 1. Incorporation of labelled precursors (protein hydrolysate C^{14}) in allozyme esterase-6 of hybrid males (number of counts in 1 minute).

♂♂ D. mel. Est-6^F/Est-6^F x ♀♀ D. sim. Est-6^S/Est-6^S	allozyme "S"	allozyme "F"
6 hours of exposure	1643	931
24 hours of exposure	2816	1770
48 hours of exposure	3503	2002
♂♂ D. sim. Est-6^F/Est-6^F x ♀♀ D. mel. Est-6^S/Est-6^S		
24 hours of exposure	1840	2640

ferent rate of the synthesis of allozymes (Table 1). It is suggested that some regions of the X-chromosome control the processes regulating the rate of the synthesis of esterase-6.

White, B.N. Queen's University, Kingston, Ont., Canada. An analysis of tRNAs in five Minutes and two suppressors.

It has been suggested that the Minute loci represent the sites of synthesis of tRNAs (Atwood, cited in Lindsley and Grell, 1968). Twardzik et al. (1971) have demonstrated that at least one suppressor locus, su(s)^2, involves a tRNA change. Since the chromatographic pattern of tRNAs in wild-type (Samarkand) flies had been established (White et al., 1973a), it was possible to compare tRNAs from the Minutes, M(3)h^y, M(3)w^{124}, M(2)S7, M(2)Z^B and the temperature sensitive Minute DTS-1 (Holden and Suzuki, 1973), as well as su(s)^2 (White et al., 1973b) and su(pr). All 20 aminoacyl-tRNAs from the Minutes, 8 from DTS-1, and histidine, tryosine, asparagine, and aspartic acid tRNAs from the suppressors were analyzed by reversed-phase chromatography.

If the Minute phenotype results from the decreased production of a particular tRNA, a quantitative change would be expected in the form of a reduced overall acceptance of an amino acid and/or an altered chromatographic profile of isoacceptors. Only one such result was obtained. One threonine isoacceptor (tRNA$_3^{Thr}$) from M(3)h^y was 57-65% that of the wild-type (Figure 1).

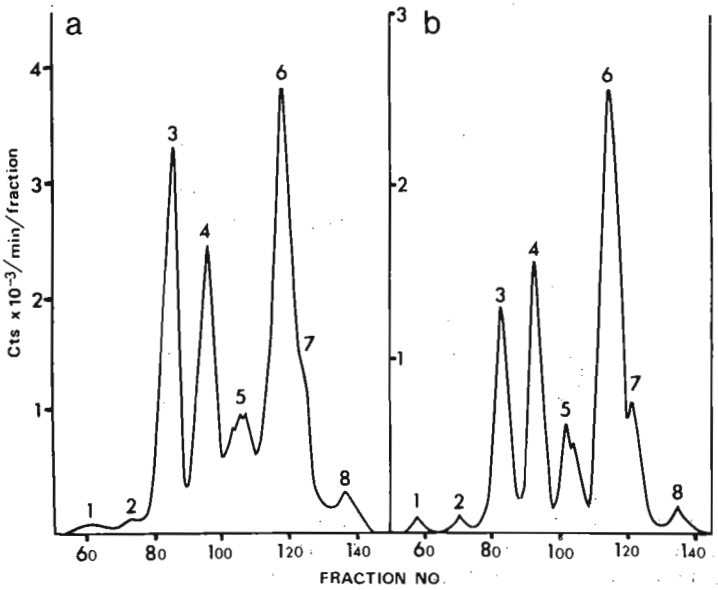


Figure 1. Chromatography of (^{14}C) threonyl-tRNA on reversed-phase 5 columns. a) wild-type (Samarkand); b) M(3)h^y.

After repeatedly crossing this locus into a wild-type background the peak was still only approximately 62% that of wild-type. This result does not prove that the M(3)h^y locus includes the tRNA$_3^{Thr}$ gene but opens the way for further analysis using in situ hybridisation (Grigliatti et al., 1973) and segmental aneuploidy (Lindsley et al., 1972). The remaining 19 aminoacyl-tRNAs from M(3)h^y and all 20 from the other 3 Minutes and the 8 tested from DTS-1 were similar to wild-type. The results suggest that some Minute loci may represent tRNA genes while others could represent genes for other components of the protein synthesizing machinery such as aminoacyl-tRNA synthetases or transfer factors.

Table 1. Quantitative analysis of δ and γ forms of histidine, tyrosine, asparagine and aspartic acid tRNAs.

Source of tRNA	HIS_1	TYR_1	ASN_2	ASN_3	ASN_5	ASP_2
	(percentage of δ form)					
Wild-type	40	36	36	29	39	72
M(3)w^{124}	47	45	42	48	46	79
M(2)S7	43	45	48	47	45	81
M(2)Z^B	58	54	57	61	62	87
M(3)h^y	85	83	81	89	86	96
M(3)h^y*	57	56	-	-	-	81
DTS-1	65	62	-	-	-	88
su(s),^{2}v;bw	88	88	65	88	91	99
su(pr)	59	56	57	59	65	96

* M(3)h^y locus in a wild-type (Samarkand) background.

Only that family of tRNAs with the nucleoside Q in the first position of their anticodons was examined in the suppressors. While there was no significant change in the overall acceptance of these amino acids, there were differences in the distribution of the δ (containing Q) and γ (lacking Q) peaks (Twardzik et al., 1971; White et al., 1973b). Similar alterations were also found in the Minutes (Fig. 2). The results shown in Table 1 express the quantity of the δ peak as a percentage. It is clear that for any one mutant the proportion of the δ peak of $tRNA_1^{His}$, $tRNA_1^{Tyr}$, $tRNA_2^{Asn}$, $tRNA_3^{Asn}$ and $tRNA_5^{Asn}$ is very similar and while the absolute amount of $tRNA_2^{Asp}$ is different the relative change follows the same trend. It is of interest that after the $M(3)h^y$ locus has been crossed into a wild-type background the percentage of δ drops from 85% to 57%.

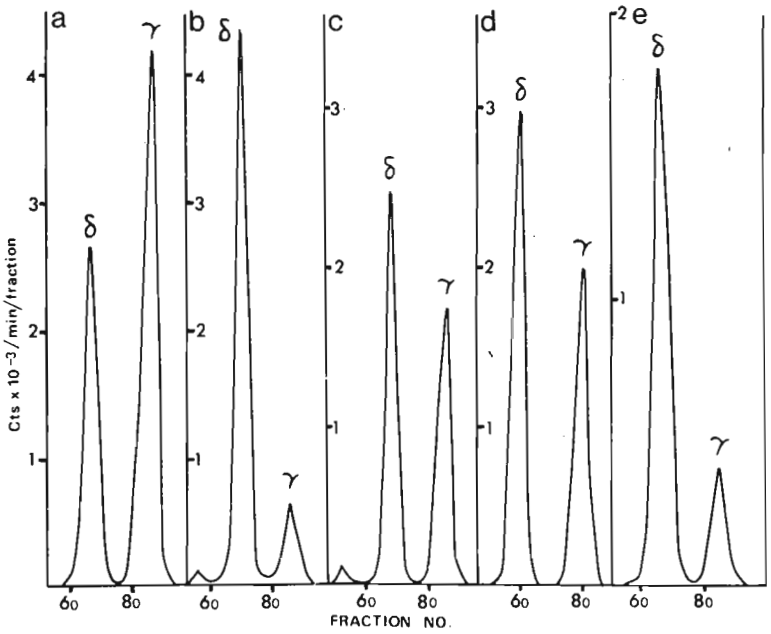


Figure 2. Chromatography of (^{14}C) histidyl-tRNA. a) wild-type (Samarkand); b) $su(s)^2$,v;bw; c) $M(2)Z^B$; d) su(pr); e) $M(3)h^y$.

These data support the model (White et al., 1973b) that the δ and γ forms differ only by the presence or absence of Q and that the proportion of each depends on the activity of a tRNA modifying system that is itself dependent on the genetic background of the fly as expressed by its metabolic state. It appears that various mutations may produce changes in the ratio of δ and γ peaks and therefore that the $su(s)^2$ locus may not be primarily involved in the control of the tRNA modifying system as previously suggested (White et al., 1973b).

Much of this work was carried out in the laboratories of Dr. D.T. Suzuki and G.M. Tener at the University of British Columbia with the technical assistance of C. Kiceniuk and J. Grigliatti.

References: Grigliatti, T.A., B.N. White, G.M. Tener, T.C. Kaufman, J.J. Holden and D.T. Suzuki 1973, C.H.S.Q.B. 38:461; Holden, J.J. and D.T. Suzuki 1973, Genetics 73:445; Lindsley, D.L. et al. 1972, Genetics 71:157; Lindsley, D.L. and E.H. Grell 1968, Carn. Inst. Pub. 627:152; Twardzik, D.R., E.H. Grell and K.B. Jacobson 1971, J. Mol. Biol. 57:231; White, B. N., G.M. Tener, J. Holden and D.T. Suzuki 1973a, Devel. Biol. 33:185; White, B.N., G.M. Tener J. Holden and D.T. Suzuki 1973b, J. Mol. Biol. 74:635.

PERSONAL AND LABORATORY NEWS

William W.M. Steiner is now associated with the Provisional Department of Genetics and Development, 515 Morrill Hall, University of Illinois, Urbana, 61801. Dr. Steiner's laboratory will be investigating relationships between ecological, physiological and genetic parameters in Drosophila and other species of animals to determine the extent of the adaptive significance of biochemical polymorphisms.

R.C. Woodruff has moved to Department of Genetics, University of Cambridge, Milton Road, Cambridge CB4 2X4, England (from Austin, Texas).

David J. Remondini has moved to Department of Biological Sciences, Michigan Technological University, Houghton, Michigan 49931, where he is an associate professor.

Vacek, D.C., O.G. Ward and W.B. Heed. University of Arizona, Tucson. Karyotype of D. micromelanica reared from Gambel oak in Arizona.

Two females and four males were reared from a Quercus gambelii slime flux collected in October 1971, at an elevation of approximately 8,000 feet on Mt. Lemmon in the Catalina Mts. north of Tucson, Arizona. Larval brain neuroblast chromosomes showed four pairs of rods, one pair of V's and one pair of dots. Three pairs of rods were autosomal and of equal length. The fourth pair of rods were sex chromosomes and were slightly longer. The X was longer than the Y and had a dot satellite. The Y had an elongated satellite. Of the four known metaphase strains of D. micromelanica, this description corresponds to strain III previously known only from Cave Creek and Kingman, Arizona (Stalker, 1965).

Reference: Stalker, H.D. 1965, Genetics 51:487-507.

Binnard, R. Ames Research Center, NASA, Moffett Field, California. Prolonged anesthesia of Drosophila melanogaster using metofane.

Metofane (methoxyflurane, Pittman-Moore) is a very effective anesthetic for small vertebrates, and in our laboratory, has proved equally effective for anesthetizing Drosophila when a prolonged treatment is necessary. The procedure is as follows: 0.5 of metofane is transferred to the cotton in a 1/2 pint anesthetizing bottle and left to vaporize for 10 minutes. Then the flies are shaken in and gently rotated for 5 to 7 minutes. This treatment will keep the flies anesthetized for about 1 hour.

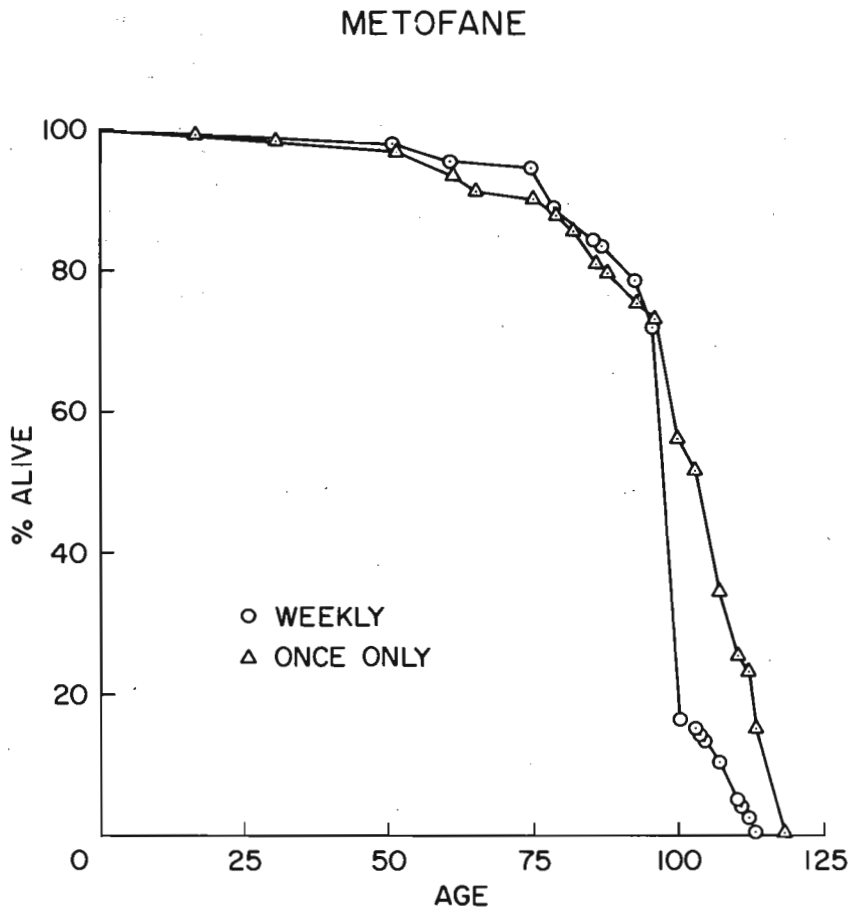


Figure 1. Survival curves of D. melanogaster males (Oregon R). O - Flies that were exposed to metofane once for 2 minutes at 2 days of age. This curve is practically identical to that shown by control flies. Δ - Drosophila exposed to metofane for 2 minutes every week starting at 2 days of age. There is a slight life shortening.

As shown by Figure 1, anesthetization with metofane once in the life time of Drosophila does not exert any detrimental influence on the survival curve of the flies. Only when anesthesia is performed every week does slight life shortening occur.

Murnik, M. Rengo and G.L. Cybul. Western Illinois University, Macomb, Illinois.

Increased recovery of X-bearing sperm after cold shock.

Mature sperm were given 24 hour temperature shocks of 15°C, 17°C, 22°C or 29°C either in the seminal receptacle of cn bw females following insemination by Oregon-R males or while in the seminiferous tubules of mature males immediately prior to mating. The 17°C shock caused a significant elevation in the recovery of females, while all other temperature treatments produced no significant effects upon sex ratio. These results suggest the presence of a threshold temperature for sensitivity to cold shock in mature sperm.

Nutrient medium, modified after Carpenter[1], was used in all experiments. In addition, a minimal medium was used during shock treatment of newly inseminated females in order to inhibit oviposition[2,3]. All females used were three to five days old. In administering tempera-

Table 1. Progeny resulting after temperature shock to sperm within the seminal receptacle.

		15°C	22°C	29°C
Replicate 1	Female	536	328	344
	Male	474	271	273
	% Female	53.07	54.76	55.75
Replicate 2	Female	357	422	246
	Male	297	359	228
	% Female	54.59	54.03	51.90
Replicate 3	Female	448	505	356
	Male	412	477	313
	% Female	52.09	51.42	53.21
Replicate 4	Female	733	724	253
	Male	605	654	222
	% Female	54.78	52.53	53.26
Replicate 5	Female	498	450	378
	Male	439	467	380
	% Female	53.14	49.07	49.86

Chi-square analysis revealed no significant differences between treatments in all replicates.

Table 2. Progeny resulting after temperature shock to mature sperm within the seminiferous tubules.

Treatment	Total Flies	Females	Males	% Females
17°C	3185	1715	1470	53.85
22°C	5094	2620	2474	51.43
29°C	4149	2125	2024	51.21

17°C vs. 22°C*
17°C vs. 29°C*
* Significant difference ($P < .05$)

Table 3. Progeny resulting after temperature shock to mature sperm within the seminiferous tubules.

Treatment	Total Flies	Females	Males	% Females
15°C	2540	1362	1178	53.62
22°C	4511	2416	2095	53.56
29°C	4224	2303	1921	54.52

Chi-square analysis revealed no significant differences between treatments.

ture shocks to sperm within the seminal receptacle, Oregon-R males and females were aged together for four and one-half days at 25°C. Males were then collected, given a 12 hour recovery period after etherization, and placed individually in shell vials with one cn bw virgin female on minimal medium for 12 hours. The males were then removed without etherization, and the females were treated at the experimental temperatures on minimal medium for 48 hours. After shocking, the females were placed together in pint-sized milk bottles containing nutrient medium and transferred to fresh bottles after days one, two and five. After day eight the females were discarded. In shocking mature sperm within the seminiferous tubules, newly eclosed OR-R males eight hours or less in age were incubated for 24 hours at the experimental temperatures, then individually mated to one cn bw female in a shell vial for 12 hours at 25°C. All transfers were done without etherization.

Table 1 summarizes the results of temperature shock to sperm within the seminal receptacle. A test for homogeneity of means for binomial populations indicated that the replicates could not be treated together. This is probably due to the small numbers of progeny recovered in each replicate. No consistent patterns in sex ratio change were evident for the broods of a particular treatment when all replicates were considered, therefore only total results are presented. In all replicates no significant differences in sex ratio between the various treatments were obtained.

Tables 2 and 3 summarize the data obtained after temperature shock to mature sperm within the seminiferous tubules. There is a significant ($P < .05$) increase in the sex ratio (expressed as per cent females) of the progeny of those males treated at 17°C compared with those treated at 22°C and 29°C replicates.

References: 1. Carpenter, J. 1950, DIS 24:96; 2. Offerman, C.A. and I.K. Schmidt 1935, DIS 3:52-54; 3. Trosko, J. and M. Myszewski 1963, DIS 37:146.

Craymer, L. University of Wisconsin, Madison. A new genetic testing procedure for potential mutagens.

Standard mutagenicity tests are not designed for efficient detection of agents affecting breakage (repair) or disjunction; as a result, such agents are likely to have their effects pass undetected. The following test should facilitate detection of these effects:

A T(1:Y) is used in which the X-chromosome break is medially located, and which is viable and fertile in males with no additional Y chromatin. Males of genotype T(1:Y)/0 are crossed to free-X bearing females. Regular progeny die prior to eclosion as a result of aneuploidy; exceptional progeny are produced by any of the following events: 1.) nondisjunction in females; 2.) nondisjunction in males, either a.) prior to second meiotic division or b.) during second division; 3.) breakage and subsequent loss of one of the elements of the T(1:Y); or 4.) recombination in males between X heterochromatin and Y heterochromatin. These classes of events should be independent, and with appropriate markers it is possible to differentiate all events, excepting 2b.) from 3.).

Several T(1:Y)'s were obtained from John Merriam for possible use in this system, of which three proved to be male-fertile with no additional Y chromatin: J2 (9A:Y^S), J8 (8C:Y^L), and B49 (11D:Y^L). These translocations carry y^+ and B^S as terminal markers for Y^S and Y^L; however, only T(1:Y)B49 had retained the B^S. A cross of yellow females with T(1:Y)B49 males produced 95 survivors out of an estimated 50,000 zygotes; a breakdown of these survivors as to phenotype, presumed composition of the XY structure derived from the father, and the event(s) giving rise to this structure are given in the following table. An additional point

Offspring from the cross of y females by T(1:Y)B49:

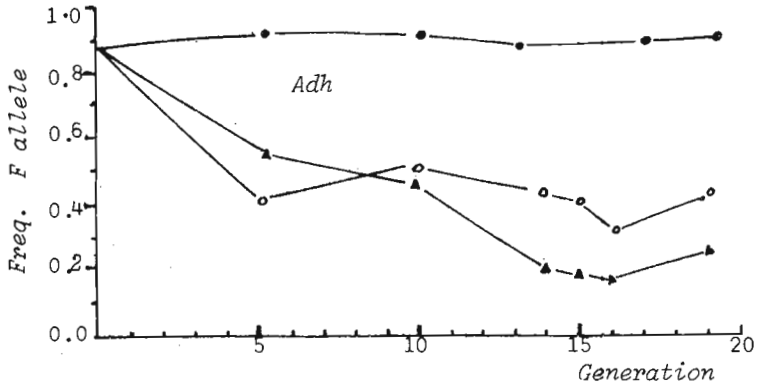
Phenotype	y^+B^S ♀♀	y ♀♀	yB^S ♀♀	y^+ ♂♂	yB^S ♂♂	y^+ Dp ♀♀
Observed	10	52	12	7	1	13
XY	T(1:Y)	0	$X^{D}Y^{LP}.X^{P}Y^{S}$	y^+Y^S.	$B^S(Y^L).?$	$X^{D}Y^{P}(y/y)$
Cause	2a.)	2a,b.), 3.)	4.)	4.)	4.)	1.)

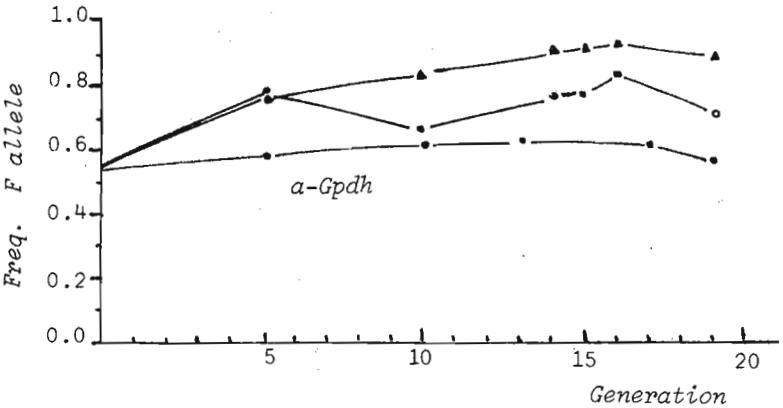
revealed by this cross is that aneuploids (larvae appear to be y) survive to pupate and die prior to eyespot formation; this provides a well-worked culture, but these larvae are too sickly to provide competition for the viable genotypes.

Alahiotis, S. University of Patras, Greece. Isozyme genotype-environment relationship in artificial cage populations originated from a Southern Greek D. melanogaster natural population.

The study of the genotype-environment relationship is very important in determining the allele frequency patterns (F.M. Johnson et al. 1969, 1973). In the present preliminary communication the selection effect of two environmental factors upon the same gene pool is reported; these factors were food and humidity. Three cage populations were started from six hundred common parents, derived from one hundred isofemale lines, collected in the summer of 1973, from the Greek island Cephalonia. Cage population (IA) was put under 25 ± 0.5°C temperature, 43% mean relative humidity, and in corn-meal-sugar-agar food medium. Cage population (ID) was treated under the same food medium and temperature conditions, but in 90% relative humidity, while population (IB) was put in 25 ± 0.5°C temperature, 43% relative humidity and in dead yeast-sugar food medium.

Samples of about 100 flies were taken at different intervals (see Figures 1, 2) and were analyzed.





Figures 1, 2. Changes in the frequencies of the Adh^F and α-$Gpdh^F$ alleles over 19 generations in the populations IA (▲) ID (o) and IB (●).

In order to characterize the genotype of individual flies starch gel electrophoresis was carried out at 300 volts, 100 mA for one hour using 0.5 M trisversene-borate buffer (pH 8.0). Gels were sliced horizontally and were stained in their lower half according to the method followed by C. Shaw and Prasad (1970), while for their upper half we used Johnson's and Denniston's staining method (1964).

Figures 1, 2 show the changes in frequency of Adh^F and α-$Gpdh^F$ alleles respectively during 19 generations (each generation under the previously mentioned conditions was estimated equal to about 15 days).

The origin of curves indicates the frequency of alleles, in the natural population from which the flies were captured. It can be seen that the changes in the frequency of the alleles Adh and α-Gpdh were dramatically rapid in the (IA) and (ID) populations, while there were no significant differences in the (IB) population.

The rapid allele frequency changes found for both loci are strongly in favor of an action of selection. Berger (1971) also seems to have obtained selection in laboratory populations towards the frequency found in nature at the α-Gpdh, Adh and Mdh-1 locus in Drosophila melanogaster. Further investigations on this matter are still in progress.

References: Berger, E.M. 1971, Genetics 67:121-136; Johnson, F.M. and C. Denniston 1964, Nature 204:906-907; Johnson, F.M., H.E. Schaffer, J.E. Gillespy and E.S. Rockwood, 1969, Bioch. Gen. 3:429-450; Johnson, F.M. and H.E. Schaffer 1973, Bioch. Gen. 2:149-163; Shaw, C.R. and R. Prasad 1970, Bioch. Gen. 4:297-320.

Heisenberg, M. Max Planck-Institut für biologische Kybernetik, Tübingen, Germany.
Isolation of autosomal mutants with defects in the optomotor response.

Five second chromosome mutants with visual abnormalities have recently been obtained. Wild type "Berlin" males were treated with EMS to give about 1.5 recessive lethal mutations per second chromosome. To make homozygous stocks for single treated chromosomes, two DTS-balancer chromosomes kindly provided by E.V. Falke (Falke and Wright 1972) were used. Flies from 400 stocks were tested for the optomotor response to broad stripes ($\lambda = 45^{o}$) at a light intensity close to the low threshold in a single-fly test apparatus described by Heisenberg and Götz (in preparation). Each of the 5 mutants belong to a separate complementation group. Some mutant properties are summarized in the following table.

mutant	electroretinogram (ERG)		deep pseudopupil		stationary optomotor response to stripe patterns with	
	RP	LP	shape	color	$\lambda = 45^{o}$	$\lambda = 7.2^{o}$
A68	+	-	+	(+)	small	small
A289	+	-	-	-	thresh. shift	-
A944	+	-	+	(-)	thresh. shift and small	-
A1709	(+)	+	+	+	thresh. shift	+
A1761	+	+	+	+	(+)	(+)

abbreviations: RP - receptor component of the ERG; LP - lamina potential

Measurement of the ERG and observation of the deep pseudopupil serve to check the optical and physiological properties of the retina. The receptor component of the ERG is found in all mutants. In A1709 however it seems to be smaller than in wild type. This slight sensitivity loss in the retinula cells R1-6 is apparently reflected in the upward shift of the low threshhold intensity of the optomotor response to broad stripes which in fact may be the only defect in this mutant. Conversely in the mutant A289 the receptor component of the ERG seems to be normal but shape and coloration of the deep pseudopupil are disturbed. This indicates that the rhabdomeres might be disoriented. Again, the deficiencies of the optomotor response in this mutant can probably be accounted for by these structural defects in the retina. The suppression of the lamina potential however seems to be an additional artefact.

In the mutant A944 the shape of the deep pseudopupil is normal but the amount of screening pigment in the retinula cells decreases towards the dorsal (and possibly also the temporal) part of the eye. If present, the pigment migrates normally in response to light intensity changes. In this mutant as in the mutant A68 the detectable retinal disturbances seem too small to explain the suppression of the lamina potential and the severe defects in the optomotor behavior observed. Neuronal defects at some level proximal to the retina have to be assumed.

The mutant A1761 is an exception: freely walking flies show a disturbance in the optomotor response which is reliably measured in the single-fly-test-apparatus. However under open-loop conditions the defects are not very apparent.

Recently M.C. Deland (personal communication) discovered that the mutant H-53, which had been isolated earlier in a search for sex-linked visual mutants in this laboratory, is also located on the second chromosome. H-53, which is described elsewhere (Heisenberg and Götz, in preparation; Heisenberg, in preparation), complements each of the mutants above.

Reference: Falke, E.V. and T.R.F. Wright 1972, DIS 48:89.

I thank Dr. M.M. Green for genetic advice.

Gassparian, S. and M.R. Vasseghi. University of Isfahan, Iran.
New mutants from Kashan City.

From six regions in Kashan, named: Fin, Zeidabad, Hossein-Abad, Lathorn, Shadian and Darb-Esfahan, 97 mutants were detected. Among them wing deformities seems to be common in all populations, but modifications were very low. Some mutants affect more than one characteristic in the phenotype. It seems that the origin of six micropopulations are the same, each of which showed variations after genetical recombinations.

Jefferson, M.C., W.R. Johnson Jr., D.G. Baldwin and W.B. Heed. University of Arizona, Tucson. Ecology and Comparative cytology of Drosophila on San Pedro Nolasco Island.

On March 20-22, 1974, the genus Drosophila was sampled by one of us (M.C.J.) on San Pedro Nolasco, an island 16 kilometers from the nearest mainland north of the coastal town of Guaymas, Sonora, Mexico (28° N latitude) in the Gulf of California. Gentry (1949) described San Pedro Nolasco as a rocky island approximately 3.5 square kilometers in area and reaching a height of 315 meters. Thirty heavy plastic bags containing about 1800 gm of fermenting bananas were placed 9 meters apart in a steep canyon in the middle of the island. The results of evening collections by aspirating from banana baits are shown in Table 1.

Table 1. Drosophila fauna of San Pedro Island

Species	No. collected	Probabl host plant
D. mojavensis	432	Lemaireocereus thurberi
D. pseudoobscura	7	†Agave chrysoglossa and Opuntia
D. nigrospiracula	4	Pachycereus pringlei
D. sp. M.	2	Pachycereus pringlei
D. arizonensis	2	†Opuntia wilcoxii (bravoana)
D. aldrichi	2	†Opuntia wilcoxii (bravoana)
D. melanogaster-simulans	1	?
Total	450	

† Identified by Dr. Richard S. Felger

In comparison to the mainland, San Pedro Nolasco was quite dense in vegetation. Most plants at this time of the year were flowering on the island, while on the mainland soil conditions were dry and few plants were in flower. This perhaps can be explained by the presence of fog surrounding the island during the early morning and late evening. Table 1 also shows 3 species of cacti found on San Pedro Nolasco that serve as host plants for the majority of these species of Drosophila on the mainland. One decaying arm of an organ pipe cactus (Lemaireocereus thurberi) was brought back to the laboratory and 126 D. mojavensis were reared from it. The agave is abundant and could support D. pseudoobscura also. The small number of specimens is typical of many desert collections.

Chromosomes were analyzed for two of the species collected. One larva from each of 92 D. mojavensis isofemales were fixed for LP (La Paz) on the 2nd and ST (Standard) on the 3rd chromosome as described by Mettler (1963). Cytologically, the island population is like that of north-central i land Sonora, which is marginal for the species. Most populations of coastal Sonora are similar but are heterozygous for MU (Mulege) on the 3rd chromosome from Desemboque (4%) to Guaymas (18%). Four populations in Baja California have been found to be polymorphic for as many as 6 gene arrangements (W.R. Johnson, unpublished).

Seven larvae from 5 isofemales of D. pseudoobscura were analyzed to determine the karyotypes of both parents. The 1 male collected was crossed to an AR-8 female, and 7 larvae were analyzed. Also 1 female was crossed to AR-8, and 7 larvae were analyzed. In contrast to D. mojavensis, the third chromosome of D. pseudoobscura was found to be highly polymorphic: 62.5% AR, 25% SC, 8.3% ST, and 4.2% CH, (N=24). The X-chromosome produced 88.9% wild-types and 11.1% sex-ratio (SR), (N=18), (determined by D.G.B.). Cytologically, the island sample is unique and a mixture of the population from northern Baja California where SC is present and ST is in high frequency, and to the population from coastal Sonora where SC is absent and AR and CH are in high frequencies, (Heed, unpublished Dobzhansky, 1951). This may be the result of either multiple invasions or unique ecological conditions.

It is interesting that the large population (D. mojavensis) is monomorphic and the presumably small population (D. pseudoobscura) contains 6 gene arrangements.

References: Dobzhansky, Th. 1951, Genetics and the Origin of Species. N.Y., Columbia Univ. Press; Gentry, H.S. 1949, Allan Hancock Pacific Exped. 13:81-180 Part I; Mettler, L.E. 1963, DIS 38:57-58

Pelecanos, M. and S. Alahiotis. University of Patras, Greece. Time of action of a series of recessive lethal mutations induced by diethyl sulphate (D.E.S.) in the second chromosome of D. melanogaster.

The embryonic and postembryonic development of Drosophila appears to be particularly sensitive to genetic disturbances at certain stages (P.J. Bryant and G. Schubiger 1971). Hadorn (1961), in a review of earlier work, concluded that there is a clustering of deaths at four periods: The early embryo, the periods just before or after hatching of the embryo as well as those just before or after the puparium formation and the eclosion of the adult. On the other hand, despite the wide mention and use of both spontaneous and induced lethals, very little is known of their time and manner of action. In view of the specific action shown by diethyl sulphate (M. Pelecanos 1966, 1971) a study of the time of action in a series of recessive lethal mutations induced by the previously mentioned mutagen in the second chromosome of Drosophila males was carried out.

Newly-hatched wild type (Oregon-K) Drosophila males were starved for 24 hours and then treated for another 24 hours with 0.5% diethyl sulphate by an adult feeding method (M. Pelecanos and T. Alderson 1964). Two types of experiments were carried out; in the first experiment (L_1) each treated male was allowed to mate with two virgin females for nine days, while in the second (L_2), for three days only. Thus, in the first case the lethals were predominantly induced in mixed post-meiotic germ cells (spermatozoa, spermatids and secondary spermatocytes), while in the second experiment all lethals derived from treated spermatozoa. Autosomal recessive lethal mutations were detected by means of the Cy L^4 technique.

In order to determine the time of action of the lethals induced by D.E.S. treatment, the outcross-backcross method described by Seto (1954) was used. This method is sometimes more reliable than the balanced lethal method, since the complications due to Cy are avoided and only the 1/1 lethal effect is detected. Cy L^4/1 males were mated to virgin cn bw (cinnabar-brown) females, and from this mating non Curly-Lobe males were backcrossed to Cy L^4/1 females. In the next generation four classes of zygotes are formed cn bw/Cy L^4, 1/cn bw, 1/Cy L^4 and 1/1, the last of which dies. The stage characterized by a large number of deaths is the time at which the lethal action occurs. Males of the cn bw/1 genotypes were allowed to mate for three days with Cy L^4/1 virgin females. Then, ten to fifteen 4-5 day old couples of the previously mentioned genotype, were transferred into ordinary Drosophila culture vials containing a medium of the following composition: Glucose 100g, dried killed yeast 100g, agar 30g and distilled water up to 1000ml. The medium was darkened by adding a small quantity of charcoal; care was taken to maintain the medium's surface horizontal in order to ensure, as much as possible, the uniformity of egg-laying. The previously mentioned couples were kept for 4 hours in the vials, thus allowing the females to lay their eggs during this period, and then they were again moved into new vials for the next four hours. The same procedure continued until 36 hours of egg collection was completed. After discarding the parents, the collected eggs were counted and 24 hours later were classified as either being "white" or "brown". All white unhatched eggs were considered to be "unfertilized" while brown eggs were taken as unhatched "fertilized" eggs. The stage distribution of mortalities was estimated on the basis of the method used by Wright (1973) as follows:

1. Total "fertilized" eggs = total eggs transferred - unhatched white eggs.
2. Unhatched "fertilized" eggs = brown eggs = total unhatched eggs - unhatched white eggs.
3. % embryonic mortality = unhatched "fertilized" eggs/total "fertilized" eggs x 100
4. % larval mortality = hatched eggs - pupae/total "fertilized" eggs x 100
5. % pupal mortality = pupae - adults/total "fertilized" eggs x 100
6. % total mortality = total "fertilized" eggs - adults/total "fertilized" eggs x 100

It should also be added that in the case of the L_2 experiment (see Table 2) a control was also carried out.

Our results are presented in three tables. Tables 1 and 2 show the stage distribution of mortalities in the L_1 (mixed germ cells) and L_2 (spermatozoa) experiments respectively. Table 3 provides the possibility of comparing the frequencies of lethals found in the two experiments over the stages examined. The classification of lethal effects was clustered in one of the following stages: E = egg, E/L = egg - larval boundary, L = larva, L/P = larval-pupal boundary and P = pupal. If the letters are in parenthesis they indicate additional deaths in other than the principal stage of death. Table 3 shows that the highest frequency of lethals, in both experiments, occurs in the stage of larva. Statistical analysis, for 2XN table, of the lethal frequencies has shown that if N is expressed in numbers, no statistically significant difference was found between the two experiments ($\chi^2_4 = 3.976$ and $P > 0.05$). On the contrary, if N is expressed in frequencies percent, the difference is statistically significant

Table 1. Stage distribution of mortalities in the L_1 (mixed germ cells) experiment.

No of lethals examined	No of eggs laid	Mort. of E%	Mort. of L%	Mort. of P%	Total Mort.	Time of action (stage)
1	266	11.2	11.2	16.2	38.6	E/L/P
2	427	12.0	14.0	3.0	29.0	E/L
3	197	-	43.2	7.8	51.0	L/(P)
4	98	2.1	12.6	24.2	38.9	L/P
5	152	13.3	28.1	0.7	42.1	E/L
6	158	11.9	23.8	-	35.7	E/L
7	527	1.6	15.5	11.6	28.7	L/P
8	354	0.6	29.9	2.9	33.4	L
9	160	32.0	30.1	1.3	63.4	E/L
10	186	2.8	7.3	4.0	14.1	L
11	534	1.0	23.7	3.5	27.2	L
12	151	21.8	5.8	-	27.6	E
13	326	2.3	27.3	3.0	32.6	L
14	251	8.1	11.4	4.8	24.3	E/L
15	595	7.1	28.6	1.1	36.8	(E)/L
16	137	2.3	12.1	13.6	28.0	L/P
17	292	0.7	21.9	18.8	41.4	L/P
18	171	1.8	24.0	4.2	30.0	L
19	215	3.0	39.1	3.6	45.7	L
20	121	1.8	25.5	0.9	28.2	L
21	435	1.0	12.1	3.6	16.7	L
22	268	3.3	32.2	11.2	46.7	L/P
23	344	2.7	45.6	15.0	63.3	L/P

Table 2. Stage distribution of mortalities in the L_2 (spermatozoa) experiment.

No of lethals examined	No of eggs laid	Mort. of E%	Mort. of L%	Mort. of P%	Total Mort.	Time of action (stage)
1	119	10.1	15.7	4.5	30.3	E/L
2	76	8.7	23.8	-	30.5	(E)/L
3	247	-	26.7	1.7	28.4	L
4	190	-	55.3	1.6	56.9	L
5	277	3.1	18.1	13.1	34.3	L/P
6	138	3.9	26.4	2.3	32.6	L
7	158	17.7	24.1	1.3	43.1	E/L
8	135	6.1	22.6	2.6	31.3	L
9	114	4.0	24.0	1.0	29.0	L
10	72	5.7	11.4	14.3	31.4	L/P
11	238	5.2	20.3	4.3	29.8	L
12	151	1.4	18.5	3.4	23.3	L
13	194	10.9	24.8	3.0	38.7	E/L
14	142	2.2	27.7	5.1	35.0	L
15	168	4.3	13.4	12.2	29.9	L/P
16	156	6.6	21.9	5.8	34.3	L
17	320	1.3	32.2	2.0	35.5	L
18	151	1.4	40.1	15.6	57.1	L/P
19	157	5.6	14.1	2.1	21.8	L
20	213	4.3	15.8	2.7	22.8	L
21	130	20.0	20.8	1.5	49.3	E/L
22	137	7.0	21.0	5.5	33.5	(E)/L
23	82	1.3	28.8	3.8	33.9	L
24	230	2.7	35.9	0.5	39.1	L
25	230	14.7	10.1	4.1	28.9	E/L
26	129	-	27.9	17.2	45.1	L/P
27	389	4.6	16.6	0.3	21.5	L

28	332	6.4	15.0	0.4	21.8	L
29	646	15.7	9.4	7.6	32.7	E/L/P
30	185	-	29.4	1.1	30.5	L
31	344	0.6	14.5	17.8	32.9	L/P
32	211	5.1	18.7	3.0	26.8	L
33	569	3.5	22.6	0.7	26.8	L

Table 3. Compared frequencies of lethals and stage distribution in the experiments (L_1L_2)

Type of Experiment	E No	E %	E/L No	E/L %	L No	L %	L/P No	L/P %	P No	P %	E/L/P No	E/L/P %	Total No of l. examined
L_1 Mixed g. cells	1	4.3	6	26.3	8	34.8	7	30.4	-	-	1	4.3	23
L_2 Spermatozoa	-	-	7	21.2	19	57.6	6	18.2	-	-	1	3.0	33

($\chi^2_4 = 13.825$ and $P > 0.01$). The same observation stands in the case in which we compare the frequencies of percent obtained in the stages L and L/P of the two experiments (see Table 3). Thus, for the L stage $\chi^2_2 = 10.46$ and $P > 0.01$ while for the L/P stage $\chi^2_2 = 4.05$ and $P > 0.05$. This discrepancy is obviously due to the small number of the lethal stocks analysed. However, the significant differences found when N is expressed as a percentage encourages further studies; thus, in order to assess whether or not the stage of peak sensitivity (spermatozoa) to the mutagen affects differently the distribution of lethals during the ontogenetic development of Drosophila, as compared with the other post-meiotic germ cell stages, more data as well as fractional sampling of the germ cell stages are needed.

The outcross-backcross method used for determining the time of action of lethals provides a theoretically expected total mortality equal to 25 percent. Yet, with the exception of two lethal stocks in the L_1 experiment, where the observed total mortality is lower than the expected one, all the other stocks, in both experiments, have shown a higher than the expected total mortality (see Table 3). These deviations may be attributed to many different factors; however, it seems to us that the main reason is very probably the fact that D.E.S. induces "mosaic lethals". The factors responsible for the higher than the expected total mortality in most of the stocks analyzed are much more difficult to attribute predominantly to one factor among a series of several probable ones. However, here again the induction by the mutagen of more than one lethal at a time favours the hypothesis that a synergistic interaction between lethal genes in heterozygous condition may have played an important role.

Our findings may be summarized as follows: The effective lethal phase in both experiments, in which second chromosome recessive lethals were induced by diethyl sulphate was found to be the larval stage. Polyphasic lethals were also detected. There is some evidence, though not yet established, that the stage of peak sensitivity to the mutagen may affect the spectrum of the distribution of lethals during the ontogenetic development of Drosophila melanogaster. Finally, in most of the stocks analyzed, the total mortality was found to be higher than the expected one.

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Weber, L., B. Yedvobnick and E. Berger. University of Connecticut, Storrs and SUNY at Albany, New York. Linear redundancy in Drosophila virilis.

Measurements of reiteration frequency for 18S + 28S r-RNA cistrons were made for D. virilis (Texmelucan) and D. melanogaster (Swedish-B) using filter hybridization to adult DNA. Saturation plateaus for the two species were reached at 0.36% and 0.42%, respectively, using homologous r-RNA. Using either direct filter binding, or competition hybridization, the level of sequence homology between the two species was found to range between 82%-90%. Melting pro-

files of homologous and heterologous hybrids indicated a higher level of homology, about 94%, assuming that a reduction of 1°C in the Tm 1/2 reflects a 1.5% difference in base sequence.

The intriguing feature of the data is that when the number of 18S + 28S r-RNA cistrons is calculated (Table 1) the value for D. virilis is nearly twice that of D. melanogaster. The

Table 1. Estimate of the number of 4S, 5S, and 18S + 28S RNA genes per haploid genome of D. melanogaster of D. virilis.

D. melanogaster: haploid genome mol. wt. 113×10^9 d.

	4S	5S(1)	5S(2)	28S + 18S
% DNA hybridized	.013	.011	.008	.42
DNA hybridized (d)	14.7×10^6	12.8×10^6	8.5×10^6	474×10^6
Mol. wt. of RNA (d)	2.4×10^4	4.0×10^6	4.0×10^4	2.1×10^6
Number of genes	612	320	213	226

D. virilis: haploid genome mol. wt. 235×10^9 d.

	4S	5S(1)	5S(2)	28S + 18S
% DNA hybridized	.008	.004	.002	.36
DNA hybridized (d)	18.8×10^6	9.9×10^6	3.5×10^6	846×10^6
Mol. wt. of RNA (d)	2.4×10^4	4.0×10^4	4.0×10^4	2.1×10^6
Number of genes	783	247	86	403

similarity in saturation level, but difference in copy number, arises from the two-fold difference in somatic cell DNA content between the two species (Kavenoff and Zimm, 1974). The excess DNA in D. virilis is primarily due to three highly repeated heptanucleotide sequences known as satellite DNA which constitute about half of the D. virilis genome (Gall et al., 1973).

We next attempted to determine the level of reiteration for 4S and 5S sequences in these species. We used as the DNA source embryonic tissue from D. virilis (DNA was kindly supplied by Dr. Blumenthal at University of Wisconsin) and adult tissue from D. melanogaster. As the RNA source we used high specific activity material (110,000 CMP/μg RNA) obtained by labelling cell cultures of D. melanogaster. Saturation levels for 18S + 28S r-DNA in D. virilis embryo DNA with D. melanogaster r-RNA were about 0.3%, after correction for 85% homology.

Preliminary experiments on 5S r-DNA indicate complete homology between the two species. Melting profiles of either species' DNA with D. melanogaster H^3 5S-RNA were nearly identical with Tm 1/2 values of 72.5°C. Saturation levels in two different experiments were reached at 0.004% and 0.002% for D. virilis, and 0.011% and 0.008% for D. melanogaster. The lower values in the second experiment are probably the most accurate since the RNA used there was twice purified on acrylamide gels. The range of values for the number of 5S sequences (Table 1) indicates, then, that either the number of copies are similar in the species, or that D. virilis contains significantly fewer genes.

Studies on 4S RNA indicate that the number of tRNA genes is about 30% greater in D. virilis (Table 1) although we have not determined whether sequence homology is complete. Thus the 30% excess is a minimum value.

The results suggest that in D. virilis the accumulation of satellite DNA has been accompanied by a two-fold increase in the redundancy of 18S and 28S r-DNA. The number of 5S genes has not changed and the number of 4S genes has increased only slightly. The increase of 18S + 28S genes may represent a physiological compensating mechanism for increased genome and, perhaps, cell size; or may simply reflect the physical proximity of these cistrons to the centric heterochromation where the satellites reside. We are presently studying relative rates of synthesis for the 4 RNA species discussed using acrylamide gels (Weinmann, 1972) and thus far find the relative ratio of rates to be similar in both species.

This research was supported by an NIH grant CEB.

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Katoh, S. and Y. Arai. Josai Dental University, Sakado-machi, Saitama-ken, Japan. Occurrence of neopterin in mutant sepia of Drosophila melanogaster.

The purine ring of guanine has been shown to be a precursor for unconjugated pteridine biosynthesis in insects[1,2] and, as generally demonstrated in the animal kingdom, dihydroneopterin (2-amino-4-hydroxy-6-tridroxypropyl-7,8-dihydropteridine) is presumed to be the first product in this case. Drosophila melanogaster contains a high concentration of total unconjugated pteridine with a large variety of pteridines in various tissues. Recently, guanosine-5-phosphate (GMP)-C^{14} and neopterin-C^{14} were fed to larvae of D. melanogaster (ry^2 and Ore-R) and subsequently the pteridines were analysed from the extract from whole flies[3]. GMP and neopterin were found to be effective precursors of biopterin and 2-amino-4-hydroxy-pteridine in that study. But the natural occurrence of dihydroneopterin (or its oxidized form, neopterin) has not been yet reported in D. melanogaster. In this study, we have isolated neopterin from mutant sepia of D. melanogaster in which large amounts of sepiapterin and biopterin (but not drosopterins) are accumulated.

Three hundred g of whole adult se flies were extracted with 3 vol of hot 95% ethanol. The concentrate of extract was applied to two continuous ECTEOLA cellulose columns (OH type, pH 8.0, 7 x 20 cm in first, 5 x 30 cm in second) and further two continuous phospholyrated cellulose columns (H type, 3 x 30 cm in first, 3 x 45 cm in second). All columns were developed with distilled water. And neopterin corresponding fraction was separated from contaminated biopterin through the last column of ethanolyzed cellulose (3 x 40 cm) eluted by n-propanol-ethyl acetate-water (7:1:2). The blue fluorescent compound obtained was identical with authentic neopterin when compared by absorption spectra (λ_{max} (nm): 247 and 320 in 0.1N HCl; 235 and 363 in 0.1N NaOH)[4], and by paper chromatography (Rf (authentic neopterin was given in parentheses): 0.10(0.10) in n-propanol-ethyl acetate-water (7:1:2); 0.21(0.22) in isopropanol-water (7:3); 0.33 (0.33) in n-propanol-1% ammonia (2:1); 0.15(0.16) in 95% ethanol-n-amyl alcohol-water (7:5:3), by ascending method).

Table 1. Comparison between neopterin from se flies and four optical isomers of authentic neopterin by paper chromatography.

compounds	moving distance (cm-40 hr at room temperature)
neopterin (from se)	10.6
neopterin (D-erythro-)	10.6
(L-erythro-)	10.7
(D-threo-)	6.7
(L-threo-)	6.3
biopterin	18.8
6-carboxypterin	3.1

There are four optical isomers of neopterin: D- and L-erythro- and D- and L-threo-neopterin. Neopterin obtained in this study was erythro- form from the results of paper chromatography by descending development with isopropanol-5% boric acid (4:1)[4] (Table 1). By the facts that GTP(GMP) is considered to be metabolized to D-erythro- isomer of dihydroneopterin and that natural neopterin found in insects is also D-erythro- isomer, neopterin obtained from se flies may also be D-erythro- isomer presumably presented as dihydro- form in vivo. The occurrence of neopterin in sepia mutant suggests that sepiapterin is metabolized via neopterin (probably dihydro- form) from GTP(GMP) as mentioned in biopterin of D. melanogaster (ry^2 and Ore-R).

References: 1) Weygand, F., H. Simon, G. Dahms, M. Waldschmidt, H.J. Schliep and H. Wacker 1961, Angew. Chem. 73:402; 2) Watt, W.B. 1967, J. Biol. Chem. 242:565; 3) Sugiura, K. and M. Goto 1967, Biochem. Biophys. Res. Commun. 28:687; 4) Remboldt, H. and H. Metzger 1963, Chem. Ber. 96:1395.

Gassparian, S. and H. Gahremannejad. University of Isfahan, Iran. New mutants from Isfahan Province in Iran.

During fourteen months of investigation, a total of 48 mutants were observed in three isolated regions, Isfahan, Asgaroon and Daran, located in Isfahan province.

The affected parts of fruit flies were as follows: eye color, wing shape, abdomen shape, body color. Only dark red eye color was common in the three populations. It seems that isolation barriers strongly affect them.

Ivanov, Y.N. Institute of Cytology and Genetics, Novosibirsk, USSR. Unstable conditions of singed mutations in D.m.

We extracted three spontaneous mutations of the gene singed in October 1973. Two alleles under arbitrary numbers 63-15 and 77-27 arose in wild type X-chromosomes isolated from the natural population Tashkent (Middle Asia). A third allele 45-7 occurred in the X-chromosome of the stock Muller-5 on the background of the same Tashkent population. These mutations are interesting because of their various stabilities. The mutation 77-27 possesses the highest rate of reversion (sn → "+") giving phenotypically normal conditions which have high rates of reversion to singed ("+" → sn). These new singed alleles occurring from the pseudonormal alleles "+" were named contrareversions. The contrareversions, in turn, mutate easily into the unstable pseudonormal alleles. The allele 63-15 is similar to the allele 77-27 though the former mutates more rarely. However, we have not found the reversions of the allele 45-7. All alleles are maintained by crossing mutant males to females y w f having attached-X chromosomes. The table contains the data about the back and contraback mutation rates of the alleles 77-27 and 63-15. Obviously, the frequencies of mutations from 77-27 to "+" and back are identical. Nearly all reversion flies in the stock 63-15 have some bristle abnormalities. Their bristles can be faintly bent, deviate slightly

Mutation rates of singed alleles in opposite directions.

Crosses ♀ y w f	Phenotypes of males sn	"+"	Total	Direction of mutation	Rate of mutation
Allele 77-27					
x ♂ sn	275	29	304	sn → "+"	9.5×10^{-2}
x ♂ rev. ("+")	2	55	57	"+" → sn	3.5×10^{-2}
x ♂ contrarev. (sn)	399	35	434	sn → "+"	8.1×10^{-2}
x ♂ contra-contrarev. ("+")	3	29	32	"+" → sn	9.4×10^{-2}
Allele 63-15					
x ♂ sn	206	4	210	sn → "+"	1.9×10^{-2}
x ♂ rev. ("+")	1	458	459	"+" → sn	0.2×10^{-2}

from their usual direction and at times have characteristic loops on their ends. The mosaic males which have normal and singed bristles at the same time were found in the stocks 63-15 and 77-27. All male offspring from crosses ♀ y w f x ♂ mosaic were either singed on phenotype or wild type partly. The stocks homozygous for the alleles 63-15 and 77-27 were obtained. The singed females also are unstable mutationally. Probably selection supports pseudonormal alleles because the stock 77-27 for four months had lost singed males; all males were of the wild type. A few unstable alleles of the gene singed with similar properties were found by Dr. Raisa L. Berg in the natural populations of the Transcaucasus and the Crimea in the same autumn, 1973. At present the nature of the mutationally unstable sn gene is not clear.

Dow, M.A. and F. von Schilcher. University of Edinburgh, Scotland. Rape in D. melanogaster.

Forced matings have been reported to be fairly common in D. melanogaster (1). For evolutionary reasons, it is important to know whether rape exists and if so to what extent. We have set up an experiment to determine this, using very young females and both winged and wingless males of a recently collected strain which has been maintained outbred. Young females are unreceptive and therefore copulations which occur should be forced (1). Furthermore, copulations involving wingless males which are incapable of producing courtship song should also be forced. Tests were conducted with 450 pairs of flies. Three hour old females were tested with both wingless and winged males, while one hour old females were tested with only winged males. Males were 3 days old. Females were collected without anaesthetization. Ten pairs of flies were placed per shell vial and observed for 150 min. Two copulations were observed, both with 3 hr. old females, one with a winged and the other with a wingless male. We therefore conclude that rape is possible but not frequent, its frequency being about 0.5% (2/450).

Reference: (1) Manning, A. 1967, Anim. Behav. 15:239-250.

Atidia, J. and S. Baker. Hebrew University of Jerusalem, Israel. Localization of a taste perception mutation of D.m.

The X-linked mutation Lot-94 affects the salt perception of Drosophila melanogaster, so that flies carrying it are less sensitive to salts than the wild-type. Under standard conditions (4-5 day old flies, starved and dessicated for 3 hr. at 28°C, fed for 20 min. at 28°C) only some 5% of the wild-type males and 3% of wild-type females consume a screening solution of 1 M NaCl + 0.1 M sucrose (with a red food coloring added to detect consumption). On the other hand, about 90% of the Lot-94 males, 30% of the heterozygous females and 50% of the homozygous females consume this solution.

The progeny of the backcross Lot-94/y^2 v f car x y^2 v f car/Y were scored for salt consumption under the described standard conditions. At the first stage the mutation Lot-94 was located to the interval between v and f.

For a further analysis a taste index (P) was constructed from the difference between the proportion of consumers among the mutants (p_1) and among the wild-type flies (p_2). The interval v-f is divided by Lot-94 into two sectors at a ratio of d and (1-d), so that among recombinants between v and f the proportion of flies consuming the screening solution would be:

$$r_1 = p_1 d + p_2(1-d) \text{ for recombinants v non f males, and}$$

$$r_2 = p_2 d + p_1(1-d) \text{ for the reciprocal recombinants (f non v males).}$$

The taste index of the reciprocal recombination groups r_1-r_2=R is a function of the location of Lot-94 in the interval. Consequently the relative position of the mutation may be calculated: d = (R+P)/2P. In three experiments with a total of 5157 flies a value of d = 0.95 was determined, putting Lot-94 at 55.5. In another experiment with 1663 flies, recombination between Lot-94 and the markers v g f Bx was studied. Lot-94 divided the interval g-f so that d = 0.55, i.e., Lot-94 should be located at 51.2.

Finally a sample of the recombinants f non v male progeny from the first experiment were mated individually to attached-X females. Examining their sons for the consumption of the screening solution allowed an unequivocal determination whether the flies were Lot-94 or Lot-94^+. Only 11 out of 324 f non v checked recombinants were Lot-94, placing the mutation 0.8 cM to the left of f. Lot-94 was placed tentatively between 55.5 and 56.0. We hope to determine the exact location by using deficiencies.

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Chinnici, J.P. Virginia Commonwealth University, Richmond, Virginia. Effect of temperature on crossing over in the distal regions of the X-chromosome of Drosophila melanogaster.

As part of another experiment dealing with the effect of monosodium glutamate on meiotic crossing over in the X-chromosome of D. melanogaster, tests with controls were run at three temperatures - 15°C, 25°C and 30°C. Comparisons of the control values at these three temperatures are presented below to show the effect of temperature alone on crossing over in the distal regions of the X-chromosome.

Two stocks of D. melanogaster were used in the experiments: a standard "wild type" laboratory stock of Oregon-R, and a "mutant" stock homozygous for four X-chromosome recessive genes: scute bristles (sc, 1-0.0), crossveinless wings (cv, 1-13.7), singed bristles (sn^3, 1-21.0) and miniature wings (m, 1-36.1). The mutant stock was constructed in early 1967 and has been maintained since by mass culture. The culture medium used was a standard yeast, dextrose, agar medium supplemented with several inorganic chemicals. Half pint milk bottles containing control medium were placed in an incubator at 15°C, 25°C or 30°C. In these bottles mutant females were mass mated with wild type males and offspring were allowed to develop. From these offspring, 25 heterozygous females from each of the three different treatment bottles were separately mated in individual shell vials with mutant males from the same treatments. Each female was two days old when mated, having been kept at 25°C since eclosion. Each female was allowed to lay eggs for a single six day period, these offspring being allowed to develop at 25°C. The offspring were then scored to determine (1) the rate of crossing over for the sc-cv, cv-sn^3 and sn^3-m intervals of the X-chromosome in the female parents, (2) fecundity of the female parents, and (3) percentage of male offspring produced. These data are presented in Table 1. Crossover percentages were determined as discussed by Chinnici (1971).

Table 1. Effect of temperature on female crossing over, fecundity and sex ratio of the offspring.

Treatment[1] Temperature	No. of offspring $\bar{X} \pm s$	% ♂ offspring $\bar{X} \pm SE$	% crossing over and 95% confidence limits sc-cv	cv-sn^3	sn^3-m	sc-m
15°C	116.80 ± 25.18**	48.88 ± 1.05	14.8 *	9.7***	19.1 *	45.3***
			12.7-17.1	8.6-10.9	17.7-20.5	43.4-47.3
25°C	133.08 ± 25.84	47.60 ± 0.49	12.4	6.2	16.9	35.8
			11.4-13.5	5.3-7.1	15.5-18.4	33.5-38.2
30°C	133.40 ± 17.30	49.44 ± 0.94	9.8***	5.7	14.0***	29.9***
			8.8-10.9	4.8-6.6	12.7-15.2	27.9-31.9

1 25 females of each treatment were tested; offspring grown at 25°C
* $0.025<P<0.050$ by analysis of variance = treatment vs 25°C
** $0.010<P<0.025$ by analysis of variance = treatment vs 25°C
*** $P<0.010$ by analysis of variance = treatment vs 25°C

These data may be summarized as follows: 1) The frequency of male offspring produced is not affected by temperature.

2) At low temperature (15°) there is a significant increase in crossing over in all three regions of the X-chromosome analyzed and in the total sc-m region.

3) At high temperature (30°) there is a significant decrease in crossing over in the sc-cv and sn^3-m regions of the X, and in the total sc-m region.

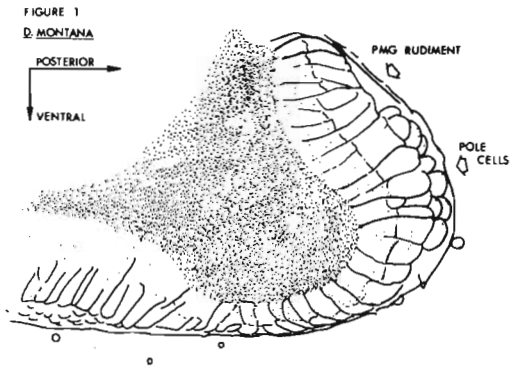
Discussion: In the X-chromosome of female Drosophila melanogaster, the effect of temperature on crossing over appears to depend on the region of the chromosome studied. Crossing over in the proximal regions of the X increases as a result of larvae grown at continuously high temperature (Stern 1926) or if a high temperature shock is received around the time of pupation or later (Grell 1966; Chandley 1968; Hayashi and Suzuki 1968). This is also true for the centromeric regions of the autosomes (Plough 1917, 1921; Grell and Chandley 1965). However, for the distal regions of the X-chromosome, there are seemingly conflicting reports of the effect of temperature on crossing over. Plough (1921) reported that growth of female larvae at a constant temperature of 31.5°C had no effect on subsequent crossing over in the sc to f regions of the X-chromosome. Chandley (1968) found that a 34°C heat shock for 24 hours given to newly emerged virgins resulted in a decrease in crossing over in the sc-v region of the X. Grell (1973), however, has found that a 35°C heat shock for 12 hours given between 132 and 144 hours of larval development (early oocyte formation including meiotic interphase and DNA replication) results in an increase in crossing over in all regions of the X (proximal > distal > interstitial regions in magnitude). A similar heat shock given between 114 and 120 hours of larval development (pre-oocyte and pre-meiotic DNA replication) results in a decrease in crossing over in all regions of the X (interstitial > proximal > distal in magnitude) except the extreme distal region (sc-cv) which shows an increase in crossing over. Grell also reports (1973) that such heat shocks given between 150 and 198 hours of larval development have no significant effect on crossing over. The effect of low temperature on crossing over has not been studied since Plough's (1917) observation that larval growth at temperatures between 9°C and 17.5° produced increased crossing over in the proximal region of chromosome 2.

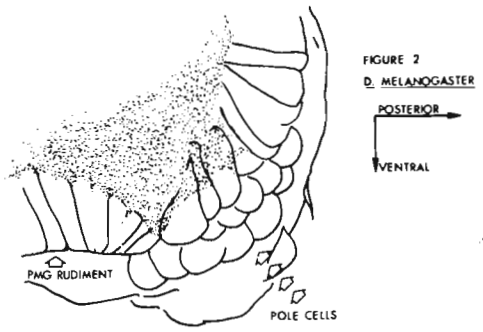
The results of the present experiment indicate a negative correlation between the temperature at which female larvae grow and the amount of crossing over subsequently measured in distal regions of the X-chromosome. The increase in crossing over at low temperature (15°C) is consistent with Plough's (1917) observations for chromosome 2, and the decrease at high temperature (30°C) is consistent with Chandley's (1968) observations for distal X regions but inconsistent with the observations of Plough (1921) and Grell (1973). The reason for this confusing state of affairs is not understood, but may be due in part at least to the fact that none of these experiments are strictly comparable in terms of stocks used, chromosome regions followed for crossing over, or techniques employed.

References: Chandley, A.C. 1968, Mut. Res. 5:93-107; Chinnici, J.P. 1971, Genetics 69:71-83; Grell, R.F. 1966, Genetics 54:411-421; _____ 1973, Genetics 73:87-108; Hayashi, S. and D.T. Suzuki 1968, Can. J. Genet. Cytol. 10:276-282; Plough, H.H. 1917, J. Exp. Zool. 24:147-210; _____ 1921, J. Exp. Zool. 32:187-202; Stern, C. 1926, Proc. Nat. Acad. Sci. 12:530-532.

Miyamoto, D.M.[1], W.L. Rickoll[2], and S. Fullilove[3]. (1 & 2) Duke University, Durham, North Carolina ([1]Zoology and [2]Anatomy) and (3) University of Nebraska, Omaha (Biology). Differences in posterior midgut invagination in D. montana and D. melanogaster embryos.

Recently we had the opportunity of comparing time lapse films of the normal embryonic development of D. montana (subgenus Drosophila, species group virilis) made by Janet Kinsey and A.G. Jacobson (University of Texas, Austin) and of D. melanogaster (subgenus Sophophora, species group melanogaster) made by Ede, Counce and Lucey (1956, "The Embryonic Development of D. melanogaster"*). It was noted that the site and manner of posterior midgut (PMG) invagination and of inclusion of the pole cells within the lumen of the extending midgut differed strikingly in the two species. In D. montana, the posterior midgut rudiment first appears as a thickening of the blastoderm dorsad to the pole cells (Figure 1), while in D. melanogaster, the rudiment is ventrad to the pole cells (Figure 2). (Figures 1 and 2 represent optical sagittal sections taken from the two films and are of





the posterior ends of the embryos just after the first indications of invagination of the rudiments can be identified). As the rudiment invaginates in D. melanogaster it scoops up the pole cells as described by Ede and Counce (1955), while in D. montana the pole cells appear at first to follow the rudiment as it moves anteriorly during germ band extension. Several other clear differences between the two species in the morphogenetic behavior of cells in equivalent regions of the embryos were also noted during early stages of embryogenesis, but have not yet been completely analyzed. Since these two species represent two major subgenera of the genus Drosophila, such developmental differences are interesting phylogenetically; in addition, comparative studies of such differences offer the possibility of interesting insights into the cellular basis of morphogenesis.

* Available commercially from the Institute of Animal Genetics, Edinburgh.

Reference: Ede, D.A. and S.J. Counce 1955, Roux Arch. Entwickmech. 148:402-415.

Barker, J.F. University of the Witwatersrand, Johannesburg, South Africa. Genetics of Drosophila seguyi.

During 1969-70, natural populations of D. seguyi were sampled in Southern Nigeria. In most banana bait trapping, small samples only of this species were obtained. D. seguyi is polymorphic in the female only, for posterior abdomen colour (dark - light). Thus for example, among females of this species trapped on 21-4-70 in the Gambari Forest, 7 were dark, 8 light, and one doubtfully dark. Preliminary breeding tests carried out in Nigeria in 1970 (to the F_2 generation) gave results largely consistent with the hypothesis that the polymorphism is controlled by a pair of autosomal alleles, dark being dominant to light, the polymorphism being sex limited. In 1973 experiments were started in South Africa using stocks which the writer originally established in Nigeria. Breeding experiments are continuing and so far support the above hypothesis. Also, large cultures, when analysed by breeding, seem to have the genotypic proportions expected from the Hardy Weinberg equilibrium on the basis of the above hypothesis. Selection experiments are being carried out and the material is proving useful for teaching population genetics.

Gateff, E. Biologisches Institut I der Albert Ludwig Universität, Freiburg, Germany. Effects of a high X-ray dose upon the primordia of the adult integument, the adult optic centers and the gonads in the embryo of D. melanogaster.

Drosophila larvae and pupae of different developmental stages, exposed to high X-ray doses, show a variety of stage specific effects in the imaginal discs (Waddington, 1942). The developmental effects of a high X-ray dose on the progenitor cells of the imaginal discs, the adult optic centers in the larval brain and the gonads were investigated in 2, 4, 7, 13 and 17 hour old embryos. The irradiation dose was 4000 r units. The X-rays were generated at 195 kV and 10 mA. Table 1a,b,c shows the results from three experimental approaches.

Table 1. X-ray irradiation of whole, anterior and posterior halves of Drosophila melanogaster embryos.

Age of irradiated embryos in hours	2-3	4-5	7-8	13-14	17-18
a. Number of totally irradiated embryos	90	90	90	90	90
Number of developed embryos	14	14	28	74	70
Developmental stage reached			prepupa		
b. Number of anteriorly irradiated embryos	90	90	90	90	90
Number of developed embryos	14	16	14	60	54
Developmental stage reached			prepupa		
c. Number of posteriorly irradiated embryos	90	90	90	90	90
Number of developed embryos	20	16	17	74	77
Developmental stage reached			adult		

When 90 embryos of each age group were subjected to a total irradiation dose of 4000 r units, two phenomena were observed: 1. The mortality rate was higher in younger than in older embryos, and 2. all embryos that survived developed into third instar larvae which formed normal puparia, but died within them as prepupae (Table 1a). Anatomical and histological investigations of these larvae, revealed the absence of all imaginal discs, the imaginal optic primordia in the larval brain and the gonads. Their phenotype, anatomy and behavior at pupation, resembled closely that of lethal mutant larvae lacking imaginal discs (Shearn et al., 1971). Apparently the high X-ray dose interfered with the development of the imaginal primordia of ectodermal origin, but had no effect on the development of the larval structures.

In order to determine whether the radiation acts directly upon the imaginal discs or upon some other factor, anterior and posterior halves of embryos in different developmental stages were irradiated with 4000 r units (Table 1b,c). In all age groups, irradiation of the anterior halves was fatal at the prepupal stage. Anatomical investigations of larvae developed from frontally irradiated embryos 2, 4 and 7 hours of age, showed in parallel to the totally irradiated embryos, absence of all imaginal discs, the adult optic primordia in the larval brain and the gonads. Larvae derived from 17 hour old frontally irradiated embryos showed in some cases more or less well developed eye-antennal imaginal discs and small abnormal genital imaginal discs. All other imaginal discs, such as the leg, the wing and the halterer were completely missing. Female gonads were never found, while male gonads were sometimes present.

Irradiation of the posterior embryonic half, in contrast, did not prevent adult development (Table 1c). The adult animals, most of which could not eclose because of missing external genitalia and the few last abdominal segments, were checked for the presence of gonads. No gonads had developed in 100% of the cases.

Thus, the strong X-ray dose applied to the posterior half of the embryo had only a local effect and did not influence the development of the imaginal primordia located in the anterior portion of the embryo. In contrast, irradiation of the anterior half of the embryo, resulted in developmental halt of all imaginal primordia of ectodermal origin and the gonads. The above results indicate that within the anterior half of the Drosophila embryo X-ray sensitive factors seem to be present, which affect the development of all imaginal primordia of ectodermal derivation and the gonads.

References: Waddington, C.H. 1942, J. Exp. Biol. 19:101-117; Shearn, A., T. Rice, A Garren and W. Gehring 1971, Proc. Nat. Acad. Sci. 68:2594-2598.

The able help of Mrs. M. Klug is greatfully acknowledged.

Sidhu, N.S. Indian Veterinary Research Institute, Izatnagar, India. A note on studies on motile and non-motile filaments (mitochondrial) in spermatozoan tail of Drosophila and their separation during spermatozoan movements.

Motile and non-motile filaments of spermatozoan tails of various species of Drosophila have been observed so as to study their dissociation during spermatozoan movements. The tail membrane appears to be having a longitudinal slit on one side. Tail's motile filament consists of $9_3 + 9_2 + 2_1$ fibrils (Sidhu, 1963, 1970). The non-motile filaments which are two, one primary and the other secondary, so-called because of the difference in their size (Sidhu, 1963, 1970) in Drosophila species, are nebenkern derivatives, these being of equal size in certain insect species. In Drosophila species, the motile (fibrillar) and the non-motile (mitochondrial) filaments get separated in the anterior region of the tail as is seen from the electron microscopic and ultraphotomicrographic studies reported in this note. This perhaps may be a natural phenomenon in Drosophila species, some of which are known to have the longest spermatozoan lengths known in living organisms (Sidhu, 1963, 1970).

Electron micrographs and ultraphotomicrographs taken during the year 1962 at the Institute of Animal Genetics, Edinburgh, have been utilized for the observations reported now.

The motile and non-motile filaments of Drosophila spermatozoa get separated primarily in the proximal part of the tail (Figure 1). This fact is observed in Drosophila species having long spermatozoan tails, e.g., immigrans, funebris. Electron micrographs of other species, like melanogaster, as well as those belonging to the obscura group, have also given such impressions and one perhaps may generalize this phenomenon. Transverse sections as well as

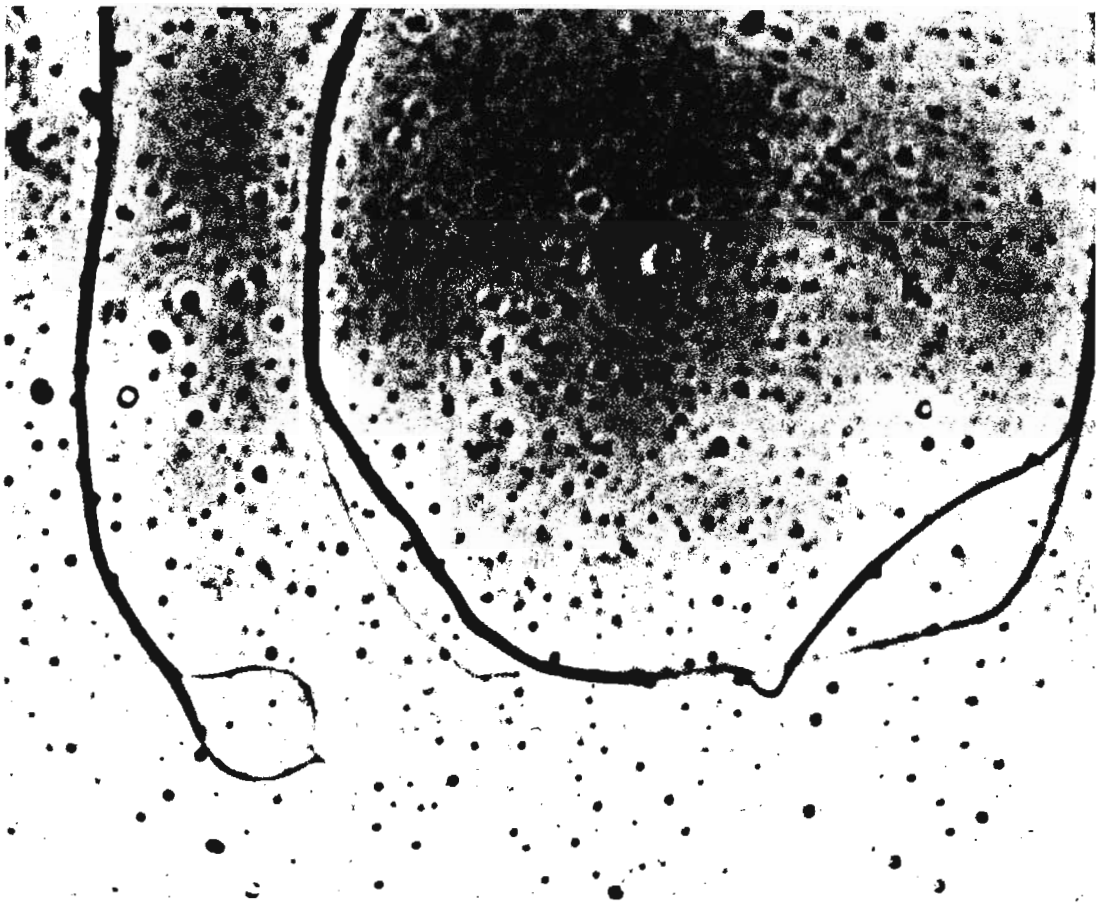


Figure 1. Ultraphotomicrograph of Drosophila spermatozoa showing the motile and non-motile filaments apart in the anterior region of tail.

longitudinal sections of spermatozoa as seen in the electron micrographs (Figure 2, 3) show the two filaments derived from the nebenkern to be apart from the motile (fibrillar) filament in a good number of spermatozoan tails out of all seen sectioned at various regions. The spermatozoan sections of D. immigrans are shown depicting the stated observations

Figure 2. Electron micrograph of transverse sections of spermatozoa of D. immigrans showing motile and non-motile filaments of a number of spermatozoa sectioned at appropriate region of the tail where these filaments get separated due to a slit in the tail sheath along its length. (X 34,500).

(Figures 2, 3) also seen in the other species.

A suspension electron micrograph of an anterior part of the spermatozoan tail of subobscura (Figure 4) also confirms the separation of the motile and non-motile components.

The electron micrographs used by other authors in their papers have been re-examined recently to check whether such a separation of the two components of spermatozoan tail can be seen. Electron micrographs of Macroglossim stellatrum (Lepidopteran insect)(Andre, 1961) depict the discontinuity of tail membrane. Similarly, electron micrographs of spermatozoan tails of Fire-brat insect, Thermobia domestica Pack (Bawa, 1964) also confirm the existence of discontinuity in the membrane. Nebenkern derived motile filaments are of equal size in Acheta (Clayton et al., 1958), Chortippus (Sjöstrand and Afzelius, 1956) and Gelastorrhinus (Yasuzumi and Ishida, 1957) but of unequal size in Drosophila species, e.g., melanogaster, obscura, subobscura, persimilis, pseudoobscura, funebris, and immigrans (Sidhu, 1963, 1970). The present work shows an important aspect to the relationship of motile and non-motile filaments of spermatozoan tails in Drosophila that at least for a part of the tail

Figure 3. Electron micrograph of spermatozoa of D. immigrans showing longitudinal sections as well as transverse sections of spermatozoa. Tail membrane shows discontinuity making the nebenkern derived mitochondrial filaments (non-motile) and fibrillar non-motile filament free. Motile filament also shows its contractile phenomena. (X 21,200).

Figure 4. Suspension electron micrograph of a part of the spermatozoan tail of D. subobscura showing motile and non-motile filaments separated. (X 8,000).

length, there seems to be a provision for the fibrillar motile filament to come out of the tail membrane and to act in a way which perhaps may help spermatozoan movements. The membrane covering the spermatozoan tail region appears to have a longitudinal slit for a certain length at least, allowing the motile filament to get separated from the rest of the tail. Such a phenomenon perhaps is common to all insect species in their spermatozoan tails; the nebenkern derived non-motile filaments run for nearly the whole length. Further confirmation of the reported observation is, however, required to come to the above conclusion.

References: Andre, J. 1961, J. Ultrastruct. Res. 5:86-108; Bawa, S.R. 1964, J. Cell Biol. 23(3):431-446; Beatty, R.A. and N.S. Sidhu 1969, Proc. Roy. Soc. Edin. (B)71:14-28; Sidhu, N.S. 1963, Ph.D. Thesis, University of Edinburgh; ____ 1970, Ind. J. Hered. 2(1):15-38.

Hofmanová, J. J.E. Purkyně University, Brno, Czechoslovakia. The effect of the genetic background on the recombination frequency in the cn-vg region of the second chromosomes extracted from natural populations of D. melanogaster.

The samples of four natural populations each of them containing ten males, were used. In these 40 chromosomes the recombination frequency was studied on the different backgrounds in three replications.

The following backgrounds were used: 1. M-5/M-5 first chromosomes and Me Sb e/H e third chromosomes. 2. Both first and third chromosomes of the wild type ⊕/+, where that marked by ⊕ was from the studied natural population and the other + was isolated from the Oregon-R stock. 3. The background was analogical as in the second case, but instead of Oregon-R stock, the Samarkand was used.

In all calculations each crossover percentage was transformed into an angular value. An analysis of variance (Table 1) showed the largest effect of genetic background and a smaller, nevertheless highly significant effect ($P<0.01$), of the other sources of variation, including single and double interactions between backgrounds, populations and males.

Table 1. Analysis of variance for recombination frequency in D. melanogaster

Sources of variation	d.f.	M.S.	F
Males (A)	9	6.28	2.79
Populations (B)	3	31.87	14.19
A x B	27	8.16	3.63
Backgrounds (C)	2	131.99	58.76
A x C	18	7.34	3.27
B x C	6	9.33	4.15
A x B x C	54	5.42	2.41
Replications (D)	2	0.80	0.36
A x D	18	2.28	1.02
B x D	6	2.91	1.29
C x D	4	1.76	0.78
A x B x D	54	2.40	1.07
A x C x D	36	1.50	0.67
B x C x D	12	1.69	0.75
Residual	108	2.25	
Total	359		

Using the random hypothesis, a weak effect of males in comparison with populations and backgrounds and also a small share of populations in comparison with backgrounds was found. Significant differences ($P<0.01$) were found between means of all three backgrounds. The highest values of recombination frequency were obtained on the background with inversions. Also individual populations showed different reactions on the substitution of the special genetic background.

The variability of recombination frequency in the second chromosomes from individual populations on the different backgrounds was also demonstrated by means of the variation coefficients, which suggested specific influence of the genetic background also on the variation of recombination frequency in this special part of the second chromosome.

The differences between backgrounds suggested the interchromosomal effect of the inversions used and the effectivity of the different genetic factors in the first and third chromosomes of the Oregon-R and Samarkand stocks or their interaction with natural first and third chromosomes. Differences between populations may be due to different modifying crossover factors, most probably of a polygenic character (Chinnici, 1971a, b and Kidwell, 1972 a, b).

References: Chinnici, J.P. 1971a, b, Genetics 69:71-96; Kidwell, M.G. 1972a, b, Genetics 70:419-433.

Leibovitch, B.A.[+], V.A. Gvozdev[+], I.F. Zhimulev* and E.S. Belyaeva*. [+]Kurchatov Institute of Atomic Energy, Moscow and *Institute of Cytology and Genetics, Novosibirsk, USSR. Disproportionate incorporation of ^{3}U-UTP and ^{3}H-ATP in some regions of D.m. salivary chromosomes in E. coli RNA polymerase reaction mixture.

Late third instar larvae and 0 hrs. prepupae of wild stocks Oregon-R-C and Swedish were used. Squashes of salivary gland chromosomes were prepared as described early (1). Glands were dissected in Ephrussi-Beadle medium, were then fixed in ethanol-propionic acid 5 min. and squashed in 45% acetic acid, frozen in liquid nitrogen for coverslip removal and transferred to water through 95%, 70% and 50% ethanols. A drop of solution containing E. coli RNA polymerase, unlabeled three phosphates, ^{3}H-UTP (sp. act. 1.68 Ci/mM) and another component (2) was placed on the squash preparation. Another experiment has been performed to test the effect of actinomycin D binding presumably with G-C pairs, as generally accepted. Chromosomes on slides were incubated in E. coli RNA polymerase reaction mixture with ^{3}H-ATP (sp. act. 3.4 Ci/mM) and actinomycin D (100 microg/ml). Both incubations have been carried out in a moist chamber. After 30 min at 25°C slides were immersed in 5% TCA and washed in water. The slides were covered with "M" liquid emulsion (Niichimphoto, Moscow) and exposed for 14 days.

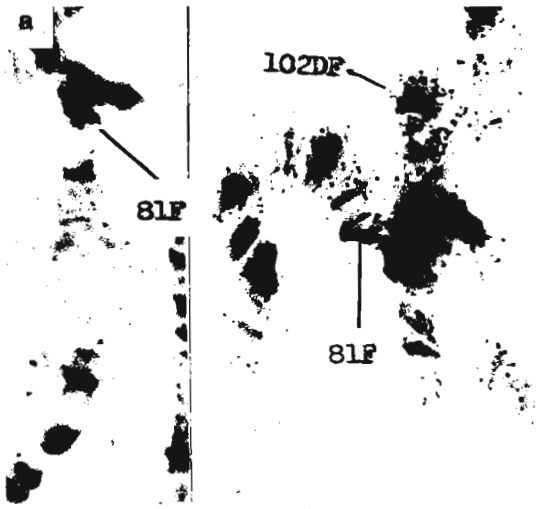


Figure 1. Disproportionate incorporation of ^{3}H-UTP in 81F, 102D and 102F regions (arrows) of polytene chromosomes of Oregon-R-C larvae. a - 0 hrs. prepupae; b - 118 hrs. larvae. Gimsa staining.

It was demonstrated recently that in E. coli RNA polymerase reaction mixture the labeling is proportional to DNA content of the chromosome regions (3). However, 81F, 102D and 102F regions show disproportionately high incorporation of ^{3}H-UTP (Figure 1).

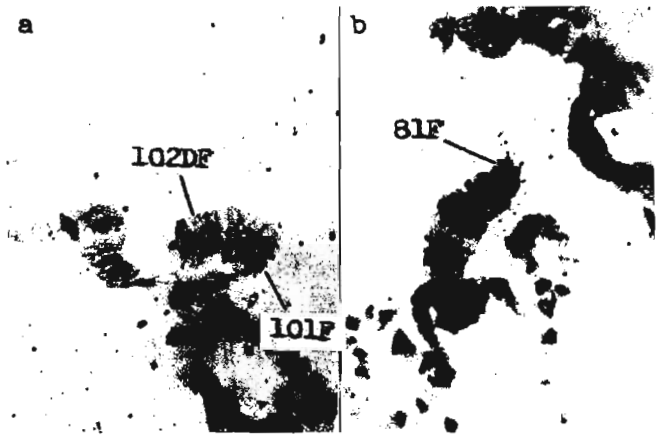


Figure 2. Labeling of the salivary gland chromosomes of Swedish larvae by ^{3}H-ATP in the presence of 100 microg. of actinomycin D.

Actinomycin D is strong inhibitor of RNA synthesis by E. coli RNA polymerase. Inhibition appears to occur for all regions of the chromosomes with the exception of 81F, 102D and 102F regions and sometimes 101F (Figure 2).

All these data show that in 81F, 102D, 102F regions A-T rich DNA is situated. It is interesting that the same regions show strong fluorescence after quinacrine staining (4-6). Results obtained are in good agreement with the view of Ellison,Barr (7) that quinacrine specifically stains A-T rich regions of chromosomes.

References: (1) Zhimulev, I.F. 1974, Chromosoma 46:59-76; (2) Leibovitch, B.A., R.B. Khesin 1974, Molec. Biol. (USSR) 8:467-474; (3) Leibovitch, B.A. et al., Ontogenez (USSR) 5:544-556, 1974; (4) Vosa, C.G. 1970, Chromosoma 31:446-451; (5) Ellison, J.R., H.J. Barr 1971, Chromosoma 34:424-436; (6) Iordansky, A.B. et al. 1971, Dokl. Acad. Nauk USSR 201:213-216; (7) Ellison, J.R. and H.J. Barr 1972, Chromosoma 36:375-390.

Hollingsworth, M.J. Medical College of St. Bartholomew's Hospital, London, U.K.

The temperature-dependence of the repair of sub-lethal radiation damage.

When Drosophila adults (F_1 hybrid melanogaster males) receive 60 krad of ionising radiations (15 MeV electrons from a linear accelerator) at a high dose rate (1 krad sec^{-1}) in two equal fractions with a 24 hour interval between the fractions, their post-irradiation survival time depends on the temperature at which they spend the interval between the 30 krad doses. If the temperature during the interval is 25°C they have 14 percent longer lifespans, on the average, than flies receiving the same dosage in a single dose or flies spending the 24 hour interval at 3°C, 6.5°C or 10°C.

	Mean post-irradiation lifespan (days)	Lifespan relative to controls	Repair factor*
3°C. First irradiation at age 4 days			
Control	26.8 ± 0.8	-	
30 (25°C) 30	18.5 ± 0.2	0.69 ± 0.03	1.18 ± 0.02
60 (25°C)	15.6 ± 0.2	0.58 ± 0.03	
30 (3°C) 30	17.0 ± 0.2	0.63 ± 0.03	1.01 ± 0.02
60 (3°C) -	16.8 ± 0.2	0.63 ± 0.03	
6.5°C. First irradiation at age 2 to 3 days			
Control	24.9 ± 0.4	-	
30 (25°C) 30	22.9 ± 0.2	0.92 ± 0.02	1.13 ± 0.02
60 (25°C) -	20.3 ± 0.2	0.81 ± 0.02	
- (25°C) 60	17.7 ± 0.3	0.71 ± 0.02	
Control	24.3 ± 0.5	-	
30 (6.5°C) 30	21.4 ± 0.3	0.88 ± 0.02	
60 (6.5°C) -	22.3 ± 0.3	0.92 ± 0.02	0.96 ± 0.02
- (6.5°C) 60	20.6 ± 0.2	0.85 ± 0.02	
10°C. First irradiation at age 1 to 2 days			
Control	27.1 ± 0.5	-	
30 (25°C)30	22.1 ± 0.3	0.8; ± 0.02	
60 (25°C)	19.9 ± 0.3	0.73 ± 0.02	1.11 ± 0.02
- (25°C) 60	20.2 ± 0.2	0.74 ± 0.02	
Control	26.8 ± 0.4	-	
30 (10°C) 30	22.6 ± 0.4	0.85 ± 0.02	
60 (10°C) -	21.7 ± 0.3	0.81 ± 0.02	1.04 ± 0.02
- (10°C) 60	20.4 ± 0.2	0.76 ± 0.02	

* A repair factor is defined as the quotient of the lifespan of the population given irradiation as two equal doses and the lifespan of the corresponding population given a single dose.

These observations indicate that the repair of sub-lethal damage in the post-mitotic tissues of adult Drosophila requires metabolically active processes. The effects of metabolic inhibitors and of hypoxia are being investigated.

Fowler, G.L.* and J. Uhlmann. University of Düsseldorf, W. Germany. Single-cyst in vitro spermatogenesis in D. hydei.

The dynamics of spermatogenesis in Drosophila (particularly D. melanogaster) has been extensively studied and well described. Whereas the bulk of the available information has come from cytological studies of the process at the level of the electron microscope (e.g., Tokuyasu et al., 1972a, b), recent observations (e.g., Peacock and Miklos, 1973) clearly show that specific questions regarding spermiogenesis in Drosophila can be approached using light microscope techniques, as well. The results of other experiments also indicate that this is the case, particularly when such observations are carried out on isolated testes of Drosophila differentiating under in vitro conditions (Fowler 1973). Even though such whole-testis culture has already shown itself to be a useful means of studying certain aspects of Drosophila spermatogenesis (Gould-Somero and Holland, 1974), an in vitro study of individual cysts of primary spermatocytes might be even better and should be considered for studies of this kind. The results of the experiments which lead to this conclusion follow.

Methods: Single cysts of primary spermatocytes were liberated from the wall of intact testes of D. hydei and placed into sterile culture medium (Mandaron, 1971) between 36 and 48 hours after the formation of the puparium. Desired cysts (Figure 1) were then aseptically pipetted to the culture chamber and observed and photographed at various times during their differentiation with an inverted phase microscope.

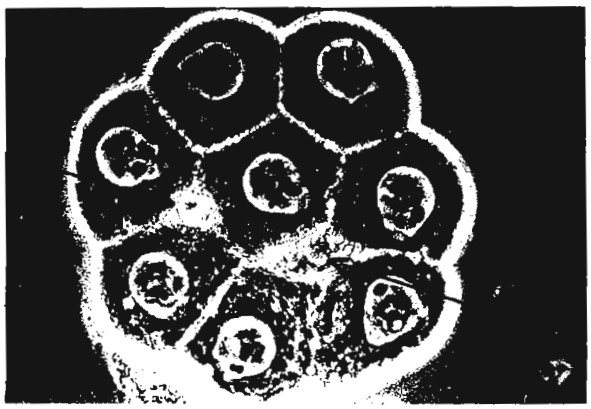


Figure 1. Cyst of 8 primary spermatocytes in the "lampbrush phase" (Hess and Meyer, 1968) of prophase I of meiosis (squash preparation X1000). The primary spermatocytes are surrounded by and individually contained within 2 cyst cells each of which possess a large nucleus (arrows).

Results: As can be seen in Figure 2, spermatogenesis of a single cyst of primary spermatocytes, beginning with a late stage of prophase I of meiosis (a) and ending with a stretched-out bundle of coiled spermatids (n; Figure 3), required approximately 30 hours under the present in vitro conditions. The actual time of differentiation is measured from the onset of metaphase I (b) since the specific stage of meiotic prophase of the explant is difficult to determine. Once the spindle apparatus has been formed and the characteristic degenerative changes in the lampbrush structures of the spermatocyte nuclei can be noted, both meiotic divisions (including a short interkinesis period, (c) are completed (d) within 6 hours. About the same amount of time is required for the formation (e) and subsequent differentiation (f,g) of the nebenkern. The formation of a "plasma cone" on one side of the differentiating cyst (at approximately 13 hours after the beginning of division of the spermatocyte nuclei) signals the beginning of cyst elongation and the distal growth of the nebenkern (h). Further elongation of both the cyst and the differentiating spermatids continues (i-m) during the next 15 hours until, at about 28 hours after the onset of division, the spermatids within the cyst begin to coil at the distal end (n; Figure 3).

Beyond this stage no differentiation of either the cyst or the spermatids is observed if, indeed, it is occurring.

In spite of the fact that further development of the cyst does not take place, the degree of its differentiation at 30 hours in vitro is roughly equivalent to that seen in cysts of intact testes which have developed in vivo for the same period of time. That is, the most advanced cysts seen in testes of normal pupae 30 hours after the formation of the puparium are similar (at least morphologically) to those which have differentiated in vitro.

Conclusions: The results of the series of experiments reported here indicate that not only can (1) the late spermatid stage be reached by isolated cysts in vitro within a period of time similar to that of cysts in vivo (i.e., about 30 hours), but also that under culture

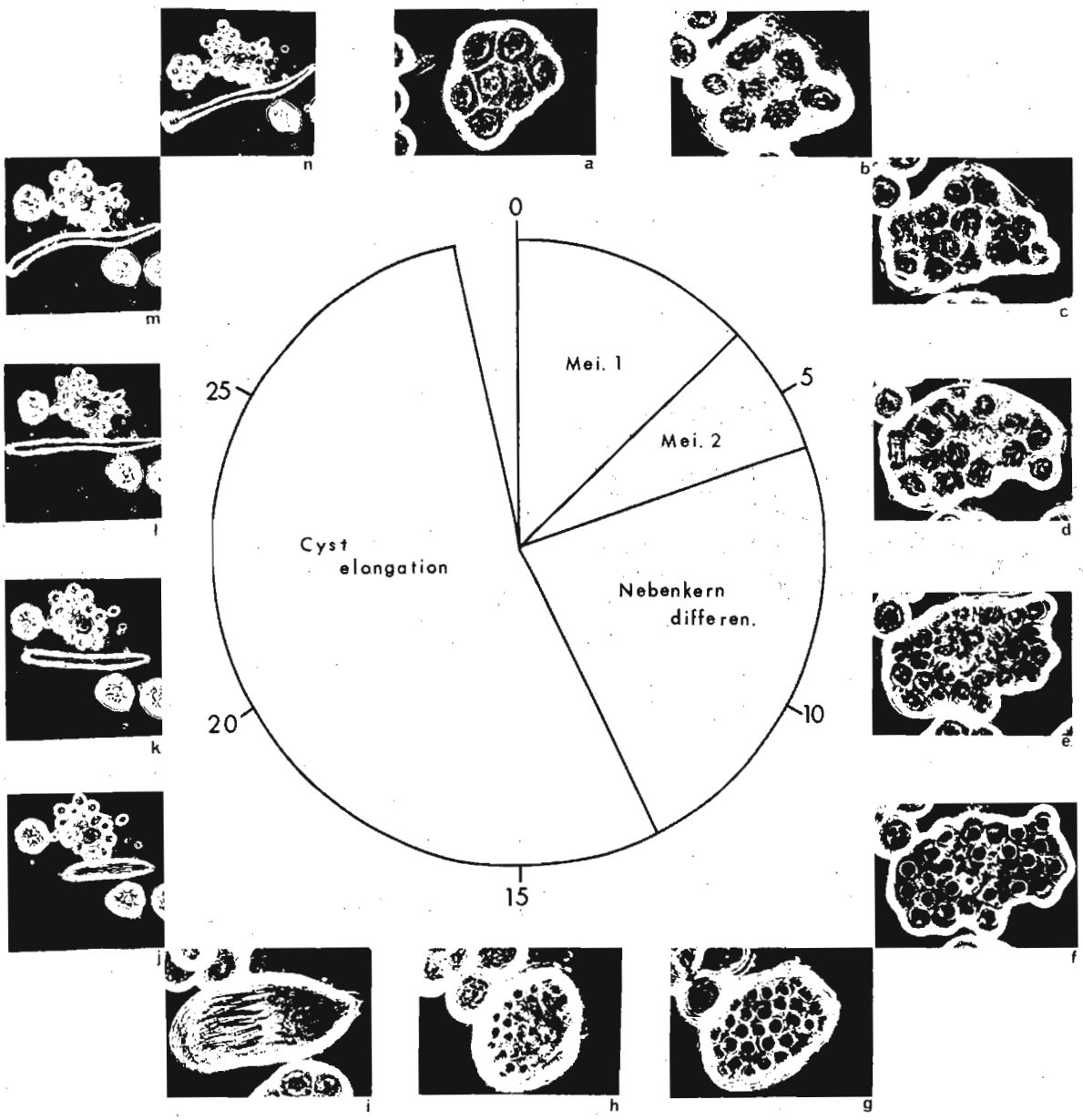


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Figure 2. In vitro differentiation (in hours) of a single cyst of seven primary spermatocytes in D. hydei. a, late lampbrush stage of prophase I of meiosis; b, metaphase I; c, interkinesis; d, anaphase II; e-g, differentiation of the nebenkern; h-n, elongation of the cyst; a-i, X500; j-n, X112.

conditions (2) it becomes possible to follow the dynamics of spermatogenesis with the light microscope with a precision that is not possible in the usual cytological preparation. Together, these findings represent an advance in the study of spermatogenesis in Drosophila and, more importantly, constitute a firm basis for further experimentation.

Acknowledgements: The authors are very much indebted to Miss Anita Graf for those countless hours in the darkroom, to the Alexander-von-Humboldt and the Richard Merton Foundations for their financial support of the project and to Professor O. Hess for his gracious hospitality and helpful suggestions during the three-year tenure of G.L.F. in his laboratory.

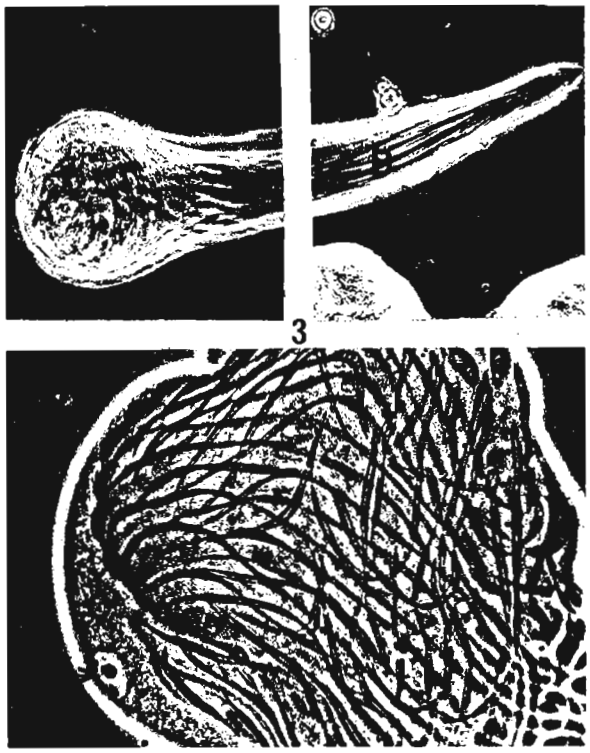

Figure 3. Detail of the cyst in Figure 2 at its most advanced stage of differentiation (Figure 2n). X1000. A. Coiling spermatids in the distal portion. B. Head region showing the elongating nebenkerns of the differentiating spermatids. C. Squash preparation showing the insertion of the spermatids into the head cyst cell with its prominent nucleus (arrow).

References: Fowler, G.L. 1973, Cell Diff. 2(1):33-42; Gould-Somero, M. and L. Holland 1974, Wilhelm Roux' Archiv 174:133-148; Hess, O. and G.F. Meyer 1968, Adv. Genet. 14:171-223; Mandaron, P. 1971, Dev. Biol. 25:581-605; Peacock, W.J. and G.L.G. Miklos 1973, DIS 50:41-44; Tokuyasu, K.T., W.J. Peacock and R.W. Hardy 1972a, Z. Zellforsch. Mikros. Anat. 124:479-506; Ibid, 1972b, Z. Zellforsch. Mikros. Anat. 127:492-525.

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Lefevre, G. Jr. and K.B. Wiendenheft.
California State University, Northridge.

Two genes in one band?

In an earlier experiment, lethal genes near vermilion (v) were sought. None was found to be included in the large band, 10A1-2, with which v is associated. Recently, a new recessive X-ray-induced lethal, not functionally allelic with v, has been recovered and appears to be in band 10A1-2. It interacts with all v deficiencies, including both those that delete only the left and those that delete only the right portion of 10A1-2. Previously, these "right" and "left" v deficiencies were shown to be noncomplementary, as though homozygous deficiency of the v locus was inviable. We now believe that the lethality is due to deletion of the lethal locus in both kinds of deficiencies.

Recombination tests show the new lethal, 1(1)L68, to be very close to v, on its right side. Less than 0.05% recombination between v and 1(1)L68 was detected in a large-scale experiment. The earlier closest righthand neighbor to v, 1(1)L12, located in band 10A4 or 5 showed 0.5% recombination with v. We conclude that the new lethal is literally a close neighbor of v in band 10A1. If it were in band 10A2, it should not interact with "left" v deficiencies, and it should show more recombination with v, less with 1(1)L12. It is possible of course that little recombination occurs within bands, but is stimulated at the margins of heavy bands. At the present time, however, we believe that this constitutes a good case for the existence of two distinct genetic functions within one specific band.

Chatterjee, S.N., A.K. Dutta Gupta and A.S. Mukherjee. University of Calcutta, India. Lack of differential transcriptional activity in the left and right arms of the X-chromosome of D. pseudoobscura.

Muller (1950) proposed that the long arm (XR of Stocker and Kastritsis 1972) of D. pseudoobscura due to its origin from an autosome (3R) of D. melanogaster, might not have achieved the optimum level of dosage compensation, while the short arm (XL of Stocker and Kastritsis 1972), being homologous to the X of D. melanogaster shows the accuracy in dosage compensation. The validity of this hypothesis has been tested at the transcriptional level to examine the possibility of differential hyperactivity of the two arms of the X-chromosome, by ^{3}H-Uridine (^{3}HUR, Sp. activity 3.27 Ci/mM, BARC, India, Conc. 200 μCi/ml, exposure time 12 days) autoradiography. The CH-1 strain of D. pseudoobscura was used for these experiments. Data are presented in Table 1. Results showed that, with reference to the variation in the grain numbers in the autosome III, the mean absolute grain numbers in neither arm are significantly different in the two sexes (tested by two sided 2 x 2 contingency test of Cochran). This is true for the proximal and distal segments of the XR, as well as the whole of XL and XR. The mean X/A grain ratios also showed some differences which are not statistically significant (t-test).

Table 1. Data on the ^{3}H-UR incorporation into XR and XL against that into a standard autosome (III Chromosome) of salivary gland cells of D. pseudoobscura.

	XR-proximal (18-29 segments) (a)	XR-distal (30-42 segments) (b)	XR-total (c)	XL (d)	3rd-total (e)
MEAN ABSOLUTE GRAIN NUMBER					
Female	156.8 (22)	140.6 (22)	297.5 (22)	128.3 (19)	191.6 (19)
Male	124.9 (14)	148.5 (14)	273.0 (14)	108.6 (14)	197.7 (14)
1. P-value	>0.10	>0.90	>0.30	>0.20	
MEAN X/A GRAIN RATIOS					
Female	0.82 ± 0.12	0.76 ± 0.14	1.60 ± 0.25	0.68 ± 0.19	
Male	0.73 ± 0.19	0.86 ± 0.13	1.57 ± 0.27	0.62 ± 0.11	
FEMALE/MALE RATIO					
	1.12	0.88	1.01	1.09	
2. P-value	>0.05	>0.05	>0.7	>0.2	

1. The difference was statistically tested by two sided 2 x 2 contingency chi-square test using the autosomal indices as standard.
2. Standard students t-test was applied.

These results lead us to conclude that both arms of the X-chromosome in D. pseudoobscura are hyperactive in the male and hence show dosage compensation. Whether there are relatively more noncompensating genes in the XR than in the XL of this species must need a more detailed investigation. The present results are, however, in agreement with the data reported by Abraham and Lucchesi (1973) with respect to the sexlinked genes G6-PD and 6 PGD in the XL and esterase-5 in the XR of D. pseudoobscura.

References: Abraham, I. and J.C. Lucchesi 1973, Proc. XIII Internat. Cong. Gen. Suppl. 74:52; Muller, H.J. 1950, The Harvey Lecture Ser. 43(1947-48):165-229; Stocker, A.J. and C.D. Kastritsis 1972, Chromosoma 37:139-176.

Gupta, J.P. Banaras Hindu University, Varanasi, India. A preliminary observation on the seasonal activity of abundant Drosophila species at Chandraprabha, Uttar Pradesh.

Studies on the population dynamics of Drosophila species with respect to the changes in the environment have been carried out for several years by many workers (Patterson, 1943; Dobzhansky and Pavan, 1950; Basden, 1954; Momma and Wakahama, 1955; Wakahama, 1956, 57, 62a,b, 1964; Momma, 1964, 65a,b). The collection records kept during the present survey conducted between July, 1965 and March, 1966, at Chandraprabha show some interesting features regarding the seasonal fluctuation of natural population of Drosophila.

However, evaluation of seasonal changes based on the frequency of individuals of different species in the natural population is difficult when not much is known about their ecology. Moreover, all the species are not similarly attracted to a single bait. The author has therefore taken into account the collection data of only those species which appeared in large numbers probably having the same degree of attraction towards the bait used by us.

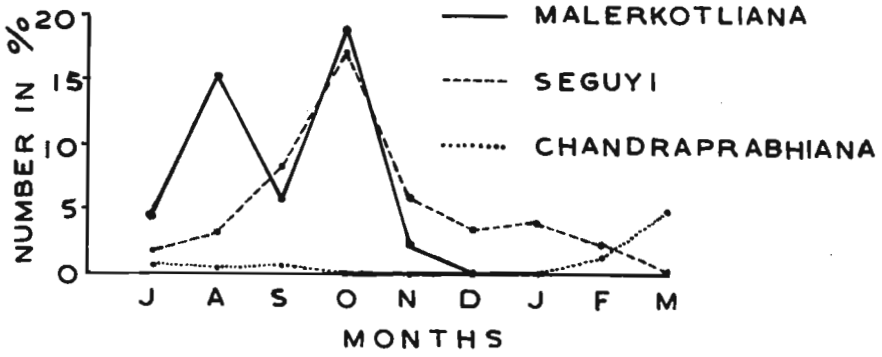


Figure 1. Graphic representation of the seasonal activity of abundant Drosophila species observed at Chandraprabha between July, 1965 and March 1966.

Collection site: Chakia forest, renowned for big game, is situated about thirty miles south-east of Varanasi, Uttar Pradesh. The forest extends over an area of about seven hundred square miles intervened here and there by plateau. The common trees in this region are Mahua (Bassia latifolia), Banyan (Ficus bengalensis) and several other unidentified wild species. Bushes of Chinese Ber (Zizypus rotundifolia) are largely seen in this region. An average annual rainfall is nearly between 20-30 inches. A place named Chandraprabha situated about fifteen miles interior within the forest and at an elevation of about 1000 feet was the site of the present study.

Method of collection: For this particular study, 15 traps were employed throughout the collecting trips. The collecting was done on five successive days in the last week of every month. Baits were always placed at the same spots so as to avoid any difference in the relative frequencies of Drosophila species due to food distribution. At the end of each collecting trip the baits were completely destroyed. The collected flies were classified and counted under stero-microscope.

Observations: A total of 1609 flies comprising three species, viz., D. malerkotliana Parshad & Paika, D. seguyi Smart and D. chandraprabhiana Gupta & Ray-Chaudhuri were collected during the entire period.

Drosophila malerkotliana: This was found to be the most abundant species in the locality (see Figure 1), showing a bimodal frequency curve. The first seasonal peak was observed in August and the second but much higher one in October. It appeared with a relatively high frequency in July and a sudden rise was seen in August followed by a decrease in September, again reached its peak in October, then showed an abrupt fall in November and disappeared during the subsequent months.

Drosophila seguyi: This species ranked as the second abundant species (see Figure 1). Its frequency gradually increased during July to September, then there was a sudden rise in October followed by a gradual decrease between November and February and became almost absent in March.

Drosophila chandraprabhiana: This species appeared in July and continued till September (see Figure 1) at a very low frequency. It was not seen at all during the period from October to January but again appeared in February, followed by the peak in March.

Table 1. Monthly collection record of abundant species at Chandraprabha

Name of species	Total no. of flies collected	Number of specimens in per cent								
		July	Aug.	Sept.	Oct.	Nov.	Dec.	Jan.	Feb.	March
malerkotliana	756	4.84	15.2	5.84	18.89	2.17	-	-	-	-
seguyi	737	1.68	3.04	8.2	17.3	5.7	3.4	3.9	2.4	0.19
chandraprabhiana	116	0.5	0.37	0.5	-	-	-	-	1.2	4.7
Totals	1609	7.02	18.61	14.54	36.19	7.87	3.4	3.9	3.6	4.89

Remarks: Observations based on the occurrence of these three Drosophila species have revealed that seguyi and malerkotliana seem to be more versatile and able to exploit a greater variety of ecological niches while chandraprabhiana was found to be more specialized and confined to fewer ecological niches. However, its appearance was observed twice to coincide with the flowering of lemon trees.

References: Basden, E.B. 1954, Trans. Roy. Soc. Edin. 72:603-654; Dobzhansky, Th. and C. Pavan 1950, J. Anim. Ecol. 19:1-14; Momma, E. 1964, Jap. J. Genet. 39:306-312; _____ 1965a, Jap. J. Genet. 40:275-295; _____ 1965b, Jap. J. Genet. 40:297-305; Momma, E. and K.I. Wakahama 1955, DIS 29:141; Patterson, J.T. 1943, Univ. Eex. Publ. 4313:7-216; Wakahama, K.I. 1956, Annot. Zool. Jap. 29:161-164; _____ 1957 Ibid 30:217-224; _____ 1962a, Ibid 35:234-242; _____ 1962b, J. Fac. Sci. Hokkaido Univ. 15:65-73; _____ 1964, Bull. Shimane Univ. 13:50-122.

Mather, W.B., P. Thongmeearkom, M. Clyde and D. Lambert. University of Queensland, Brisbane, Australia. D. sulfurigaster albostrigata from the Philippines and Western Malaysia.

In January, 1973, 15 iso-lines from Mt. Makiling, Luzon, Philippines, 3 from Penang, 32 from Kuala Lumpur and 22 from Kota Tinggi, Malaysia were established.

Twelve heterozygous inversions were detected (see Table) of which seven are new (see Figure next page). Photographs of other inversions have been published previously (Mather, W.B. and P. Thongmeearkom 1972, DIS 48:40 and 1974 DIS 50:60).

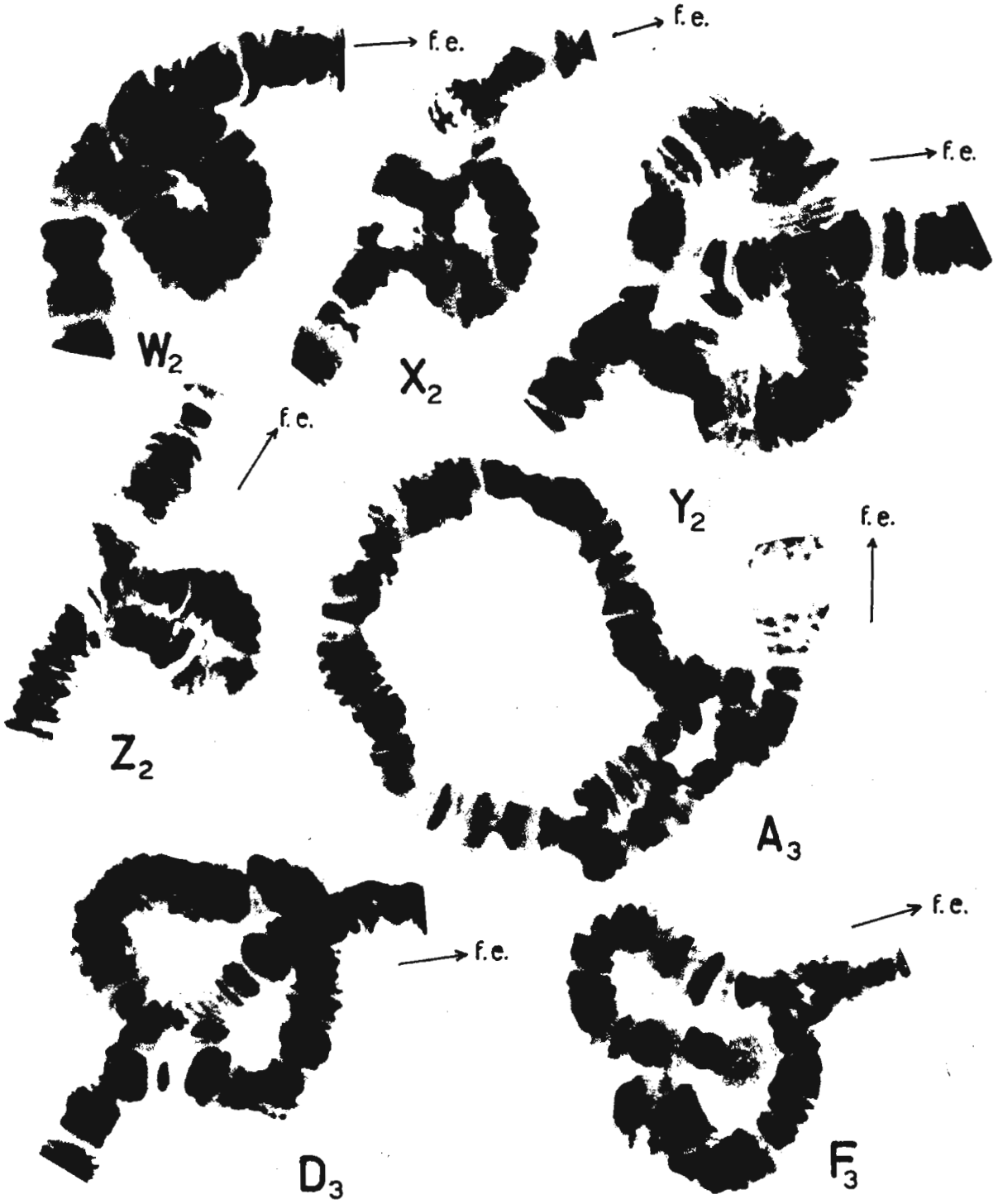
It should be noted that inversion G was not recorded from Malaysia and the inversions Y_2, A_3, D_3, F_3 have not been recorded from Mt. Makiling. The frequency of the heterozygous inversions at Kuala Lumpur are given.

The material was collected and the iso-lines established by W.B.M. The chromosomes were prepared and analysed by P.T., M.C. and D.L. and the inversions photographed by P.T.

Inversion	Type	Position	Locality	% Het. Freq. at K.L.
C	Sim.	III D	Mt. M., Pen., K.L., K.T.	27
E	Sim.	II L C	Mt. M., Pen., K.L., K.T.	78
G	Sim.	I P	Mt. M.	
Y	Sim.	III P	K.T.	
I_2	Sim.	II L C	K.L.	6
W_2	Sim.	III P	Mt. M., Pen., K.L., K.T.	42
X_2	Com.	III P	Mt. M., Pen., K.L., K.T.	63
Y_2	Sim.	II L C	K.L., K.T.	3
Z_2	Sim.	III C	Mt. M., K.T.	
A_3	Sim.	I P	Pen., K.L., K.T.	3
D_3	Sim.	II R C	K.L., K.T.	9
F_3	Sim.	III P	K.L.	39

Note: Sim.=Simple, Com.=Complex, D=Distal, C=Central, P=Proximal to centromere.
Mt. M.=Mount Makiling, Pen.=Penang, K.L.=Kuala Lumpur, K.T.=Kota Tinggi

(see Figure on next page)



Seven new inversions found in D. sulfurigaster albostrigata from the Philippines and Western Malaysia.

Alahiotis, S. University of Patras, Greece. Enzyme polymorphisms in two D. melanogaster populations of Southern Greece.

The present communication constitutes a preliminary report of a much wider investigation which had started a year ago and is still in progress.

Polymorphisms of three enzyme systems, namely, of α-glycerophosphate dehydrogenase (α-Gpdh), alcohol dehydrogenase (Adh) and malate dehydrogenase (Mdh-1) were studied in two free Drosophila melanogaster populations. The samples were collected from Gavros, a locality at a distance of about 8Km from the town of Patras (capital of Peloponnese) as well as from Cephalonia, an island in the Ionian Sea. The time of collection was June to September, for Gavros, and July to September for Cephalonia. The study was carried out by means of starch-gel electrophoresis; the methods followed were those used by Shaw and Prasad (1970) for α-Gpdh and Mdh-1, and O'Brien and MacIntyre's (1969) for Adh. Our results are gathered in Table I.

Table I

Locality	Flies scored	Frequencies			
		Adh			
		Adh^F		Adh^S	
Gavros	122	0.9877	$X^2_{1df=8.59}$ $.01<P<.001$	0.0123	$X^2_{1df=8.58}$ $.01<P<.001$
Cephalonia	209	0.9402		0.0598	
		α-Gpdh			
		$\alpha\text{-Gpdh}^F$		$\alpha\text{-Gpdh}^S$	
Gavros	138	0.6268	$X^2_{1df=5.57}$ $.01<P<.05$	0.3732	$X^2_{1df=5.57}$ $.01<P<.05$
Cephalonia	217	0.5369		0.4631	
		Mdh-1			
		$Mdh\text{-}1^S$		$Mdh\text{-}1^{rare*}$	
Gavros	137	0.9891		0.0109	
Cephalonia	216	0.9699		0.0301	

* (f) $Mdh\text{-}1^{rare}$ = (f) $Mdh\text{-}^F$+(f) $Mdh\text{-}1^{S1}$

In the Mdh-1 system a new allele was found in Greece. This allele (S_1) first discovered by a colleague in our Department (G. Yannopoulos and Christodoulou, C., in press) migrates towards the anode more slowly than the common allele S.

No significant differences were found in the Mdh-1 system; on the contrary, there were significant differences between the two populations in the frequencies of α-Gpdh and Adh alleles. In order to compare the gene frequencies, our data were subjected to a contingency chi-square analysis according to Workman and Niswander's method (1970).

Differences were also found, between the previously mentioned populations, in the rate of hybridization with Drosophila simulans; this rate was estimated equal to 8.68% in Gavros, while for Cephalonia it was 1.56%.

Cage population investigation on the selection effect of some environmental factors are in progress.

References: O'Brien, S.J. and R. MacIntyre 1969, Amer. Nat. V.103 930:113; Shaw, R. and R. Prasad 1970, Biochem. Gen. 4:297-320; Workman, D.L. and J.D. Niswander 1970, Amer. J. Human Gen. V. 22:24-49.

Wieschaus, E. Biozentrum der Universität Basel, Switzerland. X-ray induction of pattern abnormalities in early embryos.

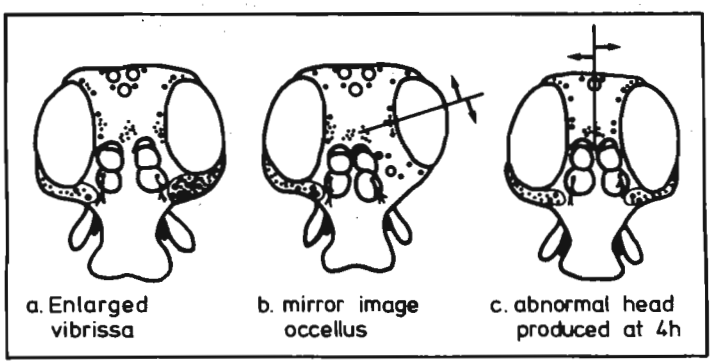
To obtain somatic recombination, we have X-irradiated (1000 r) Drosophila embryos at various ages during the first 10 hours after egg laying. We have also used these embryos to determine the earliest time at which mirror image regeneration abnormalities of the type described by Schubiger (1971), Bryant (1971) and Postlethwait (1973) could be induced.

Table I compares the various adult pattern abnormalities found with the embryonic ages at the time of irradiation. Fused forelegs, distal leg duplications and duplicated antennae

Table I: Frequency of abnormalities/age embryo irradiated.

Age Irradiated (hrs)	Leg Abnormalieies			Head Abnormalities					
	Total flies examined	Fused forelegs	Distal dupl.	Total flies examined	Dupl. occellus	Dupl. orbital	Dupl. palpus	Dupl. antenna	Enlarged vibrissae/ facia
10 ± 1	989	9	12	580	3	5	2	3	23
7 ± 1	476	5	4	262	2	-	1	-	3
3.15 ± 0.5	531	0	0	319	0	0	0	0	0
Control	650	0	1	341	0	0	0	0	0

have been described previously. On the head, mirror image duplications were found including the occellus, occiput, verticals, orbitals and palpus, in various combinations. The vibrissae and facia were the only structures not found in mirror image duplicates. Instead large tufts of vibrissae were found, with up to fives times the normal complement of bristles, arranged in no particular pattern. These abnormal vibrissae were usually (86%) associated with otherwise normal heads. On the other hand, the loss of vibrissae usually coincided with large mirror image duplications, the mirror image now apparently substituting for the missing vibrissae (see illustration Ia, Ib).



Most remarkable is the inability to induce imaginal pattern abnormalities by X-irradiation at the blastoderm stage. This could be due either to a relative insensitivity of the blastoderm cells to X-ray damages or to a greater regenerative potential of the cells during early embryonic stages when they may not yet be fully determined. However, since the total lethality after ten hours irradiation (25%) is less that that of irradiated blastoderms (63%), we doubt that greater cell damage at ten hours produces the adult defects. One might also argue that all the damaged blastoderms which would have produced the abnormal adults died, resulting in the higher blastoderm lethality. However, the connection between lethality and damage cannot be so simple, since among the survivors of a seven hour irradiation one finds adult abnormalities, in spite of the fact that seven hour embryos show a lethality roughly equivalent to blastoderm. It would be interesting to determine whether a different X-ray dose could produce such abnormalities in blastoderms or whether indeed the blastoderm cells actually have a greater regenerative potential.

Because very few embryos irradiated in stages immediately preceding and following the blastoderm stage survive irradiation, no data are presented on abnormalities induced at those times. Two of the four abnormal heads obtained from embryos irradiated at 4 ± 0.5 hr showed patterns which were not limited to a single side of the head. Instead both right and left sides were reduced equally along the midline, resulting in almost perfect left/right mirror images. This pattern was never obtained from older embryos.

It is likely that all these abnormalities were produced by X-ray induced death of an undetermined number of cells within the primordia. However, since clones induced at these stages often run through extensive regions of the head (from vibrissae to occellus, for example), killing even a single cell in an embryo might produce damage comparable to surgical techniques used on late larval disks (Schubiger, 1971; Bryant, 1971).

References: Bryant, P. 1971, Devel. Biol. 26:637-651; Postlethwait, J. et al. 1973, Devel. Biol. 32:344-360; Schubiger, G. 1971, Devel. Biol. 26:277-299.

Nagorski, L. and H. Nickla. Creighton University, Omaha, Nebraska. Gonadal dysgenesis in strains of Drosophila melanogaster carrying X-chromosome balancers.

The purpose of this report is to present frequencies of gonadal dysgenesis (Lucchesi et al., 1968) in D. melanogaster for several frequently used X-chromosome balancers. Gonadal dysgenesis is characterized by (1) variable penetrance, (2) morphologically normal external genitalia, ejaculatory duct and bulb, and accessory glands, and (3) absence or abnormal seminal vesicles and vasa deferentia. Males with gonadal dysgenesis have partially elongated or ellipsoidal testes. This condition may be bilateral or unilateral. When unilateral and the testis is ellipsoidal the right side is more often abnormal (Table 1).

Table 1. Distribution of abnormalities relating to gonadal dysgenesis in various male strains carrying the FM6 balancer chromosome.

% Left Ellipsoidal	% Right Ellipsoidal	% Unilateral Undetermined	% Both Ellipsoidal	Total Number Examined
2.48	17.39	2.17	13.97	322

Frequencies of gonadal dysgenesis in males containing various X-chromosome balancers are presented in Table 2. All cultures were reared at 22 ± 2 degrees C on standard medium: 3100 ml water, 32 g agar, 80 g yeast, 115 g sucrose, 600 ml cornmeal, 100 ml molasses, and 40 ml propionic acid. The relatively high frequency of bilateral gonadal dysgenesis reported here

Table 2. Frequencies of gonadal dysgenesis* in males containing various X-chromosome balancers.

Strain	% Unilateral	% Bilateral	Total Number Examined
FM1	0.75	3.26	398
FM4	10.95	6.69	493
FM6	17.20	20.20	500
FM7	0.60	0.20	500
In(1)dl-49,$v^{of}f$	0.00	0.00	500
In(1)y^4,y^4	0.25	0.25	400

* Classifications reported here refer only to CLASS III as described by Lucchesi et al., 1968.

may result from heterozygosity or homozygosity for the tuh-3 gene (described in Lindsley and Grell, 1968) which was present in the genetic background of all the strains tested. Gonadal dysgenesis appears to be restricted to FM4 and FM6 balancers, however, no explanation can be offered at this time for differences in penetrance observed in these two strains (Table 2).

Fertility of males having unilateral gonadal dysgenesis is markedly reduced when compared to males of identical genotype having normally differentiated testes. When Urbana females are mated to males having unilateral or bilateral gonadal dysgenesis they tend to lay fewer eggs than those females mated to genotypically identical males with normally differentiated testes (Nickla, unpublished observations).

References: Lucchesi, J.C., S.J. Counce and P.E. Hildreth 1968, J. Exp. Zoo. 168:437-450; Lindsley, D.L. and E.H. Grell 1968, Carn. Inst. Wash. Pub. No. 627.

Majumdar, S.K. and D.S. Novy. Lafayette College, Easton, Pennsylvania. The effects of 2,4,5-trichlorophenoxyacetic acid on D. melanogaster. II. Embryonic and postembryonic mortality.

Previous studies on D. melanogaster with 2,4,5-t provided information regarding differential genotypic response and mutagenic effects of this herbicide (Novy and Majumdar, 1972; Majumdar and Golia, 1974). In other studies it has been reported that 2,4,5-t produced abnormal and dead fetuses in mice and rats (Courtney et al., 1970; Courtney and Moore, 1971), induced ovary damage and female sterility in D. melanogaster (Davring and Sunner, 1971), depressed growth rate of nitrogen fixing bacteria (Foldesy et al., 1972) and caused chromosome damage in the Mongolian gerbil (Majumdar and Hall, 1973). The

present work is therefore an extension of research to study the effects of 2,4,5-t on D. melanogaster eggs, larvae and prepupae.

In order to collect a large number of eggs over a short period of time a simple egg collection chamber was developed (Majumdar and Novy, 1972). Eggs, larvae and pupae of Oregon-R flies were used in this study. The 2,4,5-t which contained no detectable amount of dioxin (Collins and Williams, 1971) was purchased from Eastman Kodak Company, Rochester, N.Y. To test the hatchability of eggs the following seven age groups were selected: 1/2 hour, 1 hour, 1-1/2 hours, 2 hours, 3 hours, 6 hours and 10 hours. The eggs were treated with water (control), 50% ethyl alcohol (control) and a sublethal dosage of 2,4,5-t (125 ppm). The herbicide was dissolved in 50% alcohol, and the culture vials were kept at $26^{\circ} \pm 1^{\circ}C$.

The eggs were treated for one minute in a drop of the 125 ppm 2,4,5-t mixture. A paper blotter removed excess moisture after one minute, and the eggs were immediately placed in groups of fifty into vials containing Carolina instant Drosophila medium. A total of 300 eggs for each age group was used. For each of these seven ages, three hundred eggs were also subjected to one minute in one drop of 50% ethanol, the solvent employed for the 2,4,5-t. With the sterile needle, these eggs were placed in groups of fifty into vials containing normal instant food. Similarly other sets of 300 eggs were treated with water and transferred in groups directly into normal food vials. Within the ordinary food, under optimum environmental conditions, eggs hatched into adult flies within 10-12 days. The number of adult flies present were etherized and counted, and the percentage was recorded.

It was desirable to elicit information regarding larval tolerance and metamorphosis within the poisoned media as well. The Larval Test Method suggested by Davring and Sunner (1971) was employed for this purpose. With a sterile needle, one hundred first instar larvae were transferred in two groups to one of four types of instant food media: one containing perfectly normal food, one with 0.075 cc of 50% ethanol, one with a concentration of 200 ppm of 2,4,5-t and the last with a dose of 400 ppm of the herbicide. Similar treatment method was used to test the second and third instars and prepupae stage.

The percentage of control eggs maturing into adult flies was somewhat low, ranging from about 70% for the 10 hr old eggs treated with 50% alcohol to about 82% for 1-1/2 hr pure controls (Table 1). This reduced productivity was probably due to damage and destruction occur-

Table 1. Effects of 2,4,5-t on D. melanogaster eggs. The table gives the percentage of eggs hatched to develop adult flies out of a total of 300 for each of seven ages of eggs directly treated with three different solutions

Egg age	Control (water)	Control (50% ethanol)	125 ppm 2,4,5-t
1/2 hour	80.3	82.7	60.7
1 hour	79.7	72.3	63.5
1-1/2 hours	82.4	76.6	62.0
2 hours	81.7	80.7	58.4
3 hours	80.6	71.1	63.7
6 hours	80.5	72.0	65.4
10 hours	75.7	70.0	65.7

ring in the transferral of eggs from the egg chamber to vials. There did not seem to be any difference in control hatchabilities with respect to age. However, in both cases of water and of alcohol control treatments, the 10 hr eggs exhibited the lowest producing percentages in their respective groups. By contrast, a noticeable difference was apparent in the development of eggs treated with the sublethal dose of 125 ppm of 2,4,5-t. Flies production from poisoned eggs ranged from a low of 58.4% for 2 hr old eggs to a high of 65.7% for 10 hr eggs.

Toxic effects of 2,4.5-t on larval stages and prepupae can be seen from Table 2. Once again, control groups with and without alcohol appeared to be not significantly dissimilar in their response. However, as a general trend, the older the larval stage, that is, the closer to the adult form, the less likely transferral and treatment were to affect their maturation process. At the first instar larval stage, the organisms reacted in much the same way as eggs

Table 2. Effects of 2,4.5-t on D. melanogaster larvae and prepupae. The table gives the percentage of larvae and prepupae developed to adult flies out of a total of 100 for each of four ages, after transferral to food media mixed with four different solutions.

Larval stage	Control (water)	Control (50% ethanol)	200 ppm 2,4,5-t	400 ppm 2,4,5-t
1st Instar (24 hours)	81.0	74.0	20.0	10.0
2nd Instar (48 hours)	90.0	86.5	27.0	6.0
3rd Instar (72 hours)	97.5	96.0	18.0	5.0
Prepupae (5-1/2 days)	100.0	94.0	93.0	53.0

had, although in alcohol, somewhat less successfully. The percentage viabilities for control groups, however, successively rose for second and third instar larvae, and by the prepupal stage all prepupae became adult flies. There thus appeared to be a very definite increase in development corresponding to a more advanced stage of fly metamorphosis. A dramatic comparison was noted with the two groups treated with 2,4,5-t. For those larvae which matured in vials of food containing 200 ppm of the chemical, percentage viability was down to 18-27% for larval groups, but the prepupal flies evidently suffered least, if at all, by maturing in this environment, their percentage remaining 93%. By comparison, doubling the concentration of the herbicide to 400 ppm clearly increased mortality among all four groups. First instar larvae developed at one-half the capacity of the other dose. Second, instar larvae fared even worse, their development-capacity being sliced from 27% to 6%. Third, instar organisms experienced a similar curtailment, decreasing from 18% to 5%. Pupae again developed with far greater success than any of the earlier stages.

The 2,4,5-t effects on eggs and larvae as shown in Tables 1 and 2 were examined separately, first by analysis of variance and then by Tukey's test (Snedecor, 1956). This study revealed in both cases, that there were statistically significant differences among the treatments' means, that is, among water, ethanol and 125 ppm of 2,4,5-t for eggs, and among water, ethanol, 200 and 400 ppm of 2,4,5-t for larvae.

When Tukey's Test was applied to 2,4,5-t effects on eggs, it was observed that both water and alcohol differed significantly from 2,4,5-t poisoning at the 0.01 confidence level. However, as anticipated, water was not determined to be different by a good statistical margin from alcohol, thus demonstrating that virtually all harmful effects were due to the herbicide alone, as ethanol was the only solvent additive to the chemical.

Tukey's Test was also evaluated for larvae treatment doses, and here again, water was determined to be not significantly different from alcohol. All other comparisons of control-concentration means were significantly different such as water or ethanol versus 200 ppm (except the pupal group) and the same control groups versus 400 ppm.

Ages of the eggs and larvae formed another basis for comparison. Tukey's Test on Oregon-R eggs collected at different periods up to ten hours, showed that none of the ages were different by a statistically significant margin from any of the others. Thus, egg age did not appear to be differentially affected by the same concentration of 2,4,5-t. In the case of larvae, it was observed that the means for first, second, and third instar flies were statistically homogeneous. However, compared with 5-1/2 day prepupae, the 24, 48 and 72-hour larval stages were all significantly different as calculated using Tukey's Test.

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Wright, C.P. Western Carolina University, Cullowhee, North Carolina. Development of Gluful-2, 1(1)EN3, a lethal mutant of Drosophila.

Gluful-2, 1(1)EN3, is a sex-linked, lethal mutant of Drosophila melanogaster which was X-ray induced by Novitski (1963). Death in this mutant occurs in the late pupal stage. Both weight and oxygen consumption measurements were made on individual male larvae and pupae from the first-instar larval stage until the time when oxygen uptake ceased. Oxygen consumption measurements were made with small respirometers in a 25°C water bath. A 20% NaOH solution was used to remove CO_2 from the respirometers, which caused movement of the NaOH drop. By measuring the volume of the droplet displacement, the oxygen consumption of a larva or pupa was determined. Control larvae and pupae were y, w, spl, sn males from the stock of Novitski (1963) in which the lethal mutant was induced. Experimental data were tested statistically against control data by use of the Mann-Whitney U nonparametric test (Tate and Clelland 1957).

Both fresh and dry weights of 1(1)EN3 larvae (Table 1) were less than in controls (Table 2). The rate of oxygen consumption per larva was significantly less in 1(1)EN3 (Table 1) than in controls (Table 2). When calculated per unit dry weight, 1(1)EN3 larvae (Table 1) used

Table 1. Average weights and rates of oxygen consumption for 1(1)EN3 larvae.

Age in hr. after oviposition	Fresh weight/ larva in mg.		Dry weight/ larva in mg.		O_2 consumption in cu.mm./larva/hr.		O_2 consumption in cu.mm./mg.dry wt./hr.	
	n	M. ± S.E.	n	M. ± S.E.	n	M. ± S.E.	n	M. ± S.E.
30 hr.		did not weigh	4	0.0028* ± 0.0002	8	0.105** ± 0.002	8	37.634 ± 0.870
48 hr.		did not weigh	6	0.009** ± 0.001	8	0.244** ± 0.020	8	27.125* ± 2.205
72 Hr.	8	0.26 ± 0.04	8	0.05** ± 0.01	8	1.028** ± 0.096	8	22.587* ± 0.876
96 hr.	8	0.93** ± 0.10	8	0.19** ± 0.02	8	3.344** ± 0.448	8	18.101 ± 1.077

* Significant at .05 level
** Significant at .01 level

more oxygen than control larvae (Table 2). This probably relates to the fact that 1(1)EN3 larvae were smaller than control larvae. Gross examination revealed no visible morphological abnormalities in 1(1)EN3 larvae. Control larvae formed puparia at about 110-116 hours after oviposition. Puparium formation in 1(1)EN3 occurred at about 120-144 hours after oviposition. About 97% of 1(1)EN3 larvae formed puparia.

Table 2. Average weights and rates of oxygen consumption for y, w, spl, sn control larvae.

Age in hr. after oviposition	Fresh weight/ larva in mg.		Dry weight/ larva in mg.		O_2 consumption in cu.mm./larva/hr.		O_2 consumption in cu.mm./mg.dry wt./hr.	
	n	M. ± S.E.	n	M. ± S.E.	n	M. ± S.E.	n	M. ± S.E.
30 hr.		did not weigh	4	0.0039 ± 0.0001	8	0.137 ± 0.004	8	35.192 ± 1.021
48 hr.		did not weigh	4	0.018 ± 0.001	10	0.390 ± 0.015	10	21.689 ± 0.851
72 hr.	10	0.29 ± 0.02	10	0.07 ± 0.01	10	1.456 ± 0.059	10	19.698 ± 0.236
96 hr.	9	1.36 ± 0.05	9	0.31 ± 0.01	9	5.108 ± 0.282	9	16.382 ± 0.441

During pupal development both fresh and dry weights of 1(1)EN3 (Table 3) were significantly less than in controls (Table 4). Measurements for controls stop at 80 hours after puparium formation because soon after this age they emerged as adults. The 1(1)EN3 individuals remained in the pupal stage until death apparently occurred at about 200 hours. Rates of oxygen consumption in 1(1)EN3 pupae followed the same general pattern as in control pupae until 104 hours, except at a significantly lower level at each point measured. Thus, until 104 hours after puparium formation, oxygen consumption in 1(1)EN3 pupae followed, to some extent, the U-shaped curve of control pupae. Prior to the upswing in the U-shaped curve, the rate of oxygen consumption in 1(1)EN3 pupae reached its minimum at 40 hours, as was the case for control pupae. But, in the ascending portion of the curve, the maximum rate of oxygen consumption in 1(1)EN3 pupae was reached at 104 hours, which was a day later than the maximum in control pupae. After this maximum rate, oxygen consumption in 1(1)EN3 pupae gradually decreased, until at 200 hours none could be detected.

Table 3. Average weights and rates of oxygen consumption for 1(1)EN3 pupae.

Age in hr. after pupium formation	Fresh weight/pupa in mg. n	M. ± S.E.	Dry weight/pupa in mg. n	M. ± S.E.	O_2 consumption in cu.mm./pupa/hr. n	M. ± S.E.	O_2 consumption in cu.mm/mg dry wt/hr. n	M. ± S.E.
3 hr.	10	0.91** ± 0.03	10	0.27** ± 0.01	10	1.919** ± 0.071	10	7.139** ± 0.185
10 hr.	10	0.83** ± 0.06	10	0.29* ± 0.03	10	1.402** ± 0.120	10	5.009* ± 0.358
20 hr.	10	0.80** ± 0.03	10	0.24** ± 0.01	10	0.642** ± 0.028	10	2.705* ± 0.123
40 hr.	7	0.90** ± 0.05	7	0.30 ± 0.02	7	0.525** ± 0.071	7	1.762* ± 0.201
60 hr.	10	0.79** ± 0.03	10	0.25** ± 0.02	10	0.555** ± 0.055	10	2.242** ± 0.183
80 hr.	8	0.72** ± 0.06	8	0.24** ± 0.01	8	0.645** ± 0.090	8	2.710** ± 0.341
104 hr.	7	0.74 ± 0.04	7	0.26 ± o.o1	7	0.936 ± 0.190	7	3.549 ± 0.731
128 hr.	8	0.62 ± 0.06	8	0.26 ± 0.02	8	0.742 ± 0.172	8	3.001 ± 0.682
152 hr.	9	0.44 ± 0.05	9	0.26 ± 0.02	9	0.392 ± 0.090	9	1.557 ± 0.404
176 hr.	12	0.33 ± 0.04	12	0.21 ± 0.02	12	0.090 ± 0.040	12	0.493 ± 0.227
200 hr.	7	0.32 ± 0.03	7	0.24 ± 0.02	7	0.000 ± 0.000	7	0.000 ± 0.000

* Significant at .05 level
** Significant at .01 level

Developing morphological characteristics observed in 1(1)EN3 pupae closely paralleled the rates of oxygen consumption. The peak of morphological development was reached at 104 hours when the pupal cases contained what looked like fully developed adults ready to emerge. After 104 hours the 1(1)EN3 pupae began to dry out as was indicated by shrivelling of the organisms. None of the apparently fully developed adults ever emerged. By 200 hours all had apparently died as pupae.

Table 4. Average weights and rates of oxygen consumption for y, w, spl, sn control pupae.

Age in hr. after pupium formation	Fresh weight/pupa in mg. n	M. ± S.E.	Dry weight/pupa in mg. n	M. ± S.E.	O_2 consumption cu.mm./pupa/hr. n	M. ± S.E.	O_2 consumption in cu.mm/mg.dry wt/hr n	M. ± S.E.
3 hr.	10	1.38 ± 0.02	10	0.38 ± o.o1	10	3.092 ± 0.076	10	8.244 ± 0.187
10 hr.	10	1.19 ± 0.03	10	0.36 ± 0.01	10	2.213 ± 0.066	10	6.261 ± 0.211
20 hr.	10	1.16 ± 0.02	10	0.34 ± 0.01	10	1.114 ± 0.037	10	3.340 ± 0.162
40 hr.	10	1.17 ± 0.04	10	0.35 ± 0.01	10	0.842 ± 0.083	10	2.433 ± 0.097
60 hr.	10	1.16 ± 0.02	10	0.35 ± 0.01	10	1.266 ± 0.049	10	3.620 ± 0.098
80 hr.	10	1.14 ± 0.02	10	0.34 ± 0.02	10	1.864 ± 0.070	10	5.542 ± 0.194

References: Novitski, E. 1963, DIS 37:51-53; Tate, M.W. and R.C. Clelland 1957, Nonparametric and Shortcut Statistics in the Social, Biological, and Medical Sciences, Interstate Danville, Illinois.

Lalor, J.H., J.P. Chinnici and G.C. Llewellyn, Virginia Commonwealth University, Richmond, Virginia. The preliminary study of the effects of aflatoxin B_1 on Larvae of Drosophila melanogaster.

The aflatoxins, first extensively studied in the 1960's, are a group of highly toxic metabolites produced by the mold Aspergillus flavus, a common agricultural contaminant. Research has shown these compounds, particularly aflatoxin B_1 to be extremely carcinogenic in mammals such as the rat (Butler, 1964). However, relatively little work concerning the effect of aflatoxin on the insects has been done. The purpose of the present investigation was to study the effects of aflatoxin B_1 on a laboratory bred strain of Drosophila melanogaster in relatively low concentrations in order to obtain an LD_{50} value.

This information could, in all probability, prove useful for future genetic studies of aflatoxin effects on D. melanogaster.

The parental stock flies (wild type Oregon-R) were kept on a standard control medium having dextrose, agar and Brewer's yeast as its major constituents and Tegosept-M as a mold inhibitor. Pure grade B crystalline aflatoxin B_1 (Calbiochem) was dissolved in acetone, incorporated into a similar sample of this control medium at a stock concentration of 20 μg/ml and diluted to the test concentrations of 2,4 and 8 μg/ml. Adult flies were allowed to deposit eggs on plates of control medium for periods of six hours and 1st instar larvae were collected 24 hours later using forceps. The collected larvae first were placed on small chips of blotting paper which were later positioned on the test media within 8 dram glass vials. Observations were made to insure the safe transfer of all larvae. Vials were then incubated at 25 ± 1°C and the emerging adults were scored for the variety of traits listed in Table 1.

Table 1. Effect of aflatoxin B_1 on viability and morphology in D. melanogaster

Morphological responses	Aflatoxin B_1 concentration (μg/ml) 0	2	4	8
Percent entering pupal stage*	100	100	45	10
Mean size of pupal case (mm)	2.91 ±0.11	2.67 ± 0.11	2.31 ± 0.11	2.15 ± 0.18
Percent emerged from pupal stage*	97	35	0	0
Mean time till emergence (days)	♀ 9.78 ± 1.16 ♂ 9.77 ± 0.96	12.16 ± 1.23 15	-	-
Mean body length (mm)	♀ 2.88 ± 0.11 ♂ 2.52 ± 0.11	2.40 ± 0.22 2.20	-	-
Mean wing length (mm)	♀ 2.39 ± 0.08 ♂ 2.04 ± 0.07	2.03 ± 0.16 2.0	-	-

* Data from vials at each concentration was grouped together, thus eliminating standard error calculations for percents entering into and emerging from the pupal stage from the data.

Several simultaneous trends were noted. Decreases in percent entering the pupal stage, pupal case size, percent emergence, mean body length and mean wing length were found between the control and all aflatoxin exposed animals (Table 1) except percent entering the pupal stage at 2 μg/ml. These data indicate that concentrations of aflatoxin B_1 above 2 μg/ml are extremely detrimental to the larvae whereas 2 μg/ml itself is a higher than LD_{50} concentration for these fruit flies. Morphological results similar to these have been previously obtained by Beard and Walton (1971) using the house fly Musca domestica. They reported decreases in rate of development and in the sizes of both the pupal case and the adult fly. Reports that higher levels of aflatoxin are inhibitory to D. melanogaster was shown by Matsumura and Knight (1967). Using a concentration equivalent to 50 μg/ml, they allowed newly emerged flies to mate and deposit eggs while feeding on this toxin. They noted a large increase in adult mortality and total inviability of all eggs produced. Their work, the above data and that of Moore (1974) with Anthonomus grandis Boheman, the boll weevil, suggest the possible use of aflatoxins as chemosterilizing agents. However, before they may be practically applied, more work should be done on the possibility of their being biotransferred and/or bioconcentrated (Nevins and Grant, 1971).

References: Beard, R.L. and G.S. Walton 1971, Bull. Connecticut Agricultural Experiment Station 725:3-26; Butler, W.H. 1964, Brit. J. Cancer XVIII (4):756-762; Matsumura, F. and S.G. Knight 1967, J. Econ. Ento. 60(3):871-872; Moore, J.H. 1974, ASB Bull. 21(2):70; Nevins, M.P. and D.W. Grant 1971, Bull. Environ. Contam. Toxicol. 6(6):552-558.

Zhimulev, I.F. and V.E. Grafodatskaya. Institute of Cytology and Genetics, Novosibirsk, USSR. A simple method of induction of anaerobiosis puffs in D.m.

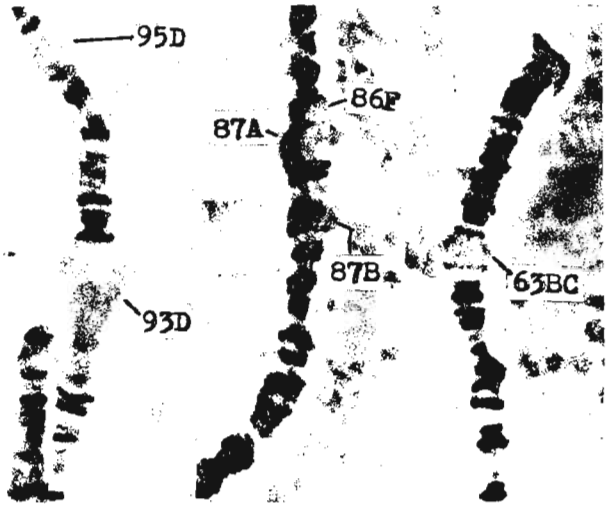


Figure 1. Anaerobiosis puffs after 1 hour anoxia.

Larvae of wild strain Batumi-L, hatched from eggs during 21-24 hrs from oviposition were collected. Puffs were induced three days late (93-96 hrs from oviposition). Larvae were placed in Ephrussi-Beadle solution for 1 hour at 25°C. The solution layer (3cm) was such that larval spiracles had no access to air. Preparations of three groups of larvae were made: a) without anoxia treatment, b) immediately after 1 hr of anoxia, c) after 20 min. recovery in air following 1 hr of anoxia. Squashed preparations of the salivary gland chromosomes were made and puffs sizes were scored visually on an arbitrary scale from 1 to 6 classes as previously described (Belyaeva et al., Tsitologia 16:440-446, 1974). 20 min. after recovery in air large puffs may be seen in regions that are known to form anaerobiosis puffs (Ashburner, Chromosoma 31:356-376, 1970) (Figure 1, Table 1).

From 83 larvae treated with 1 hour anoxia 77 flies have been hatched six days late. No phenotypic abnormalities were found.

Table 1. Induction of puff size changes.

	Puff sizes		
Puffs	96 hrs. larvae	1 hour of anoxia	20 min. after recovery in air
63BC	2.1 ± 0.06*	1.9 ± 0.04	4.0 ± 0.02
64E	2.1 ± 0.01	2.0 ± 0.03	2.7 ± 0.03
67AB	2.4 ± 0.03	2.7 ± 0.04	3.6 ± 0.03
86F	2.0 ± 0.03	2.0 ± 0.02	3.3 ± 0.05
87A	2.0 ± 0.03	2.3 ± 0.02	4.7 ± 0.06
87B	2.0 ± 0.02	2.2 ± 0.02	4.6 ± 0.04
93D	2.7 ± 0.03	2.9 ± 0.05	5.2 ± 0.04
95D	2.0 ± 0.02	1.9 ± 0.03	2.8 ± 0.05

* Standard error

Mollet, P. and R. Büchi. Swiss Federal Institute of Technology, Zürich, and Swiss Federal Research Station, Wädenswil, Switzerland. Comparison of feeding and injection technique in mutagenicity testing of Trenimon in Drosophila.

For mutagenicity testing of chemicals with Drosophila adults two application techniques are generally used: abdominal injection and feeding. In our experiments we compared the mutagenic effect of Trenimon applied with the two different techniques. One group of males was injected with 0.2 µl of saline or saline plus 10^{-5} M Trenimon, using a microapplicator set (Mollet and Würgler, 1974). Another group of males was fed during 17 hours with a 5% aqueous sugar solution or sugar solution containing 10^{-5} M Trenimon, using the feeding method of Lüers

(1953). In both series the general procedure was the same. Males, 2 to 3 days old, of the constitution R(1)2,y B/B^S Y y^+ were treated. Eighteen hours later they were mass mated in bottles with 4-day old virgin Inscy;dp bw;st p^p females for 7 hours. Then the males were discarded and groups of 4 females were put into separate vials for four one-day periods. In this way we obtained broods 1, 2, 3 and 4. The progeny collected during the mating period were included in brood 1. The progeny were counted and classified according to the phenotypes indicated in the Table. Non-Bar males represent sex chromosome loss. The frequency of Bar males

Mutagenicity testing with Trenimon applied by injection or feeding. Treated R(1)2,y B/B^S Y y^+ males were mated to Inscy;dp bw;st p^p females. Scored progeny: F, females; M, Bar males; L, non-Bar males; G, mosaics; p*, significance of difference between Trenimon data and corresponding control.

Treatment	Chemical and conc.	Brood	Total progeny F + M + L + G	% M	p*	% L	p*
Injection	saline 0.4%	1	52 + 83 + 4 + 0	59.7		2.88	
		2 - 4	116 + 176 + 2 + 0	59.9		0.68	
		1 - 4	168 + 259 + 6 + 0	59.8		1.39	
	Trenimon 10^{-5} M	1	15 + 60 + 8 + 2	70.6	0.096	9.41	0.033
		2 - 4	42 + 140 + 9 + 0	73.3	0.003	4.71	0.004
		1 - 4	57 + 200 + 17 + 2	72.5	<0.001	6.16	<0.001
Feeding	sugar solution 5%	1	153 + 230 + 4 + 1	59.3		1.03	
		2 - 4	335 + 429 + 3 + 0	55.9		0.39	
		1 - 4	488 + 659 + 7 + 1	57.1		0.61	
	Trenimon 10^{-5} M	1	148 + 538 + 40 + 2	73.9	<0.001	5.49	<0.001
		2 - 4	640 + 1983 + 91 + 4	73.0	≪0.001	3.35	<0.001
		1 - 4	788 + 2521 + 131 + 6	73.2	≪0.001	3.80	≪0.001

is used as a measure of the preimaginal death of the female progeny. Thus a mutagenic effect is expressed in an increase, above the spontaneous rate, in the frequencies of both the non-Bar males (sex chromosome loss) and the Bar males (sex ratio shift). Statistically the data of brood 1 are different from the pooled data of broods 2, 3 and 4 and are therefore given separately. The data of broods 2 to 4 are homogeneous (chi-square test).

Trenimon whether fed or injected does increase the frequency of non-Bar males and that of Bar males above the control level. This is true for the data of brood 1, the pooled data of broods 2 to 4 and the overall pooled data (broods 1 to 4). With one exception (brood 1, injection) all the differences between control and Trenimon treatment are statistically significant for both application methods used (see Table). Thus the mutagenicity of Trenimon is detectable with both techniques.

For a comparison of the two application methods with respect to the mutagenic effect of Trenimon we use the overall pooled data. The frequency of non-Bar males after correction with the corresponding control frequency is higher (4.8%) with injection than with feeding (3.2%). In contrast the corrected frequency of Bar males is 31.5% with injection and 37.5% with feeding. However these differences are not significant: for the sex chromosome loss chi square is 2.1 and p is 0.15; for the sex ratio shift chi square is 1.6 and p is 0.20. Thus our data indicate that with a highly active alkylating agent injection and feeding result in similar mutation frequencies.

References: Lüers, H. 1953, Arch. Geschwulstforsch. 6:77-83; Mollet, P. and F.E. Würgler 1974, DIS 50:202.

Acknowledgements: For helpful discussions we thank Prof. F.E. Würgler and Drs. U. Graf and D. Turner. The work was supported by grant No. 3.7040.72 from the Swiss National Foundation for Scientific Research.

Korochkin, L., A. Onischenko and N. Matveeva. Laboratory of Developmental Genetics, Institute of Cytology and Genetics, Novosibirsk, USSR. Esterase activity during the development of the salivary glands in Drosophila virilis.

Using histochemical and microchemical methods, we investigated the changes of the activity of esterases of salivary glands in inbred 9-8 L of D. virilis from a Batumi population. α-naphthyl propionate, α-naphthyl acetate, β-naphthyl acetate and indoxyl acetate were used as substrates. The system of α- and β-esterases was described by us before (Korochkin et al., 1973). In this case it was observed that the esterase activity is very low immediately after the 2nd moulting. At this moment the salivary glands contain α-esterases. The traces of β-esterase are determined only in imaginal parts of glands. The activity of esterases increases gradually, especially quickly between 0 and 30 hours after the 2nd moulting. In this time the activity of α-esterases is rather high and the traces of β-esterase can be seen in the larval part of salivary glands.

This period is actinomycin D-sensitive with respect to the β-esterase synthesis. RNA for the α-esterases synthesis is probably synthesized during the 2nd instar larvae time. The actinomycin treatment of larvae at 50 hours after the 2nd moulting does not decrease the esterases activity and can increase this activity to 70 hours.

Acknowledgement: The authors are very grateful to Prof. N. Sokoloff and Dr. M. Evgeniev for providing them with D. virilis stocks.

Reference: Korochkin, L., N. Matveeva, M. Golubovsky and M. Evgeniev 1973, Biochem. Genetics 10:363-393.

Valentin, J. University of Stockholm, Sweden. X-ray induced recessive lethals in the low recombination mutant, mei-1.

Recombination in the distal part of the X-chromosome is drastically reduced in D. melanogaster females homozygous for mei-1 (Hereditas 75:5-22, 1973). A pilot experiment has been performed to obtain a first approximation of the X-ray sensitivity of this mutant. 0-4 hr old virgins of genotypes mei-1/mei-1 resp. +/+ (control) were irradiated with 3640 r, and subsequently aged for 24 hr. Thereafter, they were mated to $Y^{S}X.Y^{L}$, In(1)EN+dl49,y v f car males, and progeny obtained during the following 6 days (4 broods) were tested individually for presence of X-linked recessive lethals induced in the oocytes of irradiated females.

Totally, 62 lethals were obtained in 1165 chromosomes in the mei-1 series (5.3%), and 92/2159 in the control (4.3%). Neither this total material nor any single brood shows any significant difference between mei-1 and control with respect to the frequency of X-ray induced recessive lethals. Since the test was both crude and small in size, only considerable differences in X-ray sensitivity between the series could have been detected. On the other hand, the suppression of recombination encountered with mei-1 is so effective that the total map-length of the X-chromosome is halved. Even with this pilot test, it is possible to state that differences in X-ray sensitivity of such a magnitude cannot occur between mei-1 and control.

130 of the total 154 lethals were also localised within the X-chromosome, using w ct f as markers. The distribution of the lethals over the X-chromosome, divided into 15 cM regions is shown in the Table. There is a significant difference between series, such that a higher

Table. Distribution of lethals in the X-chromosome in mei-1 and control.

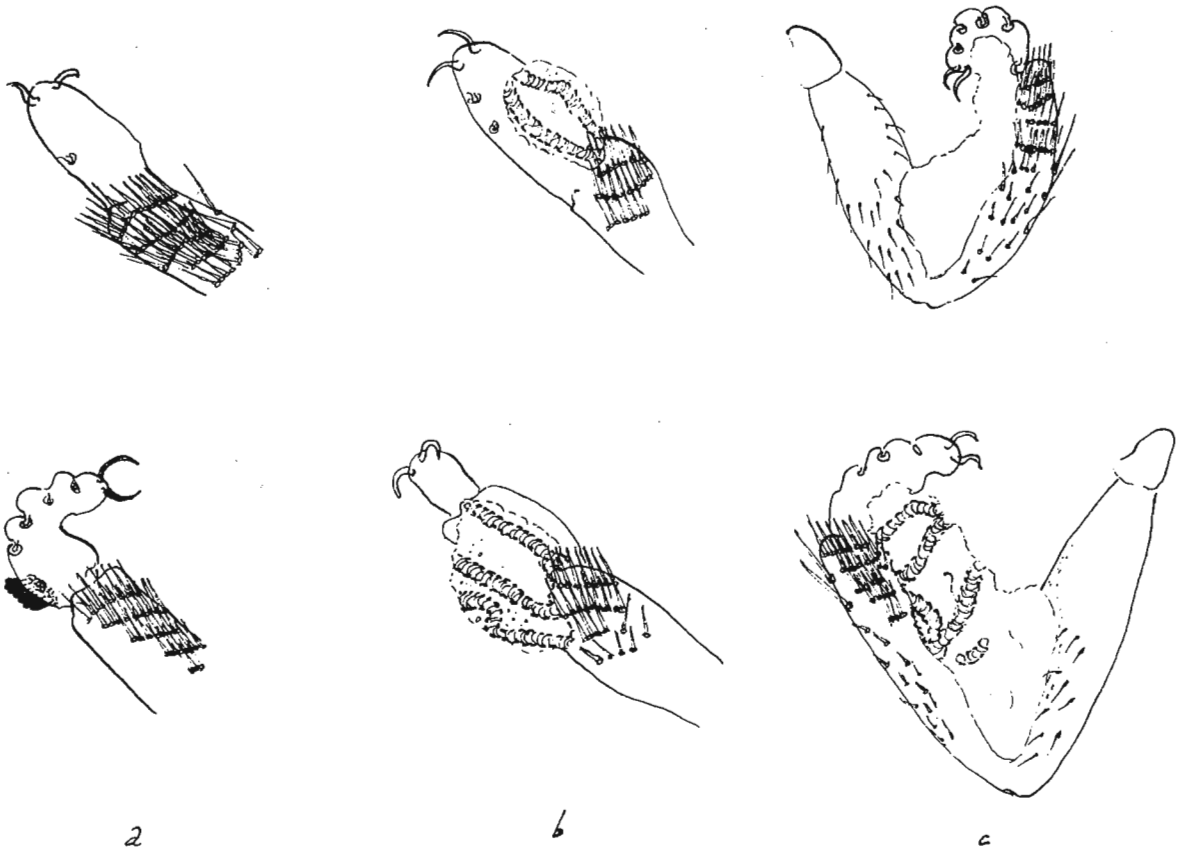
Map position	0-14.9	15.0-29.9	30.0-44.9	45.0-59.9	60.0-centromere
mei-1	19	6	7	7	12
control	8	24	18	13	16

$\chi^2_4 = 17.3$, $P = 0.002$

proportion of the lethals in the mei-1 series occur in the most distal region. This redistribution of lethals compared to the control situation mimics the effect of mei-1 on recombination, where a reduction is localised to the distal part of the chromosome while proximal parts are unaffected or show some increase of recombination. Thompson (Genetics 45:1567-1580, 1960) has shown that asynaptic chromosome regions are more sensitive than other regions to X-ray induced damage during meiosis. Thus, the present results are compatible with the hypothesis that mei-1 acts by local impairment of synapsis in the distal part of the X-chromosome.

Mglinetz, V.A. Institute of Medical Genetics, Moscow. Transdetermination in situ?

On culturing male foreleg imaginal discs in flies, G. Schubiger and E. Hadorn (Develop. Biol. 17:584, 1968) observed transdetermination of the leg blastema into proboscis in 2 of 289 test implants (0.7%). The elements of proboscis in the test implants (pseudotracheas, labellar kalotte) were found adjacent to the distal parts of the forelegs. I observed similar phenomenon in situ on the forelegs (in some rare cases on hindlegs, but never on the 2nd legs) of 33 among 13520 ss^{a40a} flies (0.24%). Three degrees of expression of such in situ transdetermination could be recognized: (a) considerable shortening and fusion of all the tarsal segments, (b) presence on one side of the reduced



tarsus of transparent appendage with a certain number of proboscial pseudotracheas, and (c) involvement of distal half of femur in the transformation. All attempts to select for or isolate the genetic factor (factors) controlling the transformation failed.

I observed also some cases of another type of in situ transdetermination: occurrence of eye facets on homoeotic legs of Nasobemia flies; similar phenomenon was also observed earlier in Hadorn's experiments with discs cultured in adult flies.

Gassparian, S. and Y. Nadjafi. University of Isfahan, Iran. Results and new mutants in Hossein-Abad.

A total of fifty-seven mutations and modifications were observed in Hossein-Abad population of D. melanogaster, three of which are presented as follows: 1. Concave-like compound eyes, with bright red eye color; 2. Short-winged males; 3. Flies with short wing and small body size. The pure stocks of the mutants are available.

Berg, R.L. 9 Colborne Rd. Apt. 11, Brighton, Massachusetts. A simultaneous mutability rise at the singed locus in two out of three Drosophila melanogaster population study in 1973.

The simultaneous rise in the rate of occurrence of a sex linked mutation singed (sn) in two of three D. melanogaster populations studied in 1973 is shown in Table 1. The identity with forked is excluded by crossing wild sn males and sn mutants arisen during the experiment with y v f B, Muller-5 females. All sn mutants are similar in their 100% penetrance, extreme expressivity and specificity of expression: abdominal bristles are curved inwardly. The two sn alleles found in the Magarach population and

Table 1. Concentrations and the rate of occurrence of singed in the populations of Drosophila melanogaster of Magarach, Dilizhan and Ararat.

Population	Year	Concentration sn & f mutants			Rate of occurrence sn & f mutants		
		N	Σ sn & f	%	N	Σ sn & f	%
	1937	7355	5[1]	0.069	9064	1[1]	0.011
	1938	1260	0		4339	0	
	1960	12821	0		49205	0	
						1 mosaic	
the winery	1963	11493	1[1]	0.009	27741	0	
Magarach	1965	7138	0		5679	0	
near Nikita	1966	9203	1[3]+1[2]	0.022	19474	0	
Botanical	1967	6502	0		6948	0	
Gardens,	1968	18311	0		27831	0	
the Crimea	1969	661	0		16314	0	
	1971	876	0		3336	0	
	1972	886	0		1920	0	
	Total	76506	8[1]	0.011	171851	1[1]+1 mosaic	0.0006
	1973	676	2[3]	0.296	15026	12[3]	0.080
	1939	5495	0		28051	1[1]	0.004
	1960	7523	0		6177	0	
	1961	1366	0		45892	0	
	1962	939	0		38996	0	
						1 mosaic	
Dilizhan,	1964	824	0		49393	3[2]	0.006
Transcauscasus	1965	478	0		24100	0	
	1966	173	0		-	-	
	1967	324	0		4570	0	
	1968	278	0		968	0	
	1969	793	0		3819	0	
	1971	836	0		2916	0	
	1972	274	0		137	0	
	Total	19303	0		205019	4[1]+1 mosaic	0.002
	1973	270	0		2687	4[3]	0.149
	1939	2881	0		8056	0	
	1961	2315	0		1350	0	
The winery	1965	5889	0		6665	0	
Ararat	1967	2111	0		1268	0	
in Erevan	1968	3521	0		5426	0	
	1969	1684	0		17509	0	
	1971	471	0		967	0	
	1972	504	0		331	0	
	Total	19376	0		32272	0	
	1973	1256	0		8423	0	
Totals	1937-1972	115185	8[1]	0.007	409142	5[1]+2 mosaics	0.001
Totals	1973	2202	2[3]	0.091	26136	16[3]	0.061

1) f and sn not identified and summarized; 2) f identified; 3) sn identified

the one arisen in the spermatogenesis of wild Dilizhan males proved to be alleles of one and the same gene. The Magarach alleles, although causing indistinguishable phenotypes, differ in respect to their mutability (Table 2). The mutable one (sn-7) reverts toward the normal state in 1 out of 274 gametes, while not a single reverse mutation was observed among 2818 gametes bearing the stable allele (sn-8). Reverse mutations cause only partial restoration of the normal state. One of the two reverse mutations arisen in the spermatogenesis of the wild sn

Table 2
Frequency of occurrence of sn mutations and sn reverse mutations among wild D. melanogaster males and their laboratory-raised progeny derived from three natural populations.

Mutation rate in the gametogenesis of flies	Magarach Population											
	Normal			sn-7			sn-8			sn-7 reverses		
	sn+	sn	%sn	sn-7	sn+	%sn+	sn-8	sn+	%sn+	sn-7 rev.	sn	%sn
wild males	669	1	0.149	105	2	1.305	64	0	0			
progeny wild ♂♂ x y v f B, M5 ♀♀	14357	11	0.077	2905	9*)	0.310	2754	0	0	2364	0	0
Total	15026	12	0.080	3010	11	0.365	2818	0	0	2364	0	0

Table 2 continued:

	Dilizhan						Ararat		
	normal			sn-11			normal		
	sn+	sn	%sn	sn-11	sn+	%sn+	sn+	sn	%
wild males	100	1	1.000				116	0	0
progeny wild ♂♂ x y v f B, M5 ♀♀	2587	3	0.116	1543	2	0.130	8307	0	0
Total	2687	4	0.149	1543	2	0.130	8423	0	0

*) In addition to these nine males with slightly abnormal bristles, due to germinal reverse mutations, arisen in the progeny of the wild sn-7 Magarach male-7, one F_2 sn male manifested a lateral thoracic area with normal bristles due to a somatic reverse mutation. No somatic reverse mutations were observed in the progeny of mutant sn-8 and sn-11 males.

male sn-7 is sn with 100% penetrance and weakened expressivity: only the tips of bristles of homozygous and hemizygous flies are hooked. The bristles of the other mutant are but slightly diminished, bent and deviate from their normal position and direction. Both alleles as well as all other reverse mutations are incompletely dominant in heterozygotes with the sn allele; this in respect to intermediate expressivity, penetrance in heterozygotes being 100%. No singed mutations arose among 2364 gametes of flies bearing reverse mutations. Reverted alleles are more stable than the normal allele of the sn gene. Several sn mutations arose in the y v f B, Muller-5 chromosome. Table 3 shows the roughly correct rate of their occurrence.

Table 3. Frequency of occurrence of sn mutations in the y v f B, Muller-5 chromosome.

Mutation rate in the gameto-genesis of flies	Origin of wild males											
	Magarach			Dilizhan			Ararat			Total		
	N	sn	%sn	N	sn	%sn	N	sn	%sn	N	sn	%sn
y v f B, M-5 laboratory stock	838	0	0	105	0	0	116	0	0	1059	0	0
offspring of y v f B, M-5 ♀♀ x wild ♂♂	≈18000	4	≈0.02	≈4000	2	≈0.05	≈8000	1	≈0.01	≈30000	7	≈0.02
Total	≈19000	4	≈0.02	≈4000	2	≈0.05	≈8000	1	≈0.01	≈31000	7	≈0.02

Mutant y sn v f B, Muller-5 males had no offspring.

The recent rise in mutation rate involves other genes as compared with the thirties when yellow was most abundant among sex-linked mutations (Berg et al. 1941; Berg, 1961; Kryshova et al. 1970). Mutability fluctuations are rhythmical in respect to the total number of mutations but not to the rate of occurrence of mutations at single loci. Each of the two peaks is formed by mutations of different loci. Yu. N. Ivanov in 1973 observed recurrent sn mutations in the progeny of wild flies captured in Tashkent and no sn mutations among approximately the same number of gametes in the progeny of flies from Samarkand (in press). The global rise of mutability at the singed locus is mosaic in nature: only three out of five populations are involved. The same was observed in the thirties when mutations arose frequently at the yellow locus. During 1937-1940 six populations were studied: Yellow males were found among wild flies and/or among their progeny of five populations. This global mosaic pattern of mutability fluctuations indicates that some external cosmic agent acts through mediators - supposedly mutant microorganisms which are widely distributed but not omnipresent. They intensify the mutagenic action of the primary physical agent and lend specificity to mutability changes. Different spectra of arising mutations in different populations involved in the mutability rhythm is one of the meaningful aspects of this specificity.

References: Berg, R.L. et al. 1941, Zhur. Obshchei Biol 2(1):143-158; Berg, R.L. 1961 Vestnik Leningr. Univ., Ser. Biol. 23(3):77-89; Kryshova et al. 1970, Genetika (Russ.) 6(3): 135-151.

Kuhn, D.T. Florida Technological University, Orlando. Relationships between eclosion, sex ratio, karyotype, penetrance, and larval competition in a tumorous-head strain of Drosophila melanogaster.

The present paper describes a comprehensive study designed to determine frequency of eclosion, sex ratio, karyotype and proportion of head tumors, among flies from a tumorous-head strain of D. melanogaster, when larval competition can be eliminated as a possible cause of the observed differences.

Thirty randomly chosen non-virgin male and female tuh(ASU) flies of various ages were placed in each of 8 empty 1/2 pint milk bottles that were inverted over a medium source. Eggs were collected every 12 hours for 8 consecutive days and transferred to 25 x 95 mm shell vials containing standard Drosophila media and reared at 25 ± 1°C. The eggs were distributed 10 at a time in the following manner: A) 800 eggs in each of 2 vials from day 1 through 7, on day 8 only 1 vial was used; B) 400 eggs in each of 2 vials each day on days 1, 3, 5 and 7, and in each of 3 vials each day on days 2, 4, 6 and 8; C) 200 eggs in each of 4 vials each day; D) 100 eggs in each of 8 vials each day; and E) 50 eggs in each of 16 vials each day. All of the resulting flies were counted and then classified according to sex, presence or absence of the tumorous-head trait, and karyotype.

The differences in sex ratio, karyotype, and penetrance of the tumorous-head trait in D. melanogaster cannot be accounted for by the larval competition resulting in vials containing 800, 400, 200, 100 or 50 eggs. Even in the 800 eggs per vial group the resulting lower viability (Table I), least aberrant sex ratio in favor of males (Table II), and lower overall penetrance (Table IV) were not significantly different from the results of those vials with fewer eggs.

When comparisons were made between the sexes, karyotypes, and tumor types, significant differences were found. It was demonstrated from the analysis of the aberrant sex ratio in favor of males that a highly significant interaction exists between the tumor type and karyotype (Table II). Woolf and Lott (1965) suggest that females are more apt to develop abnormally than males, and that heterozygosity for chromosome 3B increases the probability that female and male zygotes will survive to the adult stage in spite of the developmental abnormality. The high proportion of males observed among the tumorless heterokaryotypes is produced because fewer tumorless-heterokaryotypic females are present than tumorless-heterokaryotypic males. So that the expected aberrant sex ratio is even more enhanced due to the interaction between the sex and tumor type and the karyotype. The highly significant interaction between

the sex and tumor type that is responsible for the divergent proportions of homokaryotypes among the 4 sub-categories can be explained on the same basis (Table III). The higher proportion of homokaryotypes among the tumorless flies is present because proportionally the homokaryotypes are the most likely to be tumorless and heterokaryotypes the least. Since females are more able to tolerate the developmental problems associated with the abnormality than males, the highest percentage of homokaryotypes would be expected and is observed among the tumorless females, with the proportion of tumorless males also rather high. Therefore, the proportion of homokaryotypes for each sub-category is dependent upon the highly significant interaction between the sexes and the tumor types, where the tumor types are strongly influenced by chromosome 3B with In(3L)P st. Similar conclusions are reached concerning the results presented in Table IV. The buffering effects of In(3L)P st insure that heterokaryotypes develop the highest proportion of head tumors. Since females, evidently those with In (3L)P st, are more able to tolerate the developmental abnormality, the interaction between the sex and karyotype is responsible for the observed values. The overall results of this study

Table I. Viability of tuh(ASU) flies eclosing from vials in which 800, 400, 200, 100 or 50 eggs were placed.

		Egg transfer groups				
Category	Totals	Group A 800 eggs trans	Group B 400 eggs trans	Group C 200 eggs trans	Group D 100 eggs trans	Group E 50 eggs trans
% viability	17.1% (6,951/39,200)	15.4% 1,843/12,000	18.9% 1,512/8,000	18.1% 1,160/6,400	18.9% 1,210/6,400	19.2% 1,226/6,400

Table II. Sex ratio measurements of tuh(ASU) flies eclosing from vials in which 800, 400, 200, 100 or 50 eggs were placed.

		% males			
		tumored		tumorless	
Egg transfer groups	Totals	h 3A/h 3A	h 3A/3B	h 3A/h 3A	h 3A/3B
800	71.2% (1,313/1,843)	71.2%	70.4%	73.6%	81.0%
400	72.0% (1,088/1,512)	70.6%	72.3%	73.5%	78.1%
200	76.6% ((888/1,160)	78.4%	75.3%	75.0%	88.2%
100	76.2% (922/1,210)	80.7%	74.5%	63.0%	80.0%
50	72.2% (885/1,226)	77.3%	69.2%	70.0%	89.7%
50-800 mean % males	73.3% (5,096/6,451)	74.6% (1,501/2,012)	72.1% (3,249/4,508)	71.5% (133/186)	82.8% (193/233)

Source of variation	Degrees of freedom	Sum of squares	Mean square	F value
Total	19	382.49		
Treatments				
tumor type	1	29.67	29.67	3.31
karyotype	1	52.48	52.48*	5.86
tumor type x karyotype	1	148.73	148.73**	16.59
Egg groups	4	44.02	11.01	1.23
Error	12	107.57	8.96	

Table III. Frequency of tuh(ASU) homokaryotypes among the various categories derived from vials in which 800, 400, 200, 100 or 50 eggs were placed.

Egg transfer groups	Totals	% h 3A/h 3A			
		males		females	
		tumored	tumorless	tumored	tumorless
800	32.1% (592/1,843)	30.3%	36.4%	31.0%	46.7%
400	32.1% (486/1,512)	30.9%	43.9%	32.8%	50.0%
200	31.6% (366/1,160)	31.4%	44.4%	27.7%	66.7%
100	29.6% (358/1,210)	30.4%	37.8%	23.6%	58.8%
50	31.6% (348/1,147)	33.5%	44.4%	24.3%	75.0%
50-800 mean % h 3A/h 3A	31.5% (2,190/6,951)	31.6% (1,501/4,750)	40.8% (133/326)	28.6% (503/1,762)	57.0% (53/93)

Source of variation	Degrees of freedom	Sum of squares	Mean square	F value
Total	19	1301.87		
Treatments				
sex	1	87.65	87.65*	5.86
tumor types	1	766.94	766.94**	51.28
sex x tumor types	1	203.46	203.46**	13.60
Egg groups	4	64.35	16.09	1.08
Error	12	179.46	14.96	

Table IV. Penetrance of the tumorous-head trait among tuh(ASU) flies eclosing from vials in which 800, 400, 100 or 50 eggs were placed.

Egg transfer groups	Totals	% tumors			
		males		females	
		h 3A/h 3A	h 3A/3B	h 3A/h 3A	h 3A/3B
800	92.6% (1,706/1,843)	90.8%	92.4%	91.7%	95.6%
400	95.0% (1,437/1,512)	92.7%	95.7%	93.7%	96.8%
200	94.3% (1.094/1,160)	91.6%	95.0%	90.0%	97.9%
100	94.9% (1,148/1,210)	94.0%	95.6%	86.5%	96.7%
50	93.6% (1,147/1,226)	90.6%	94.1%	86.8%	98.4%
50-800 mean % Head tumors	94.0% (6,532/6,951)	91.9% (1,501/1,634)	94.4% (3,249/3,442)	90.0% (503/536)	96.9% (1,259/1,299)

Source of variation	Degrees of freedom	Sum of square	Mean square	F values
Total	19			
Treatments				
sex	1	3.26	3.26	0.69
karyotype	1	174.70	174.70**	36.90
sex x karyotype	1	42.25	42.25*	8.93
Egg groups	4	14.78	3.70	0.78
Error	12	56.81	4.73	

suggest that extensive interactions exist in determination of the sex ratio, proportion of homokaryotypes, and penetrance of the tumorous-head trait within the tumorous-head strain.

Reference: Woolf, C. M. and M.O. Lott 1965, Amer. Nat. 99:511-513.

Alexandrov, I.D. Research Institute of Medical Radiology, USSR Academy of Medical Sciences, Obninsk, USSR. The uniformity of detected frequencies of radiation-induced viable point mutations in the different post-meiotic male germ cells of D. melanogaster.

It is well known that the frequencies of mutations associated with chromosome breakage phenomena (translocations, partly recessive lethals) are different in the fully mature spermatozoa, immature sperm and spermatids of D. melanogaster when the males 3-4 days old are exposed to high doses of X-irradiation. The observed frequencies "produce characteristic 'U-shaped' curves" (Shiomi, 1967). The frequencies of viable point mutations at the specific loci seems "to be little, if any, different in all post-meiotic male germ cell stages" (Lefevre, 1967). Our results on other loci in other wild-type stocks confirm this statement.

Gamma-irradiation-induced (4 kr) mutability of the loci y^+, w^+, b^+, cn^+ and vg^+ in mature spermatozoa (first-day brood), immature sperm (second-day brood) and spermatids (3-5 day brood) of the males from two mass-bred wild-type stocks (D-18 and D-32) was studied. The stocks differ from one another in regard to both spontaneous and irradiation-induced breakage of the chromosomes. The treated virgin males 3-4 days old were individually mated to five virgin females of the genetic constitution $In(1)sc^{S1L}$ sc^{8R+} dl-49, y sc^{S1} sc^8 w^a;b cn vg. In making brood changes, the males were not treated with ether. The inseminated females of each brood were sub-cultured two times at 3-day intervals after which they were discarded. Mutants F_1 identified in each brood were thoroughly analyzed. Point (true gene) mutations have been separated from chromosome rearrangements according to the viability, fertility and results of the cytological and genetical (complementation, intergenic recombination) analysis. The results for all visible mutants on the whole are shown in the Table. It may be seen that the detected frequencies of true gene mutations at both stocks are practically the same in the

	Spermatozoa (1-day brood)			Immature sperm (2-day brood)			Spermatids (3-5-day brood)		
	Total	Mutations		Total	Mutations		Total	Mutations	
Stock	chr.	Point	Rearr.	chr.	Point	Rearr.	chr.	Point	Rearr.
D-18	98209	(29)*2.95**	(73) 7.43	66049	(14) 2.12	(68) 10.28	70877	(21) 2.96	(87) 12.26
D-32	86475	(20) 2.31	(62) 7.16	50926	(13) 2.55	(65) 12.74	49583	(12) 2.41	(41) 8.26

* In parenthesis the number of mutations found is given ** All frequencies x 10^{-4}

Table: The frequencies of viable point (true gene) mutations (all y, w, b, cn, vg on the whole) and rearrangements (all sterile, lethal and viable mutants resulted from aberrations) related to mutations at the specific loci in different male germ cells following gamma-irradiation (4 kr).

germ cells studied. The frequencies of rearrangements related to mutations at specific loci varied with irradiated germ cell stages differently for each of two stocks. In the light of these data it seems that processes which determine the final yield of detected genic and chromosome mutations in the germ cells are essentially different at the radiation mutagenesis.

References: Lefevre, G.Jr. 1967, Genetics 55:263-276; Shiomi, T. 1967, Mutation Res. 4:323-332.

Gassparian, S. and A.R. Safa. University of Isfahan, Iran. Allelism between three regions in Isfahan Province in Iran.

Two separate collections of fruit flies from each of the regions of Daran, Asgaroon and Isfahan show some allelism between detected mutations. The cases of allelism are shown in the Table. In all cases, the high frequency of allelism seem to be caused by a genetical drift in each of the populations.

Region	First collection	Second collection	Cases of allelism
Daran	22	23	10
Asgaroon	8	23	8
Isfahan	17	15	8

Begon, M. University of Leeds, England.

Drosophila and "dead" laboratory medium.

A common class of laboratory media for rearing Drosophila, are the "dead" media, David and Clavel (1965), Shorrocks (1971), so called because no live yeast is added to what is usually a cereal/killed yeast base. A very simple experiment has shown that "dead" is a misnomer in this context.

Eight 7.5cm x 3.0cm tubes were filled with Shorrocks' medium to a depth of approximately 1.5cm, and each tube provided with tissue paper for pupation and cotton wool plugs. Tubes, paper and plugs were all sterile. Three male and three female D. subobscura, taken from a population cage containing Shorrocks' medium, were added to each of six of the tubes. Flies were removed from two of the tubes after four days, from a further two after eight days, and from the remaining two after twelve days.

Five identical short cylinders of medium were then removed from the surface of each tube, serially diluted in sterile water, and the suspensions plated on malt-extract agar. The table shows standardised colony counts for each of the eight tubes as means (of five cylinders) with attached standard errors.

	Replicate	Yeast				Bacterium		Mean total colony count/ cylinder (with SE)
		A	B	C	D	X	Y	
No flies	I	1 colony in toto	0	0	0	0	0	-
	II	0	1 colony in toto	0	0	0	0	
4 days	I	0	0	49.6 ± 17.2	38.0 ± 15.4	1446 ± 171.7	0	1374 ± 130
	II	0	0	412 ± 78.3	39.2 ± 17.2	714 ± 104.9	49.2 ± 1.7	
8 days	I	0	0	0	108.6 ± 32.0	2252 ± 511.4	0	1950 ± 302
	II	0	0	138 ± 55.8	71.2 ± 65.1	1332 ± 185.8		
12 days	I	0	0	104.8 ± 23.6	19.4 ± 13.5	2714 ± 801	0	2581 ± 406
	II	0	0	1120 ± 111.4	164 ± 91.0	1040 ± 67.8	0	

The most immediate conclusion is that flies are instrumental in producing a very rich microbial flora on this "dead" medium, which really is dead in their absence. Furthermore t-tests on the total colony counts per sample for 4, 8 and 12 days, whilst not quite significant for the 4/8 and 8/12 comparison, show that the 12-day count is significantly larger than the 4-day count with $0.02 > p > 0.01$. There appears, therefore, to be a cooperative effect between the flies and the microbes, over this 12-day period at least, the flies presumably eating the microorganisms whose numbers in turn increase as the length of their association with the flies increases.

The importance of these results is that when flies from a different population cage were used in a similar experiment, similar results were obtained, but the yeasts and bacteria were different. Standardisation of laboratory media would therefore appear to be a more difficult task than is often realised, and studies of competition, fecundity, etc. in Drosophila must be at risk from differences in the "microbial luggage" of different flies.

References: David, J. and M.F. Clavel 1965, Bull. Biol. Fr. Belg. 99:369-378; Shorrocks, B. 1971, DIS 46:149.

Elens, A. Facultés Universitaires N.D. de la Paix, Namur, Belgium. Alcohol Dehydrogenase coding alleles and phototactic behaviour.

In a previous paper (DIS 49:71), it was reported how a selection for positive phototactism has succeeded, in a few generations, in obtaining phototactic substrains from an original ebony e^{11} strain. This strain was found polymorphic for the ADH^F and ADH^S alleles; but the new phototactic substrains are homozygous ADH^F/ADH^F.

To test the hypothesis that ADH^F/ADH^F ebony flies are more positively phototactic than ADH^S/ADH^S ones, other homozygous "Fast" (ADH^F/ADH^F) and "Slow" (ADH^S/ADH^S) ebony substrains selected from the same original ebony e^{11} strain (Libion-Mannaert, et al., in press) have been investigated for phototactic behaviour, according to the "countercurrent distribution method"

Table 1. Fractionation for positive phototactism.

	Number of responses toward light (countercurrent tube number)															
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
Ebony "Fast"	27	5	4	5	3	12	9	31	17	35	37	77	77	73	77	65
Wild Canton S	2	1	3	0	2	3	1	1	2	0	3	8	27	66	80	293
Ebony "Slow"	399	13	3	7	4	7	7	7	3	3	14	12	25	44	14	13
Wild Canton S	5	2	2	5	3	2	0	3	3	1	2	8	32	42	110	292

Table 2. Fractionation for positive and for negative phototactism.

Ebony "Fast"

Away from light (number of responses)	Toward light (number of responses)					
	0	1	2	3	4	5
5	0	1	0	2	11	2
4	1	1	2	2	3	4
3	1	1	0	0	4	4
2	0	1	4	9	5	3
1	2	0	7	10	5	3
0	148	69	96	100	67	44

Wild Canton S

Away from light (number of responses)	Toward light (number of responses)					
	0	1	2	3	4	5
5	0	0	0	0	0	3
4	0	1	0	3	0	10
3	1	0	5	1	3	3
2	0	3	2	3	7	6
1	0	3	1	0	6	6
0	45	14	20	65	101	271

Ebony "Slow"

Away from light (number of responses)	Toward light (number of responses)					
	0	1	2	3	4	5
5	1	0	0	0	0	0
4	1	0	0	2	1	1
3	2	2	0	2	3	0
2	1	0	1	3	0	0
1	3	2	2	4	1	0
0	455	67	67	55	53	27

Wild Canton S

Away from light (number of responses)	Toward light (number of responses)					
	0	1	2	3	4	5
5	0	0	0	2	0	1
4	0	0	0	0	0	4
3	0	4	0	4	9	2
2	0	0	1	4	2	15
1	1	0	1	0	7	8
0	50	12	8	50	81	307

of Benzer (1967). In each test wild flies of the same sex, from the strain "Canton Special" (homozygous ADH^S/ADH^S) were used as controls, in the same number as the ebony ones.

Table 1 shows the results of fractionation experiments according to positive phototactic responses. From such results, one could conclude that the "Slow" substrain is less phototactic than the "Fast" one.

However, other experiments in which the populations of flies are assayed for either negative or positive phototactic responses have shown that both "Fast" and "Slow" substrains are normally phototactic. But ebony flies are much more "sluggish" than wild ones. The flies of the "Slow" substrain, especially, move much less both toward and away from light (Table 2). According to Jacobs (1968), the very bad coordination of movements of ebony flies is a consequence of their higher concentration in beta-alanine, which also inhibits their glucose catabolism. To explain the less abnormal behaviour of the flies of the "Fast" substrain, we have suggested a special ability to use alcohol as a source of energy (Libion-Mannaert et al., in press).

The present results explain quite well how we have so easily succeeded in selecting phototactic substrains, which were ADH^F/ADH^F homozygous as well: the ADH^F/ADH^F flies run more quickly toward light.

References: Benzer, S. 1967, Proc. Nat. Acad. Sci. 58:1112; Jacobs, M.E. 1968, J. Insect Physiol. 14:1259.

Wright, T.R.F. and M.M. Green. University of Virginia, Charlottesville; University of California, Davis. T(Y;2)CyO,DTS-513, a new versatile translocation for making virginator stocks.

The translocation T(Y;2)CyO,DTS-513 was induced by X-raying CyO,DTS-513/Tft ♂♂ with 4000r (see DIS 48:89 for the origin of the CyO,DTS-513 chromosome). Apparently a translocation was induced between the Y-chromosome and the CyO,DTS-513 chromosome since Curly males carrying this translocation when mated at room temperature to wild type females produce predominatly wild type females and Curly male progeny. Occasional sterile non-Curly males appear along with even fewer Cy females. Both types of these exceptional progeny probably arise through primary non-disjunction of the X-chromosomes in the wild type female parent. No cytological analysis of the translocated chromosomes has been made.

When T(Y;2)CyO,DTS-513 males are mated to wild type females at 30°C the progeny consist almost exclusively of wild type females (virgins if parents are removed prior to eclosion of the progeny) with less than 1% sterile, non-Cy males. In a carefully controlled experiment 1000 eggs were laid, picked, and maintained at room temperature (22°C) and another 1000 eggs laid, picked, and maintained at 30°C. At room temperature the 1000 eggs gave rise to 210 + ♀♀ 135 Cy ♂♂, 2 Cy ♀♀ and 2 + ♂♂, whereas at 30°C 170 + ♀♀, 0 Cy ♂♂, 0 Cy ♀♀ and 6 + ♂♂ were produced. The stage distribution of mortalities of these eggs was: Rm. Temp. - Egg mortality 36.1%, Larval 27.6%, Pupal 1.4% for a Total Mortality of 65.1%; at 30°C - Egg mortality 35.3%, Larval 44.1%, Pupal 3.0% for a Total Mortality of 82.4%.

It is possible to construct virginator stocks for virtually any desired type of non-temperature sensitive female in two generations by crossing T(Y;2)CyO,DTS-513 males to females of the type to be virginated. F_1 T(Y;2)CyO,DTS-513 males are then backcrossed to the same type of female, and the new virginator stock is established by a second backcross of the appropriate type of F_2 T(Y;2)CyO,DTS-513 males to stock females of the desired type. (One less cross is needed to establish virginator stocks for X-chromosomes.)

The versatility of this method of virginating stocks is exemplified by the following list of virginator stocks constructed at Charlottesville of which only a few are still maintained since it is so easy to remake them. Stock A is the initial stock.

A. $\frac{+}{+}$; $\frac{+}{+}$ ♀♀ × $\frac{T(Y;2)CyO,dp^{lvI}Cy\ pr\ cn^2(DTS\text{-}513)}{+\ ;\ +}$ ♂♂

B. $\frac{w\ m\ f}{w\ m\ f}$; $\frac{+}{+}$ ♀♀ × $\frac{T(Y;2)CyO,DTS\text{-}513}{w\ m\ f\ ;\ +}$ ♂♂

C. $\frac{+}{+}$; $\frac{SM5}{Sp\ bw^D}$ ♀♀ × $\frac{T(Y;2)CyO,DTS\text{-}513}{+\ ;\ Sp\ bw^D}$ ♂♂

D. $\frac{+}{+}$; $\frac{SM5,al^2Cy\ lt^vcn^2sp^2}{al\ dp\ b\ \underline{l}(2)amd^{Hl}pr\ bw}$ ♀♀ × $\frac{T(Y;2)CyO,DTS\text{-}513}{+\ ;\ al\ dp\ b\ \underline{l}(2)amd^{Hl}pr\ bw}$ ♂♂

E. $\frac{+}{+}$; $\frac{CyO,dp^{lvI}Cy\ pr\ cn^2}{al\ dp\ b\ \underline{l}(2)amd^{Hl}pr\ bw}$ ♀♀ × $\frac{T(Y;2)CyO,DTS\text{-}513}{+\ ;\ al\ dp\ b\ \underline{l}(2)amd^{Hl}pr\ bw}$ ♂♂

F. $\frac{+}{+}$; $\frac{SM1,\ al^2Cy\ cn^2sp}{al\ dp\ b\ \underline{l}(2)amd^{Hl}pr\ bw}$ ♀♀ × $\frac{T(Y;2)CyO,DTS\text{-}513}{+\ ;\ al\ dp\ b\ \underline{l}(2)amd^{Hl}pr\ bw}$ ♂♂

G. $\frac{+}{+}$; $\frac{CyO}{al\ dp\ b\ pr\ Bl\ c\ px\ sp}$ ♀♀ × $\frac{T(Y;2)CyO,DTS\text{-}513}{+\ ;\ al\ dp\ b\ pr\ Bl\ c\ px\ sp}$ ♀♀

H. $\frac{+}{+}$; $\frac{+}{+}$; $\frac{TM3,Ser}{Sb}$ ♀♀ × $\frac{T(Y;2)CyO,DTS\text{-}513}{+\ ;\ +}$; $\frac{TM3,Ser}{Sb}$ ♂♂

I. $\frac{+}{+}$; $\frac{+}{+}$; $\frac{e(lethal\text{-}free)}{e(lethal\text{-}free)}$ ♀♀ × $\frac{T(Y;2)CyO,DTS\text{-}513}{+\ ;\ +}$; $\frac{e(lethal\text{-}free)}{e(lethal\text{-}free}$ ♂♂

One should note that not all of the above stocks were useful since the yield of virgin female progeny per bottle at 30°C may be very low. At Charlottesville yields are determined and virgins routinely collected by putting bottles started with 20 pairs of parents at 30°C on Friday (Day 0). The parents are then carefully cleared the following Friday (Day 7), and virgins collected on Monday (Day 10) and Friday (Day 17) after which the bottles are discarded even though a few more virgins will still hatch. Virgins are routinely stored in vials for 5 to 7 days before use. The absence of larvae in these vials verifies that the occasional males that hatch were sterile.

The SM5/Sp bw^D virginator stock (Stock C above) when first made produced 174.7 ♀♀/bottle with 3 Cy Sp bw ♂♂/bottle, but two months later it produced only 32.3 ♀♀/bottle plus 0.33 Cy Sp bw ♂♂/bottle. Just what happened is not known. Virtually the same thing happened to the SM5/al dp b 1(2)amd^{Hl}pr bw virginator stock (Stock D). It appears that the SM5 chromosome in these stocks does not do well at 30°C, but the CyO chromosome does very well. In an experiment designed to compare yields the SM5/al dp b 1(2)amd^{Hl}pr bw virginator stock produced 26.7 ♀♀/bottle and 1.2 al Cy ♂♂/bottle, the SM1/al dp b 1(2)amd^{Hl}pr bw virginator stock (Stock F) produced 227.4 ♀♀/bottle and 3.5 al Cy ♂♂/bottle, and the CyO/al dp b 1(2)amd^{Hl}pr bw virginator stock (Stock E) produced 330.2 ♀♀/bottle and 6.2 dp^vCy pr ♂♂/bottle. This last stock (Stock E) continues 10 months later to produce very high progeny yields at 30°C. Most of the male progeny from this experiment were tested for fertility and all those tested were found to be sterile even after 10-14 days at room temperature suggesting that they are X/O males derived from primary non-disjunction of the X-chromosomes in the female parents.

At some time there may be no alternative but to try to virginate a stock that normally produces very few progeny when raised continuously at 30°C. A significant increase in yield might be accomplished by shifting cultures from 30°C to room temperature at an appropriate time. Just such a procedure was tried in an attempt to increase the 32.3 ♀♀/bottle yield of the SM5/Sp bw^D virginator stock (Stock C). Bottles were initiated with 20 pairs of parents on Day 0 and were immediately put at 30°C. On Day 3 parents were cleared and the bottles replaced at 30°C for three more days. On Day 6 the bottles were removed from 30°C and placed at room temperature. Progeny were collected as usual on Day 10 and Day 17. This procedure more than doubled the yield up to 71.3 ♀♀/bottle along with 0.6 Cy Sp bw ♂♂/bottle.

The initial stock, T(Y;2)CyO,dp^{lvI}Cy pr cn^2(DTS-513) (Stock A), can be obtained from the Pasadena, California and Bowling Green, Ohio, stock centers.

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Zacharopoulou, A. University of Patras, Greece. Seasonal variations of inversion polymorphism in a wild Greek Drosophila melanogaster population.

The present note constitutes a preliminary communication of a wider investigation which is still in progress.

The flies were captured in a place called Koleika, which lies at a distance of about 8 Km N.E. of Patras (largest town of Peloponnese). Collections were made during Summer and Autumn, 1973. Captured males were individually mated with virgin Or-K female homozygotes for the standard gene arrangement. In order to know their karyotype, eight larvae in the progeny of each wild male were examined. Moreover, a single male (son) from the progeny of each wild female was analysed in the same way, except for a few cases in which we had not been able to study but a haploid set of chromosomes. The breakpoints of the inversions were identified on the basis of the salivary gland chromosome maps provided by Bridges and Brehme (1944) and Lindsley and Grell (1967).

Table I.

Season of capture	No. of individuals examined	No. of chromosomes tested	No. of chromosomes bearing inversions	Total inversion frequency - %	
Summer (August)	81	156	25	16,3	$X^2$1df=0.7 P > 0.05
Autumn (November)	136	270	52	19.26	

Table II. Numbers, types & distribution of inversions found in Summer & Autumn collections

Chromosome	Types of inversions	No. of wild chromosomes bearing inversions	
		Summer	Autumn
2L	22D-34A	2	8
	26A-33E	1	-
2R	47F-60A	1	-
	54C-60D	-	2
	52A-60A	1	-
	52A-56F	2	2
3L	63C-72E	3	10
	66A-79B	-	1
	69C-72C	-	2
3R	89D-96A	4	12
	92D-100F	11	15

Our results are gathered in Tables I and II. It should be also added that the inversion frequency found in wild males as well as in the sons of wild females of both collections did not differ significantly. Hence, we have pooled the data together. Table I shows no significant difference between the total inversion frequencies in the two seasons. However, as it can be seen in Table II, eleven types of inversions were detected from this population, the distribution and frequency of which seems quite different in the two seasons. No conclusions or plausible explanations can be suggested for the time being; more data are needed and, as previously mentioned, the investigation is still in progress.

Bicudo, H.E.M.C., M.K. Hosaki and J. Machado Faculdade de Filosofia, Ciências e Letras de São José do Rio Preto, São Paulo, Brazil. New arrangements in D. austrosaltans.

D. austrosaltans from the saltans group and saltans subgroup is known as exclusively occurring in Brazil. Magalhães (1962) reported this species from Piraçununga (State of São Paulo) and Carolina and Imperatriz (State of Maranhão); Bicudo (1973) reported it from Mirassol (State of São Paulo). This occurrence in regions so far apart as the States of São Paulo and Maranhão, together with the results of interspecific cytological analysis brought about by the last mentioned author, led her to admit a wider geographic distribution of this species and its probable occurrence in other Brazilian regions, especially Central Brazil. In January 1973, D. austrosaltans was collected in Campo Grande (State of Mato Grosso), partly confirming such an assumption. The present note is concerned with the cytological analysis of this new sample.

Six strains initiated with single females inseminated in nature were analyzed in relation

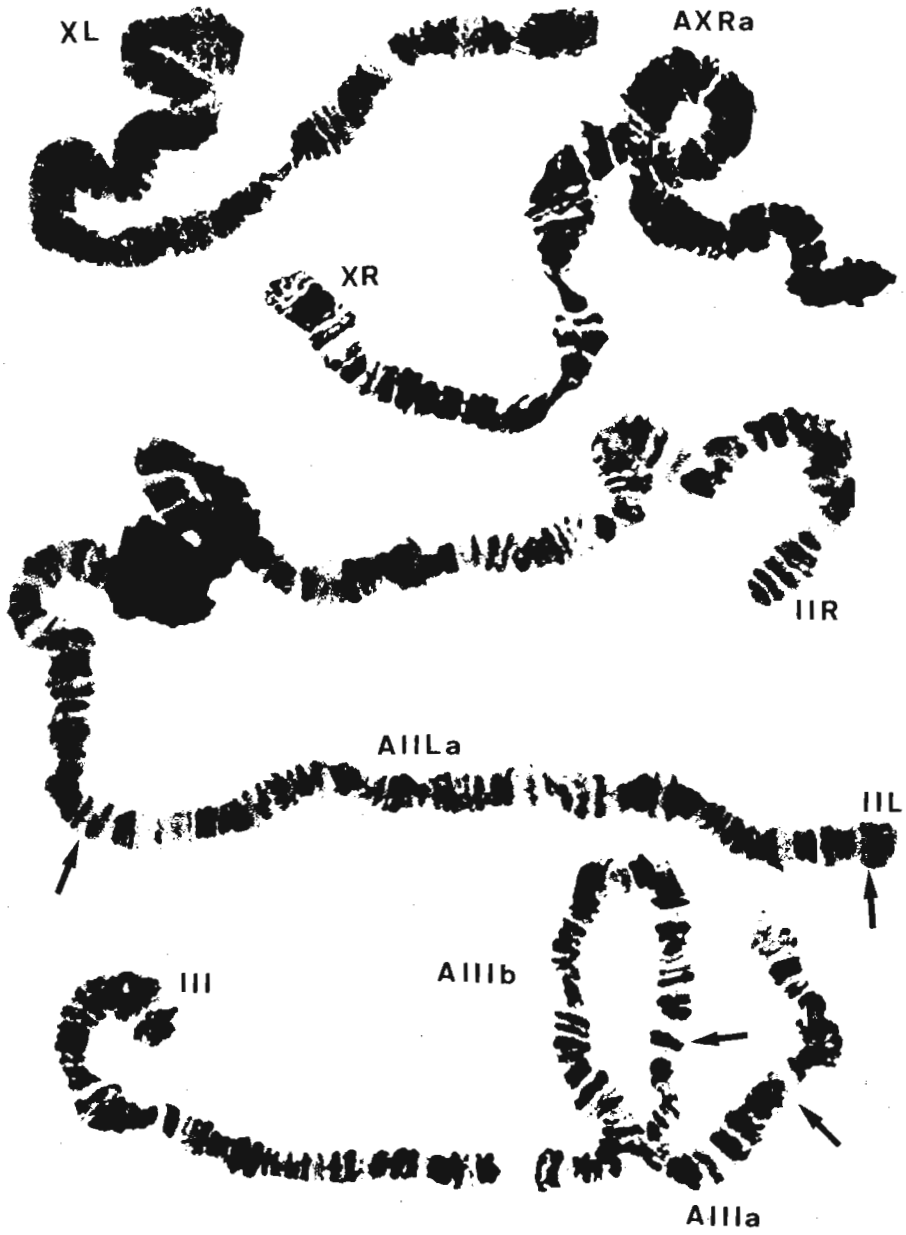


Figure 1. Chromosomal arrangements in D. austrosaltans: the standard arrangements in chromosomes XL, IIL, and the new inversions in the XR and III. Arrows indicate the breakpoints of the inversions previously described in the IIL and III. (See note by Bicudo, Hosaki and Machado on the preceding page.)

to the same standard strain used by Bicudo (1973). Two new intraspecific inversions were found: one in the XR arm (AXRa) and the other in the chromosome III (AIIIb). With the inversions AIILa and AIIIa, previously described, a total of four inversions has been recorded in this species. This number put D. austrosaltans in almost the same situation as D. saltans which, with six inversions, is the second species, in decreasing number order of inversions, in the saltans subgroup. In Figure 1 (next page) the arrangements found in Campo Grande and the breakpoints of the inversions previously described by Bicudo (1973) are presented.

Table 1

Strains		Chromosomal arrangements				
Origin	Reference	XL	XR	IIL	IIR	III
*Piraçununga (SP)	A_1	+	+	a	+	a
*Mirassol (SP)	A_2 & A_3	+	+	+	+	+
Campo Grande (MT)	A_4, A_5 & A_6	+	+/a	+	+	+/b
	A_7	+	a	+	+	+/b
	A_8 & A_9	+	a	+	+	+

* Bicudo, 1973

Table 1 shows the distributuon of arrangements found up to the present in D. austrosaltans. It may be seen that the strains from the three origins are distinguishable on the basis of their chromosomic constitution: Piraçununga and Campo Grande by the presence of inversions which are exclusive in each of them; and Mirassol by the presence of the standard arrangement in homozygous condition for all of its chromosome arms. The standard arrangement for all chromosomes is also present in Campo Grande, appearing however in heterozygous condition in chromosomes XR and III.

Acknowledgements are due to the Fundação de Amparo à Pesquisa do Estado de São Paulo, and to the graduate students S.F. Pereira and E.J. Marques who collected the sample analyzed.

References: Bicudo, H.E.M.C. 1973, Genetica 44:520-552; Magalhães, L.E. 1962, Univ. Texas Publ. 6205:135-154.

Zouros, E. and C. d' Entremont. Dalhousie University, Halifax, N.S., Canada. Sexual isolation among populations of D. mojavensis race B.

D. arizonensis and D. mojavensis constitute a pair of sibling species capable of exchange genes under laboratory conditions. Males from the cross arizonensis x mojavensis are sterile, but females are fertile; the reciprocal cross gives fertile hybrids of both sexes. Mettler (DIS 38:57, 1963) divided mojavensis into two races: race A has been collected from the desert regions of Southern California and the most northern part of Baja California; race B is abundant in Sonora, Baja California and the islands of the Gulf of California. Race A tends to be larger in size and to have a lighter body color. Laboratory crosses between the two races produce numerous fertile offspring. Zouros (Evolution 27:601, 1974) used the term subrace BI to denote population of mojavensis from Sonora and Sinaloa, and subrace BII to denote populations from Baja California and the neighboring islands of the Gulf of California. The two subraces have been reared from different cactus plants (Fellows and Heed, Ecology 53: 850, 1972) and shown to be cytologically (Johnson, Dissertation for M.S., University of Arizona) and genetically differentiated (Zouros, ibid.). We have now evidence of incipient sexual isolation between the two subraces.

Males and virgin females were collected from strains of mojavensis subrace BI which were homozygous for the slow form of the alcohol dehydrogenase locus ($ADH^{.85}$) and from strains of mojavensis subrace BII homozygous for the fast form ($ADH^{1.00}$), and released into a population cage. Care was taken that all males of cages 1 and 2 be of the same age. This was the case for the males as well as the females of cages 3 to 6. The number of females from each subrace was equal in these cages. Five days after the establishment of the cage the flies were removed and the females were placed individually in food vials. After the appearance of larvae in the food females from cages 3 to 6 were assayed electrophoretically for ADH to determine the subrace to which they belonged. In all cases one or more offspring was assayed in order to determine the origin of the male to which a given female was mated. The results are given in Table 1. When the probabilities given in the Table are combined (Sokal and

Table 1. Numbers of intra and inter sub-race matings and the probability (P) that they could be attributed to change.

Cage	Females	Male BI	Male BII	BI/BI+BII in males	P
1	BI	6	1	0.50	0.062
2	BII	3	18	0.42	0.018
3	BI	4	1	0.25	0.016
	BII	4	19		0.277
4	BI	11	5	0.50	0.138
	BII	0	3		0.125
5	BI	1	0	0.50	0.500
	BII	0	8		0.004
6	BI	9	1	0.50	0.011
	BII	1	7		0.035

Rohlf, Biometry, p. 621) we obtain for BI $\chi^2 = 28.20$, d.f. = 10, $0.001 < P < 0.005$, and for BII $\chi^2 = 32.51$, d.f. = 10, $P < 0.001$. The subdivision of D. mojavensis race B in two subraces originally based on ecological differences now seems to be justified in terms of genetic and ethological differences as well. We are investigating now the relative importance of geographical and ecological separation in promoting this reproductive isolation. Biochemical markers in this context present a clear advantage over morphological ones which in many cases have been shown to interfere in the choice of mate.

(Supported by Grant No. A9624 from NRC of Canada.)

Vogel, E. and H. Lüers. University of Leiden, The Netherlands and Free University of Berlin, W. Germany. A comparison of adult feeding to injection in D.m.

In the course of a comparative study on the mutagenic effects of aryldialkyltriazenes in both Drosophila and the host-mediated assay, certain triazenes with a solubility less than 0.01% were weakly mutagenic when injected in the mouse but revealed strong mutagenicity when fed to Drosophila. The results let us compare in Drosophila the mutagenic effectiveness of 2,4,6-trichloro-phenyldimethyltriazene when fed or injected. The water solubility of 2,4,6-triCl-PDMT is about 0.001%. For comparison, the aziridine derivative Trenimon was used for positive control.

A modified technique of the method described by Lüers (Arch. f. Geschwulstforschung, 77-83, 1953) was applied for adult feeding. This simple but effective procedure (Figure 1) consists of a 30 ml glass filter (1D3 of Schott, Mainz, W. Germany) being large enough to treat about 30 flies. The filter is placed into a 100 ml beaker which contains 15-20 ml test solution. (For smaller amounts, the 15 ml filter 10D3 or the 8 ml filter 63D3 might be used.) The test solution should nearly contact the filter plate's lower part. A wick of cellulose-wadding is used to keep contact between the test material and the plate. A cotton ring around the filter's upper part prevents water evaporation. Filters contaminated with a mutagen are decontaminated and cleaned up by hot water containing a detergent, distilled water, 70% ethanol, ethanol and acetone. This procedure is particularly essential to clean the filters from insoluble ingredients.

In order to achieve a fine suspension of 2,4,6-triCl-PDMT, the triazene was completely dissolved in DMSO and then diluted with 5% sucrose solution down to 2% DMSO and the desired triazene concentration. 2-day old Berlin K males were fed a series of concentrations of 0.001 mM to 1.0 mM triazene for 3 days and the induction of recessive lethals in spermatozoa was tested (Table 1a). For 2,4,6-triCl-PDMT, there exists a clear-cut concen-

Table 1a. The induction of recessive lethals by 2,4,6-triCl-PDMT in sperm after adult feeding

Concentration (mM)	X-Chromosomes tested	n Lethals	% Lethals
0.001	1942	5	0.26
0.003	1017	6	0.59
0.01	411	10	2.4
0.10	295	18	6.1
0.25	165	26	15.8
0.50	374	66	17.6
1.0	739	171	23.1

tration-dependent induction of recessive lethals after adult feeding, despite the fact the higher concentrations were administered as suspensions.

Repeats with three different concentrations of 1.0 mM 2,4,6-triCl-PDMT show a sufficient reproduction of the data (Table 1b).

Table 1b. Reproduction experiment with 1.0 mM 2,4,6-triCl-PDMT

	Chromosomes tested	n lethals	% lethals
Expt. 1	298	65	21.8 ± 2.4
2	255	66	25.8 ± 2.7
3	186	40	21.5 ± 3.0

For injection, the 2,4,6-triCl-PDMT suspension was prepared in the same way but 0.7% NaCl was used instead of 5% sucrose solution. 0.2 µl of a 0.25 or 1.0 mM triazene suspension were injected according to the procedure described by Mollet and Würgler (see DIS 50:203). However in sharp contrast to its strong mutagenicity when fed, 2,4,6-triCl-PDMT was completely ineffective when injected (Table 2). No indication of a mutagenic effect of the triazene could be found in a brood pattern experiment with 1.0 mM.

Table 2. Failure to detect mutagenicity of 2,4,6-triCl-PDMT after injection

	Days after injection	X-Chromosome tested	n Lethals
0.25 mM	1. - 3.	416	1
1.0 mM	1. - 3.	502	1
	4. - 5.	498	-
	6. - 7.	509	-

In the Trenimon experiment, 2-day old Berlin K males were fed 10^{-2} mM Trenimon for 17 hours. For injection, 0.2 µl of 10^{-2} mM Trenimon in 0.7% NaCl was used. One brood of two day's duration each was set up (Table 3).

Table 3

	X-Chromosomes tested	n lethals	% lethals
Feeding	197	13	6.6
Injection	400	21	5.3

Apparently, the mutation frequencies do not differ noticeably, although the size of the experiment has been rather small. The lack of mutagenicity of 2,4,6-triCl-PDMT after injection might be a transport problem, that is to say, the insoluble triazene might have remained at the site of injection. This simple explanation is supported by the fact that diethylnitrosamine (DEN) which requires metabolic activation like 2,4,6-triCl-PDMT can be detected as a mutagen when injected. DEN, however, is water-soluble while the triazene is not. Failure of triazene activation after injection thus hardly can account for the negative result with the triazene. The problem considered here deserves particular attention in further studies, because the great majority of environmental constituents (e.g. most pesticides) to be tested for their possible mutagenicity are water insoluble compounds - in contrast to widely used reference mutagens like EMS, MMS, or Trenimon.

Shiomi, T., I. Yoshikawa and T. Ayaki. Nagasaki University, Japan. Relative biological effectiveness of 14.1 MeV neutrons to X-rays for mutation induction in relation to stage sensitivity in Drosophila melanogaster.

There is disagreement in the study of mutagenesis regarding the relative biological effectiveness of neutrons to X-rays in Drosophila. The causes of this discrepancy were thought due to the poor neutron dosimetry for the early reports, and then considered as due to the heterogeneity of sperm samples. Recently, there is evidence for an increased effectiveness of fission neutrons over those of higher energies.

Since 1965, we have studied the mutagenic effectineness of 14.1 MeV neutrons and 180-200 kV X-rays in D. melanogaster, and obtained the RBE values from induced frequencies of sex-linked recessive lethals and of 2-3 translocations after irradiation of mature spermatozoa (7-day-old males) and late spermatids (2-hr-old males). Employed dose range was 500-2380 rad. Canton-S males were mated with dual-purpose stock females (y sc^{S1} In49 sc^{8};bw;st p^{p}), so that tests for sex-linked recessive lethals and translocations between 2nd and 3rd chromosomes could be carried out simultaneously. The 7-day-old males divided into two groups, S1 and S2. S1 males were mated immediately after irradiation, whereas S2 and 2-hr-old males (T), one day later. Sperm samples were obtained from the first 6 hrs (brood A) and the next 18 hrs (brood B) matings (1 male:female).

Table. RBE values of 14.1 MeV neutrons to X-rays in D. melanogaster

X-linked recessive lethals

stage	brood A	brood B	pooled
S1	1.16	0.89	1.01
S2	0.71	0.99	0.85
S	0.94	0.93	0.94
T	1.51	1.57	1.56

2-3 translocations

dose	S	T A	T B
500	1.62	8.22	3.86
1000	1.30	4.10	2.37
1500	1.15	2.74	1.79
2000	1.05	2.06	1.46
2500	0.98	1.66	1.26

The frequencies of X-linked recessive lethals are markedly higher for mature spermatozoa than for spermatids in X-ray experiments. A regression analysis shows that the data on recessive lethals with either radiation are consistent with a linear increase with dose. A statistical comparison of the regression coefficients shows that in mature spermatozoa neutrons are less effective in producing recessive lethals than X-rays, whereas, on the contrary, neutron irradiation is significantly more effective in inducing lethals in late spermatids than X-rays. The RBE values were calculated as the quotient of the estimated regression coefficients obtained for the respective stages of sperm development.

The results obtained for translocations show also that neutrons in comparison to X-rays are more effective in producing genetic damage in the anoxic late spermatids than in the mature spermatozoa characterized by a higher degree of oxygenation. With neutrons, the translocation frequencies increase linearly with dose. On the other hand, X-ray induced frequencies increase with a power function of the dose. It reveals that frequencies of neutron induced translocations are higher in brood A than in brood B with both germ cell stages. As the results of these differences in dose-effect relationship, the RBE values for translocations depend on dose, and the shape of the dose response curves indicates that RBEs will tend to increase with decreasing dose. This tendency is more remarkable at the stage of spermatids than the spermatozoa. Calculated RBE values are shown in the Table.

The discrepancy of recent results on RBE values in Drosophila may be mainly due to the difference of LET. RBEs tend to increase with decreasing neutron energy from 15 MeV to the fission energy spectrum.

Tonzetich, J. Bucknell University, Lewisburg, Pennsylvania. Frequencies of recessive lethals in Drosophila melanogaster populations from Barbados.

During the month of January, 1974, males of Drosophila melanogaster were collected in the region of Holetown, Barbados. The frequencies of second and third chromosome recessive lethals were determined independently using the Cy/Pm;D/Sb method. Lethals were scored as those cultures having less than 3% wild type flies in the F_3. As seen in Table 1, the frequencies of recessive lethals are approximately the same for both chromosomes and comparable to those of continental North America populations.

Table 1. Frequencies of second and third chromosome recessive lethals.

	No. chromosome tests	No. lethal chromosomes	%
Chromosome II	120	43	35.1
Chromosome III	119	37	31.1

Mollet, P. and Th. Skripsky. Swiss Federal Institute of Technology, Zürich and Swiss Federal Research Station, Wädenswil, Switzerland. α-amanitin does not induce sex chromosome loss in ring-X bearing Drosophila melanogaster males.

The chemistry and action of α-amanitin was reviewed by Fiume and Wieland (1970). This component of a poisonous mushroom is a selective inhibitor of RNA polymerases in eukaryotic organisms. In connection with experiments on repair processes in Drosophila conducted in our laboratory (Gerth, 1973) the question arose as to whether α-amanitin could be a mutagen besides inhibiting transcription. We therefore tested this compound for its capacity to induce sex chromosome loss in ring-X bearing Drosophila males. The data are compared with results obtained in separate experiments with Tretamin (TEM), a potent mutagen.

Four day old males of the constitution R(1)2,y B/B^S Y y^+ were injected abdominally with 0.2 µl saline containing either TEM or α-amanitin using a microapplicator set (Mollet and Würgler, 1974). As controls we used untreated males. In other experiments we found approximately the same rate of sex chromosome loss with nontreated and with saline injected males (Mollet, Graf and Würgler, in preparation). One day after injection the males were mated individually to three virgin y sn females in vials. After 48 hours the surviving males were counted and then the flies were discarded. The progeny were counted and classified according to the phenotypes given in the Table.

Mutagenicity testing with Tretamin (TEM) and α-amanitin. R(1)2,y B/B^S Y y^+ males were mated to y sn females. F, yellow Bar-eyed females; M, singed Bar-eyed males; L, yellow singed males; PL, singed males + yellow singed Bar-eyed males; G, mosaics; N, exceptional phenotypes; TL, sum of L + PL + G.

Treatment of males	Concentration	Total progeny						Loss frequency	
		F	M	L	PL	G	N	% L	% TL
no injection	0	1994	1766	37	2	7	4	0.97	1.21
TEM	1.0×10^{-4}M	604	853	86	4	6	1	5.53	6.18
α-amanitin	1.1×10^{-5}M	1589	1701	21	0	3	3	0.63	0.72

The mortality of the males 3 days after treatment was as follows: No injection, 27% (12/45); TEM injection, 68% (28/41); α-amanitin injection, 34% (21/62). In the mutagenicity tests (see Table) we obtained with TEM over five times more sex chromosome loss than in the control. On the other hand, α-amanitin does not induce sex chromosome loss.

Acknowledgments: We thank Prof. F.E. Würgler and Drs. J.Fantes and U. Graf for stimulating discussions. The work was supported by grant No. 3.7040.72 from the Swiss National Foundation for Scientific Research.

References: Fiume, L. and Th. Wieland 1970, FEBS LETTERS 8:1-5; Gerth, J. 1973, Diplomathesis, unpubl.; Mollet, P. and F.E. Würgler 1973, DIS 50:202; Mollet, P., U. Graf and F.E. Würgler 1974, Archiv für Genetik 47:in press.

Ranganath, H.A. and N.B. Krishnamurthy.
University of Mysore, Manasa Gangotri, Mysore, India. Further studies on the competition between Drosophila nasuta and Drosophila neonasuta.

Interspecific competition studies involving chromosomally polymorphic and monomorphic populations of D. nasuta and D. neonasuta have revealed that irrespective of the strains in competition, D. nasuta supplants D. neonasuta. Here in all instances the initial frequency of the two competing species was 1:1 (25:25) (Ranganath and Krishnamurthy, 1973).

In an extension of these, interspecific competition studies were made with the initial frequency of 1 D. nasuta : 4 D. neonasuta (10:40). Simultaneously the interspecific competition process with 1:1 ratio was also followed in these two species. Serial transfer technique of Ayala (1965) was adopted to maintain the experimental populations. 4 replicates were made for each set of experiments. The experiment was conducted at 21°C. The females of these 2 species are morphologically (phenotypically) indistinguishable. However the males of the two species can be differentiated from one another. Males of D. nasuta have complete silvery frons while the males of D. neonasuta have silvery markings around the frontal orbits only. Therefore at each census the number of males of each competing species and the total population size were recorded. The cultures were maintained till the elimination of any one of the competing species.

Figures 1 and 2 illustrate the dynamics of the interspecific competition of D. nasuta and D. neonasuta initiated with 1:1 and 1:4 frequencies respectively. D. nasuta eliminates D. neonasuta when the initial ratio was 1:1 but that itself faces extinction when the founder population was in the ratio of 1:4. The species performance as measured by their mean number of males and the average population size maintained during competition are presented in Table 1. Perusal of this table indicates the reversal of dominance of the 2 contesting species in

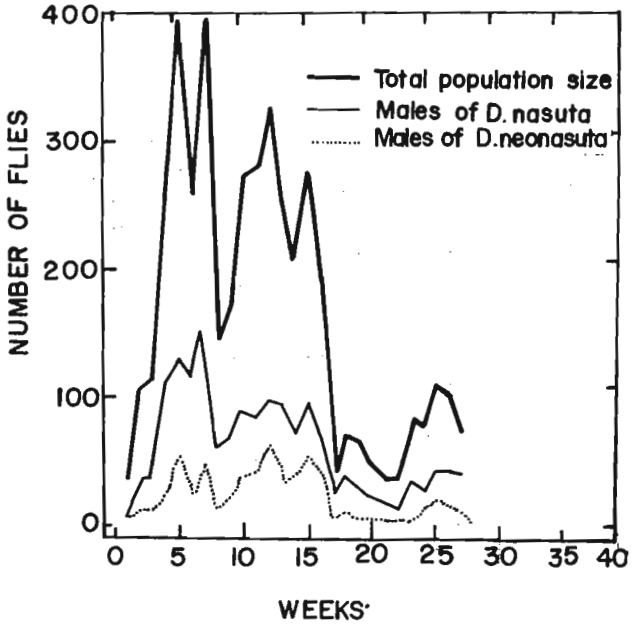


Figure 1. Total population size, and the number of males of D. nasuta and D. neonasuta during competition started with 1:1 frequency.

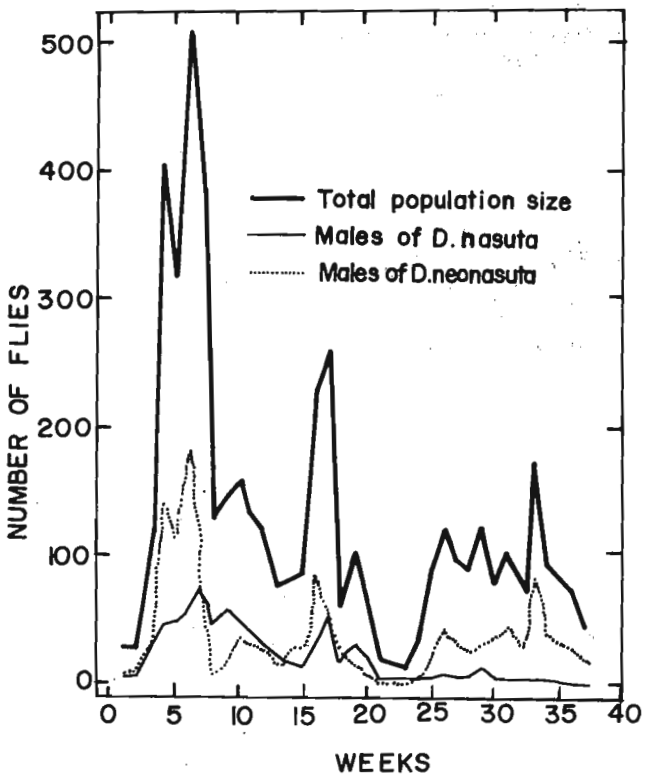


Figure 2. Total population size, and the number of males of D. nasuta and D. neonasuta during competition started with 1:4 frequency.

in the 2 sets of experiments.

Here the outcome of competition is determined by the initial frequencies of the competing species. The initial advantage gained because of its higher frequency of the founder popula-

Table 1. Mean number of males of D. nasuta and D. neonasuta and total population size maintained during interspecific competition.

	D. nasuta males	D. neonasuta males	Total population size
Expt. 1			
1:1	50.57 ± 6.93	14.09 ± 3.67	135.71 ± 18.17
"	54.75 ± 5.68	11.20 ± 2.13	140.29 ± 14.06
"	61.73 ± 7.18	23.57 ± 3.67	168.73 ± 22.49
"	44.42 ± 4.95	19.00 ± 3.14	137.07 ± 18.14
Average	52.86	16.96	145.45
Expt. 2			
1:4	22.92 ± 5.80	36.48 ± 9.60	118.62 ± 29.89
"	17.00 ± 3.89	41.10 ± 7.42	119.13 ± 22.38
"	26.13 ± 3.88	39.36 ± 7.76	140.10 ± 22.02
"	33.29 ± 7.65	44.12 ± 9.98	152.41 ± 33.19
Average	24.83	40.26	132.56

tion has made D. neonasuta to outvie D. nasuta. In the light of this, the universality of frequency dependent selection and the natural selection favouring a sparse species, remains to be further investigated.

Acknowledgements: The authors are deeply indebted to Dr. M.R. Rajasekarasetty, Professor and Head of the Department of Zoology for his help and encouragement. This work is financially supported by Mysore University research grants and C.S.I.R. New Delhi.

References: Ayala, F.J. 1965, Genetics 51:527-544; Ranganath, H.A. and N.B. Krishnamurthy 1973, DIS 50.

Hess, C. and E. Hauschteck-Jungen.
Strahlenbiologisches Institut der Universität Zürich, Switzerland.

Replication patterns of the distal end of the A-chromosome from the salivary gland of D. subobscura.

Bands are supposed to replicate their DNA once they have started without interruption to the end[1]. Therefore in a sequence of label patterns homologous bands are not interrupted in their label (right side of the table). However label patterns have been found which can not be arranged in the sequence without interrupting the label of several bands (exceptional label patterns, left side in the table).

A labelled band is covered with a minimum of two silvergrains as the background does not exceed 1 grain/31 x 10^{-3} μ^2 in each preparation.

Table: I to VII: numbers of bands of the distal end of chromosome A.
+: band labelled; -: band unlabelled

Exceptional label patterns:													
I	II	III	IV	V	VI	VII	I	II	III	IV	V	VI	VII
-	-	-	-	-	-	+	-	-	-	+	-	-	-
-	-	-	+	+	+	+	-	+	+	+	-	-	+
+	-	+	+	-	+	+	+	+	+	+	+	-	+
+	+	-	+	-	-	+	+	+	+	+	+	+	+
							+	+	+	-	+	+	+
							+	+	-	-	-	+	+
							+	+	-	-	-	-	-

References[1]: Arcos-Teran, L. 1972, Chromosoma (Berl.) 37:233-296; Haegele, K. 1973, Chromosoma (Berl.) 41:231-236.

Cuello, J. and M. Aguadé. University of Barcelona, Spain. Variation of the frequency of the allele ebony in experiments of selection for wing length.

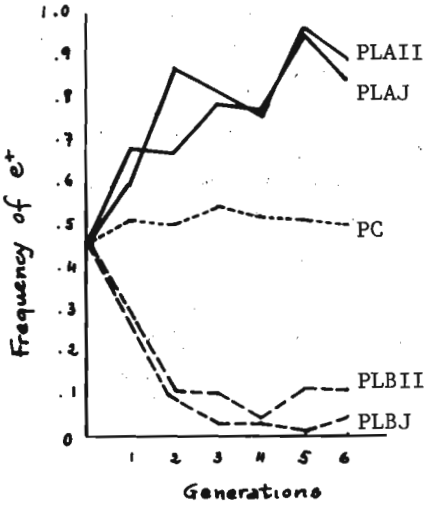


FIGURE.

Selection for wing length has been practised during six and nine generations in five different populations of D. melanogaster. Variation in enzyme frequencies has also been controlled during the course of selection (1). One of the strains used to start two of these populations has been marked with the allele ebony (2). In the first of these two populations there was actually an increase of the allele ebony in the lines selected for short wings. In the second population this variation has been followed quantitatively. Variation in the frequency of e^+ has been plotted against generation number (see figure: where PC = control unselected line; PLAI and PLAII = two replicates of long wing selected lines; and PLBI and PLBII = two replicates of short selected lines).

Although the gene controlling the character ebony is located on the third chromosome as well as those controlling the enzymes studied in these experiments (Est-6, Aph-1, Xdh and Lap-D), we have noted no linked response in the variation of the frequency of ebony to those observed at these enzymatic loci. If we consider that the strain marked with ebony (as well as other strains for ebony maintained in the laboratory) show a smaller mean wing length than the other e^+ strains, we can say that the character ebony has actually an effect on body size and that at 17°C the allele e is not being selected against in the control line - the frequency of the allele ebony has been maintained during the considered period.

References: (1) Aguadé, M., J. Cuello and A. Prevosti, In press; (2) Bridges, C.B. and T.H. Morgan 1923, Carn. Inst. Wash. Publ. 327:50.

Loukas, M. Agricultural College of Athens, Greece. The heritability of the character "duration of the pupal stage" in Drosophila subobscura.

As larvae pupated near the surface of the food, it was possible to collect the newly formed pupae daily and place them in vials until adults hatched. Thus we had a precise estimate of the duration of the pupal stage. The heritability was estimated as the slope of the regression line of the middle parent values on their progeny. 79 pair matings were formed by using individuals the pupal period of which was previously measured. In spite of our precautions for randomness, a positive correlation ($r = +0.383$) was found between parents. This coefficient is statistically different from zero ($P < 0.001$). This does not bias the estimation of h^2, but seems to reduce the variance of h^2 (Falconer 1960 p.171, Reeve 1965). The number of offspring in each family was found to be between 35 and 89 with a mean 53. Some 4,193 progeny flies have been used and their duration of pupal stage was also measured individually. No correlation was found between number of adults per vial and duration of pupal stage ($r = 0.0246$, $0.80 < P < 0.90$). Thus crowding does not seem to influence this character. The heritability was determined as the following:

Mean value of all parents	9.608
Variance of mid-parent values	0.129
Mean value of all offspring	9.789
Variance of offspring values	0.460
Covariance between mid-parent value and their offspring	0.0441

From these calculations the slope of the regression line, which estimates the heritability was found to be 0.342 ± 0.028.

The duration of the pupal stage as a part of the developmental time, is a component of r, the intrinsic rate of increase. Lewontin studying the selection for colonizing ability found that developmental time is one of the most important components of the selection for higher r. When populations increase in size, natural selection is toward higher r. In such cases we expect to be selected the factors influencing the time from pupation to eclosion.

Doane, W.W., R.E. Martenson* and G.E. Deibler*. Yale University, New Haven, Connecticut. Amino acid content of α-amylase from D. hydei.

α-Amylase-7 (EC 3.2.1.1, α-1,4-glucan-4-glucanohydrolase) was purified from the Zürich strain of D. hydei, homozygous for an Amy^7 allele, and a standard amino acid analysis performed on it (Doane et al., DIS 50:50, 1973, and in press). The average moles percent from duplicate runs is listed in Table 1, column two. For comparison, the moles percent of amino acids in α-amylases from several vertebrate sources were calculated from the literature and included in the table.

The tryptophan level is typically high in α-amylases; this was true of the Drosophila enzyme as well. Tyrosine, which is also quite high among amylases, did not appear in our analysis, but this may be due to residual acrylamide in the purified samples (see Lees and Paxman, J. Neurochem. 21:1031, 1973). This point is being clarified by a slight alteration

Table 1. Amino acid content of α-amylases from D. hydei and other sources.[1]

Amino Acid	D. hydei Amylase-7	Chick (Pa2) Pancreatic[2]	Rabbit (P2) Pancreatic[3]	Human (B) Parotid[4]
Tryptophan	3.3	3.2	1.6	3.2
Lysine	7.5	4.4	4.1	4.5
Histidine	2.4	2.6	2.0	2.3
Arginine	2.8	6.5	5.5	5.9
Aspartic Acid	12.1	14.5	14.7	15.3
Threonine	5.4	4.9	4.7	4.1
Serine	10.4	6.1	6.7	6.3
Glutamic Acid	7.4	7.3	7.2	6.7
Proline	3.8	4.3	4.1	5.2
Glycine	13.0	10.2	11.6	9.9
Alanine	8.0	6.4	5.4	4.7
1/2-Cystine or Cysteic Acid	2.7	1.1	2.1	3.4
Valine	6.4	7.3	8.1	7.2
Methionine	1.0	2.5	1.3	2.0
Isoleucine	4.0	4.6	5.6	5.4
Leucine	5.9	4.4	6.0	5.0
Tyrosine	0	4.4	4.3	4.1
Phenylalanine	3.9	5.3	4.8	5.2
Total:	100	100	100	100
Molecular Weight:	54500	55000	54000	56000[5]

[1]Amino acid residues are expressed as moles percent. A standard amino acid analysis was performed on amylase-7. Samples containing 0.5-1 mg protein were hydrolyzed for 24 hr at 110°C in constant boiling HCl in an evacuated desiccator, with or without 4% thioglycolic acid to preserve tryptophan. Hydrolyzates were analyzed on a Beckman 121 Amino Acid Analyzer operated in the high sensitivity mode employing a Beckman System AA Computing Integrator. Analyses for basic amino acids, tryptophan, and amino sugars were carried out on an 8 cm PA-35 resin column; neutral and acidic amino acids were analyzed on the standard AA-15 resin column. Cystine + cysteine and methionine were determined as cysteic acid and methionine sulfone, respectively, after performic acid oxidation.

[2]Lehrner and Malacinski, Third Intl. Conf. Isozymes, 1975, in press, and in preparation.
[3]Malacinski and Rutter, Biochem. 8:4382, 1969
[4]Kauffman et al., Arch. Bilchem. Biophys. 137:325, 1970
[5]Keller et al., Biochem. 10:4867, 1971

in our purification procedure which will eliminate acrylamide from the final product. Further comparison shows real differences in lysine, arginine, serine and alanine between α-amylase-7 and the other amylases listed in the table. In addition, the content of aspartic acid, glycine and phenylalanine appears somewhat different. Methionine and histidine residues are typically low in α-amylases, as the table indicates.

A small amount of galactosamine was detected chromatographically in the amylase-7 samples; this could not be quantitated. Carbohydrates are associated with some amylase molecules, but not all (cf. Watanabe and Keller, Biochem. Biophys. Acta 336:62, 1974 and Lehrner and Malacinski, 1975, in press).

To our knowledge, this is the first protein from Drosophila to be purified in sufficient quantities to be analyzed for its total amino acid residues. Preliminary results from an amino acid analysis on α-amylase-8, purified from the Chile (Amy^8) strain of D. hydei, indicate several significant quantitative differences between its residues and those of α-amylase-7. (Supported by USPHS grant GM 18729-02).

* National Institutes of Health, Bethesda, Maryland.

Miklos, G.L.G. and J. Smith. Australian National University, Canberra. Segregation in Drosophila melanogaster males carrying a predominantly heterochromatic free X duplication.

R.F. Grell (1958, 1968; X International Congress of Genetics Proc. Vol. 11; Genetics abs. s23) has presented data indicating that fertile XYY males of D. melanogaster may exhibit preferential segregation, the frequency of XY/Y:X/YY classes varying from 8.7:1 to 4.7:1 depending on the origin of the X or the Y. Furthermore XYY males exhibited semi-lethality and lethality. It should be stressed that Grell's results were obtained with almost completely isogenic backgrounds; a single second, third and fourth chromosome being made homozygous using standard balancing techniques. The two Y's in any given male were also identical Y's.

Merriam (1968, DIS 43:135) using $In(1)w^{m4}/y^+Y/y^+Y$, $In(1)w^{m4}/B^SY/y^+Y$ and $y/B^SY/y^+Y$ genotypes has also presented data showing non-random segregations. Once again the frequency of X-YY separations (18 percent) was lower than expected (33 percent).

We have begun studies on males carrying free X duplications marked with y^+ in order to test some properties of $X/B^SY/Dp(1;f)$ males. We report our preliminary findings here and also present initial results from $X/B^SY/y^+Y$ males, extensive genetic and electron microscopic studies of which will be presented elsewhere.

Table 1. Frequencies of recovered sperm types from $y/B^SY/y^+Y$ males mated to y females.

Y,Y	X	y^+Y	X,Y	B^SY	X,Y	Total Progeny
.09	.10	.22	.16	.23	.20	3241

It can be seen from Table 1 that X-YY separations from XYY males (19 percent) are in less than the expected random frequency (33 percent). There are indications from other experiments that in our system at least, progeny fertilised by either YY or X bearing sperm may have reduced viability. These results await confirmation from egg hatch studies, and an evaluation of zygote viability of all six classes is underway.

Table 2. Frequencies of recovered sperm types from $y/B^SY/Dp(1;f)y^+$ males mated to y females.

	Y,Dp	X	Dp	X,Y	Y	X,Dp	Total Progeny
Dp(1;f)52	.15	.15	.14	.15	.20	.20	2670
Dp(1;f)3	.13	.18	.17	.14	.19	.19	3485

The results of Table 2 indicate that in XYDp males approximately 40 percent of the separations are Y-XDp. The two free duplications tested in this study are at the large end of the spectrum as far as size is concerned; we have not investigated the behaviour of free X duplications which are substantially deleted in the basal heterochromatin.

It seems necessary now to determine what proportion of the six progeny classes in XYY and XYDp males has been influenced by viability complications and what is due to nonrandom segregations. At the moment it is not possible to decide if in fact XYY males exhibit nonrandom segregation at meiosis, or if the decrease in the classes fertilised by X or YY bearing sperm is due to viability problems. If indeed the segregation ratios from XYY and XYDp are truly so dissimilar, then XYDp and XYY_{frag} males may provide excellent systems for analyses of the extent to which X and Y heterochromatin is involved in interactions during male meiosis.

Lakovaara, S., P. Lankinen, J. Lokki, J. Lumme, A. Saura and A. Oikarinen. University of Oulu, Finland. The Scandinavian species of Drosophila.

The most extensive lists of Drosophila species occurring in northernmost Europe have been compiled by Hackman (1954) and Frydenberg (1956). They cover Finland and Denmark, respectively. We have for several years studied the Drosophila fauna of the Scandinavian countries and present here an updated list of species. It should be noted that the list is most incomplete for Norway. The list shows whether any species has been recorded from a country. The names of the Scandinavian countries are abbreviated as follows: Denmark = DK, Finland = SF, Norway = N and Sweden = S.

Subgenus: Scaptodrosophila
- deflexa (DK, S, SF)

Subgenus: Dorsilopha
- busckii (DK, N, S, SF)

Subgenus: Lordiphosa
- fenestrarum (DK, S, SF)
- forcipata (DK)
- nigricolor (SF)

Subgenus: Sophophora
- melanogaster group
  - melanogaster (DK, N, S, SF)
  - simulans (DK, SF)
- obscura group
  - alpina (N, S, SF)
  - ambigua (DK, SF)
  - bifasciata (N, S, SF)
  - eskoi (N, S, SF)
  - obscura (DK, N, S, SF)
  - subobscura (DK, N, S, SF)
  - subsilvestris (DK, N, S, SF)
  - tristis (DK, S)

Subgenus: Hirtodrosophila
- cameraria (SF)
- ingrica (SF)
- lundstroemi (SF)
- subarctica (N, S, SF)

Subgenus: Drosophila
- funebris group
  - funebris (DK, N, S, SF)
- virilis group
  - ezoana (N, S, SF)
  - littoralis (N, S, SF)
  - lummei (S, SF)
  - ovivororum (N, S, SF)
- repleta group
  - hydei (DK, S, SF)
- immigrans group
  - immigrans (DK, S, SF)
- quinaria group
  - limbata (DK, SF)
  - phalerata (DK, N, S, SF)
  - transversa (DK, N, S, SF)
- testacea group
  - testacea (DK, N, S, SF)
- miscellaneous
  - confusa (DK)
  - histrio (N, SF)
  - picta (DK, SF)

References: Frydenberg, O. 1956, Ent. Medd. 27:249-294; Hackman, W. 1954, Notul. Entomol. 34:130-139.

Vartanian, G. and A.J. Gallo. Faculdade de Filosofia, Ciências e Letras de São José do Rio Preto, São Paulo, Brasil. Genetic load of natural populations of Drosophila melanogaster from Brasil.

We have sampled 133 wild second chromosomes of D. melanogaster from natural populations of São José do Rio Preto, State of São Paulo, Brasil. The CyL/Pm technique described by Wallace (1956) has been used. By means of the Wallace and Madden (1953) technique we have estimated the frequencies of subvital, normal, and supervital chromosomes among the quasi-normal.

The observed results were: 19 or 14.29% of complete lethals (0% of viability in F_3 generation); 39 or 29.32% of complete lethals more extreme semilethals (0% - 10% of viability); 64 or 48.12% of lethals more semilethals (0% - 50% viability); and 69 or 51.88% of quasi-normal chromosomes (50% - 140% of viability). Among the quasi-normal we have found 15 or 21.74% of subvital and 54 or 78.26% of normal chromosomes.

Between the 55 chain crosses of the control experiment we have found 1 or 1.82% of semilethals; 3 or 5.45% of subvitals; 50 or 90.91% of normal; and 1 or 1.82% of supervital chromosomes.

References: Wallace, B. 1956, J. Genet. 54:280-293; Wallace, B. and C. Madden 1953, Genetics 38:456-470.

Mandal, S., R. Rahman, A. Nag and A.S. Mukherjee. University of Calcutta, India.

Effect of X-ray dose rate fractionation and mitomycin treatment on whole body and fractional mutations in the X-chromosome of Drosophila melanogaster.

Mutations in X-irradiated oc ptg males at specific loci of the X-chromosome of D. melanogaster, using the Muller's maxy stock, with or without pre- and post-treatment with mitomycin C were examined, in three consecutive two-day (post-meitoic) broods (a, b, c). The frequency of complete mutations with 1/2 KR to 4 KR (750 R/min) X-ray, increased linearly with the increase in the dose (regression coefficients for broods a and b were 0.0001 and 0.00015, respectively), while that of fractionals remained constant. This confirms the findings of Inagaki and Nakao (1966). Mitomycin C (MC) at 10 μg/ml and 100 μg/ml, (injected intra-abdominally) produced very few complete mutations, but at least with 100 μg/ml, it produced considerable amount of fractionals. The frequency of fractionals increased significantly from the first to the third brood ($P<0.05$). In the a and b broods, dose rate had no effect on either complete or fractional mutations (Table 1). But

Table 1. Effect of dose rate (total 2000R)

Dose rate	Brood a			Brood b			Brood c		
(R/min)	Nch	WB	F	Nch	WB	F	Nch	WB	F
600 R	15270	0.12	0.16	13611	0.20	0.26	2176	0.64	0.87
300 R	12161	0.19	0.24	10087	0.12	0.29	1894	0.63	1.00
100 R	8299	0.22	0.29	9525	0.13	0.23	5969	0.50	0.40*
60 R	10700	0.16	0.14	8860	0.19	0.24	968	0.52	0.10*

* Significant at 5% level by equality of proportion test.

in brood c the frequency of fractionals considerably decreased with decreasing dose rate from 600 R/min toward 60 R/min; the decreased frequencies at 100 R/min and 60 R/min dose rates were significantly different (tested by Cochran's 2 x 2 two-sided contingency test, $P<0.025$). Splitting the dose with a gap of 48 hrs did not affect either fractional or whole body. Both pre- and post-treatments of X-irradiated males with MC caused a significant decrease (tested by both 2 x 2 contingency and equality of proportion tests) in the frequency of fractionals in

Table 2. Effect of mitomycin pre- and post-treatment. MC-XR: pre-treatment of X-irradiated flies with mitomycin C, XR-MC: post-treatment with mitomycin C.

Treatment	Brood a			Brood b			Brood c		
	N	%W	%F	N	%W	%F	N	%W	%F
1. Control	6782	0	0.03	6320	0	0.03	5827	0.02	0
2. MC 100 μg/ml	7335	0.03	0.29^{c}	4369	0	0.64^{b}	3504	0	0.77^{c}
3. 2000 R (600-800R/min)	16756	0.15^{c}	0.28^{c}	13748	0.28^{c}	0.27^{c}	3068	0.55^{c}	0.39^{c}
4. MC + 2000R* (2 + 3)	24091	0.18	0.57	18117	0.28	0.91	6572	0.55	1.16
5. MC-XR	9057	0.19	0.39^{MCR}	6480	0.19	0.60^{MCR}	1308	0.31	0.84
6. XR-MC	10621	0.18	0.21^{MCR}	5723	0.26	0.40^{MCR}	1150	0.17	0.52^{MCR}

* In this set the data of MC and 2KR have been added up and not averaged. This is done on the basis of an assumption that MC and X-ray may produce an additive effect. Significance tests have been done on the basis of this assumption.

c Significantly different from control at 5% level, tested by equality of proportion test.

MCR Significantly different from MC+2KR data at 5% level, tested by equality of proportion test.

virtually all three broods, despite no change of complete mutations by such treatment (Table 2). These results suggest that the complete and fractional mutations differ not in the

mechanism of induction of premutational damage but rather in the processing and fixation of the premutational damage through "potential mutational determination" (Altenberg and Browning 1961; Dubinin 1968). The significant decrease in the same by dose-rate dilution, with a delayed effect, suggests further that at least a considerable proportion of fractional mutations are fixed through failure of proper functioning of the repair system. However, further work must be done in order to exclude the effect of the mutagens on cell selection (Tates 1968).

References: Altenberg, E. and L.S. Browning 1961, 46:203-212; Dubinin, N.P. 1968, Proc. XII Intern. Cong. Gen. 3:79-92; Inagaki, E. and Y. Nakao 1966, Mutation Res. 3: 268-272; Tates, A.D. 1969, Mut. Res. 5:109-116.

Denell, R.E. Kansas State University, Manhattan, Kansas. Preliminary analysis of the "triplo-lethal" region.

Lindsley and Sandler et al. (1972) characterized the effects of autosomal aneuploidy on survival to the adult stage in Drosophila melanogaster, and demonstrated the existence of a uniquely dose-sensitive region at salivary chromosome position 83D-E. Whereas most short heterozygous deficiencies and all other short duplications are compatible with survival, one or three doses of 83D-E in an otherwise diploid individual are associated with lethality.

We recently attempted to induce new genetic events (presumably duplications) that would complement the deficiency formed by the $3^{P}Y^{D}$ element of T(Y;3)L132 (broken in Y^{S} and at 83CD) and the $Y^{P}3^{D}$ element of T(Y;3)A109 (broken in Y^{L} and at 83E). C(1)DX, y;+/T(Y;3)A109, y^{+} B^{S} females were treated with 4000R of gamma radiation and crossed to $Y^{S}X.Y^{L}$, In(1)EN, y;In(3LR) TM6, ss^{-} bx^{34e} Ubx^{67b} e/T(Y;3)L132, y^{+} B^{S} males. We wished to recover $Y^{S}X.Y^{L}$, y; duplication/ $3^{P}Y^{D}$(L132), y^{+} + $Y^{P}3^{D}$(A109), y^{+} males, which should express a strong Hairy wing phenotype due to two doses of the Y-borne y^{+} duplication.

Among a total of 28,502 progeny, five Hairy wing males showed the genetic behavior expected of the event we wished to recover. That is, the treated third chromosome is compatible with survival only in the presence of the segregational deficiency. Specifically, the following test crosses were performed:

(1) Hairy wing males crossed to C(1)M3, y^{2} bb;TM6/T(Y;3)L132, y^{+} B^{S} females yield Hairy wing daughters and Bar Ultrabithorax sons. Moreover, when Hairy wing daughters are backcrossed to $Y^{S}X.Y^{L}$, y;TM6/T(Y;3)L132, y^{+} B^{S} males, their progeny consist of the expected Hairy wing males and Bar Ultrabithorax females.

(2) When Hairy wing flies from different lines are crossed inter se, they produce only Hairy wing progeny.

(3) When crossed to wild type flies, putative duplication/$3^{P}Y^{D}$(L132), y^{+} + $Y^{P}3^{D}$(A109), y^{+} individuals should yield no adult progeny. This prediction was fulfilled in initial test crosses for all five lines. It was noted that while there was extensive embryonic lethality among the progeny of these crosses, some individuals survived to the larval stage and a few survived to form pupae. In later crosses of 550 Hairy wing females from one of these lines (E6) to wild type males, two adult progeny did emerge. These flies were male-like in size, pigmentation, and sex comb development, but possessed intersexual genitalia. Their expression of Hairy wing was equivocal. While these progeny could represent new spontaneous genetic events, it seems likely that they represent rare aneuploids that have survived to the adult stage.

An analysis of the salivary chromosome complement of the E6 line confirms the presence of the two elements from T(Y;3)'s. The treated third chromosome shows a short repeat, with limits tentatively placed at 83C-D and 84B distal to the initial doublet. Thus the intersex phenotype is not due to aneuploidy in the more distal dsx or Ix regions.

Finally, Hairy wing males from the E6 line were crossed to C(1)RM, y^{2} sc w^{a} ec/In(1)FM6, y^{31d} sc^{8} dm B triploid females. Few progeny emerged, but they included putative hyper-triploid females and intersexes and a single putative hypo-triploid female. This cross is being repeated in greater numbers.

With respect to diploids, the best interpretation of these preliminary studies is that the original conclusion of a lethal effect of aneuploidy for 83D-E is correct, but that rare aneuploids may survive to the adult stage. In addition, hyper- and probably hypo-triploids also survive.

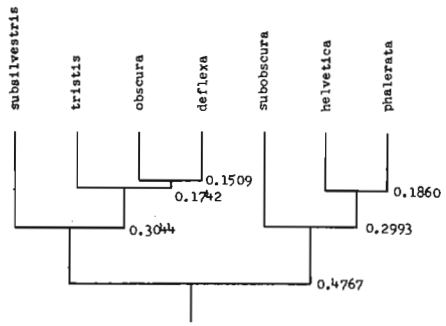
This work was supported by the Bureau of General Research, Kansas State University. A cobalt-60 source was made available through the courtesy of the Department of Nuclear Engineering, Kansas State University.

Shorrocks, B. University of Leeds, England. Food preferences in Drosophila.

During an undergraduate field course in June 1973 a series of comparisons were made in order to investigate the attractivity of Drosophila to a number of baits; the results given below may may be of interest. The experiment was carried out in Southern England using six baits - potato, carrot, mushroom, tomato, banana and apple - exposed in open boxes on the ground.

Species	potato	carrot	mushroom	tomato	banana	apple
subobscura	14	97	18	68	52	51
obscura	-	2	-	7	22	7
subsilvestris	-	-	-	1	3	-
tristis	-	1	-	-	6	4
helvetica	-	4	-	8	-	6
deflexa	-	5	1	5	14	9
phalerata	-	1	-	10	1	4

Three sites were examined and at each, all six baits were placed in a two metre diameter circle so that they would 'compete' for the flies. The results are shown in the table. Tomato, banana and apple attracted the most species although carrot attracted large numbers of D. subobscura.



It is possible to calculate the amount of overlap or joint use of a resource or resources, by two or more species using the formula:

$$d_{ij} = \sqrt{\left(1/2 \sum_{h} (p_{ih} - p_{jh})^2\right)}$$

This measures the geometric distance between two species (i and j) on a graph whose coordinates represent the frequency (p_{ih} and p_{jh}) in different environments (h). (Martinez et al., 1965; Shorrocks, 1974). The matrix of d values has been reduced to a dendrogram in which the numbers represent the geometric distance between species within a cluster. Although the baits used are not 'natural', the groupings may reflect a difference between the species in the wild.

References: Martinez, M.P., C. Maldonado and R. Levins 1965, Carib. J. Sci. 5:29-38; Shorrocks, B. 1974, J. Nat. Hist. 8:215-222.

Mather, W.B., M. Clyde and D. Lambert. University of Queensland, Brisbane, Australia. D. sulfurigaster albostrigata from Cebu, Philippines.

In August, 1973, 43 iso-lines were established. Four heterozygous inversions were detected in variable frequencies (see Table).

Photographs of these inversions have appeared in previous publications (Mather, W.B. and P. Thongmeearkom 1972, DIS 48:40 and Mather, W.B., P. Thongmeearkom, M. Clyde and D. Lambert 1975, DIS 51:86).

The material was collected and the iso-lines established by W.B.M. The chromosomes were prepared and analysed by M.C. and D.L.

Inversion	Type	Position	% Heterozygous frequency
C	Sim.	III D	80
E	Sim.	II L C	90
G	Sim.	I P	20
W_2	Sim.	III D	2.5

Note: Sim=Simple, D=Distal, C=Central & P=Proximal to centromere

Van den Haute, J. and A. Elens. Facultés Universitaires N.D. de la Paix, Namur, Belgium. Alcohol Dehydrogenase isozymes coding alleles and sexual competition.

A strain ebony e^{11}, from our laboratory stocks, is polymorphic for the ADH^F and the ADH^S alleles. From this polymorphic strain, "Fast" (ADH^F/ADH^F) and "Slow" (ADH^S/ADH^S) homozygous substrains have been derived (Libion-Mannaert et al., in press), which differ by their enzymatic activity level. They differ as well in sexual activity. Table 1 shows the matings recorded, in 4 hour observation periods, for 5 x 30 virgin pairs 4 to 5 day old flies, from the strain wild

Table 1. Sexual activity without competition.

1. Homozygotes

wild Canton Special	ebony e^{11} "Fast"	ebony e^{11} "Slow"
83/150	70/150	53/150

2. Heterozygotes wild - ebony e^{11} "Fast"

From mother wild	From mother "Fast"
132/150	143/150

3. Heterozygotes wild - ebony e^{11} "Slow"

From mother wild	From mother "Slow"
146/150	124/150

4. Heterozygotes ebony "Fast" - ebony "Slow"

From mother "Fast"	From mother "Slow"
82/150	90/150

"Canton Special" (ADH^S/ADH^S) used as control, the substrains "Fast" and "Slow", and heterozygotes produced by crossing them. In comparison with the wild control, sexual activity of the "Fast" strain is not significantly lower, although the "Slow" strain is indeed much less active. Heterosis effects are evident, in the products of almost every cross.

In competition experiments, the mating successes of four groups of 30 virgin pairs of flies were recorded at the same time in four observation chambers. The first chamber contained 30 pairs of wild flies, the second and third ones 15 pairs of ebony and 15 pairs of wild flies, the fourth one 30 pairs of ebony flies.

In the presence of ebony flies, the wild males become very active (much more than in the presence of females from their own strain), when the ebony males seem to be inhibited by the presence of the wild ones. As previously published in some other papers, ebony females are preferred by wild males; but our present experiment shows that matings with "Slow" ebony females are much more frequent than with "Fast" ebony females (Table 2).

Table 2. Sexual competition between wild Canton Special, ebony "Fast" and ebony "Slow".

	wild alone	Both strains in competition				ebony alone
	♂+ ♀+	♂+ ♀+	♂+ ♀e	♂e ♀+	♂e ♀e	♂e ♀e
1. Total flies	103/150 pairs wild	99/150 pairs wild	60	17	34/150 pairs ebony "Fast"	110/150 pairs ebony "Fast"
2. Total flies	84/150 pairs wild	71/150 pairs wild	84	12	21/150 pairs ebony "Slow"	81/150 pairs ebony "Slow"

Table 3 concerns two so called "B" substrains, an ebony one and a wild one, selected after more than fifty generations backcrossing ebony males with phenotypically wild females, originating from a cross between ebony e^{11} and wild "Canton Special". Both "B" substrains are homozygous ADH^F/ADH^F (Deltombe-Lietaert et al., in press). Competition experiments show that "B" ebony males are no more inhibited by the presence of wild "B" ones and that the ebony "B" females are no more preferred by wild "B" males. Heterogamic matings are less frequent

Table 3. Data concerning the "B" strains.

1. Sexual activity without competition

Homozygotes (wild)	Heterozygotes (father wild)	Heterozygotes (mother wild)	Homozygotes (ebony)
109/150	109/150	97/150	70.150

2. Sexual competition

	wild alone	both strains in competition				ebony alone
	♂+ ♀+	♂+ ♀+	♂+ ♀e	♂e ♀+	♂e ♀e	♂e ♀e
Total flies	126/150 pairs wild	74/150 pairs wild	45	37	50/150 pairs ebony	86/150 pairs ebony

than e.g. when "Slow" ebony flies are in competition with Canton Special wild (with the "Fast" substrain the difference is not significant). However, it remains a difference in activity level between wild and ebony "B" flies.

Hennig, W., I. Hennig and O. Leoncini. Institut für Molekularbiologie II der Universität Zürich, Switzerland, and Max-Planck-Institut für Biologie, Tübinger, Germany. Some observations on spermatogenesis of D. hydei.

The transcriptional activity of the fertility genes in the Y chromosome of Drosophila during primary spermatocyte development is one of the essential prerequisites for male fertility (review: Hess & Meyer, 1968). In D. melanogaster sperm differentiation proceeds even in the absence of the Y chromosome and only a final maturation step required for motility seems to be inhibited. In D. hydei, on the other hand, deletions or inactivity of one or several of the Y chromosomal fertility genes results in a drastic disorganization of sperm morphogenesis, often in very early developmental stages (Hess & Meyer, 1963; Hess, 1965; Meyer, 1968). In X/O males sperm development in D. hydei was initially reported to be arrested in the primary spermatocyte stage (Hess & Meyer, 1963; Hess, 1965).

Our recent studies suggest that additional genetic factors besides the activity of the Y chromosomal fertility genes may contribute to the abnormal morphogenesis of sperm in D. hydei in the case of Y chromosomal deficiencies. In several temperature sensitive Y chromosomal mutants induced by EMS never an inhibition of spermiogenesis in early development was found as it occurs in certain deficiencies of the Y chromosome obtained by X-Y translocations. Moreover, blocks in spermiogenesis prior to or during meiotic divisions were never observed in a multiplicity of mutations in the Y chromosome by EMS leading to complete sterility. Spermatogenesis in these cases always proceeds into postmeiotic stages. We also recently found that even in X/O males spermatogenesis proceeds considerably beyond meiosis and spermatids of advanced stages are regularly observed after aging of the X/O males. In consequence the Y chromosome is not required for the passage through the meitoic divisions or even to advance early postmeiotic development.

One explanation for the observed defects in early development might be derived from our recent conclusions on the location of ribosomal DNA in two different regions of the Y chromosome (Meyer and Hennig, 1974). In all Y deficient males studied by electron microscopy or DNA/RNA hybridization the number of ribosomal cistrons in primary spermatocytes was found to be reduced as compared to wild type spermatocytes. Since in these translocation stocks usually the X chromosomal nucleolus organizer is missing, we concluded that nucleolus organizer regions in the Y chromosome occur close to the kinetochore and in a terminal region on the long arm. It seems therefore possible that insufficient supplementation with ribosomes as a consequence of deletions in the nucleolus organizer regions introduces a retardation in the postmeiotic development or prevents the development into advanced stages of differentiation in case of particularly small numbers of ribosomal cistrons. In further studies on postmeiotic defects in spermiogenesis the contribution of deficiencies in the ribosomal DNA earns some special attention.

References: Hess, O. 1965, Chromosoma (Berl.) 16:222-248; Hess, O. and G.F. Meyer 1963 J. Cell Biol. 16:527-539; Hess, O. and G.F. Meyer 1968, Adv. Genetics 14:171-223; Meyer, G.F. 1968, Z. Zellforsch. Mikroskop. Anat. 84:141-175; Meyer, G.F. and W. Hennig 1974, Chromosoma (Berl.) 46:121-144.

Baldwin, D.G. and W.B. Heed. University of Arizona, Tucson. A newly-discovered inversion in the second chromosome of D. pseudoobscura.

In July, 1971, 9 female and 7 male D. pseudoobscura adults were aspirated from an oak slime flux at Butterfly Trail in the Santa Catalina Mountains, Tucson, Arizona. One of the females was heterozygous for a previously undescribed inversion of the second chromosome (see Figure 1). Standard is the only other gene arrangement that has been found on the second chromosome in this population. The third and X chromosomes are quite polymorphic; three gene arrangements exist for the third (ST, AR, CH) and the X has a relatively high frequency of the "sex ratio" inversions.

The proximal break point of the new inversion occurs between the two most basal heavy dark bands in section 48. The distal break point is in section 57, between the light doublet in the middle of the bulb and the dark doublet immediately distal to the bulb. The inversion is approximately the same length as the Zuni arrangement (1) but is shifted somewhat distally from it. It is considerably proximal to the subterminal Santa Lucia arrangement (2). Until it is found again in a natural population this new inversion will remain unnamed.

Figure 1. The heterozygous configuration of the new inversion and the Standard gene arrangement.

References: 1. Dobzhansky, Th. and A.H. Sturtevant 1938, Genetics 23:28-66; 2. Tan, C.C. 1935, Genetics 20:392-402.

Korochkin, L.I. and E.S. Belayeva. Institute of Cytology and Genetics, Novosibirsk, USSR. Organospecific slow esterase (S-esterase) in Drosophila of the virilis group.

Slow esterase fraction is present on electropherograms of single flies of D. imeretensis and D. montana. This esterase splits preferentially β-naphthyl acetate when the mixture of α- and β-naphthyl acetate is used as substrate. We designate this enzyme fraction as S-esterase. It was not observed in D. virilis, D. texana or D. littoralis. There is sexual dimorphism on this trait in D. imeretensis and D. montana. S-esterase can be seen only in males. We localized S-esterase in bulbus ejaculatorius of males and mainly in waxy plug. According to preliminary data, S-esterase as a component of waxy plug may transfer into female's sexual organs and accumulate within receptaculum. S-esterase appears in imago on 3th-4th day after emergence. Probably it is a variant of β-esterase (esterase-3). However this hypothesis must be verified. The activity of S-esterase in hybrid ♀ D. virilis x ♂ D. imeretensis is decreased or inhibited. The mechanism of this phenomenon is studied.

Acknowledgments: We are grateful to Prof. W. Baker for supplying us with flies used in this study and to Prof. N. Sokoloff and Dr. M. Evgeniev for different stocks of D. imeretensis.

Reference: Korochkin, L. and E.S. Belayeva 1973, DIS 50:125.

Steiner, W.W.M. University of Illinois, Urbana. Genetics of five allozyme loci in a Hawaiian Drosophila.

The only previously published data for segregation of biochemical polymorphisms in the Hawaiian Drosophila has been by Rockwood (1969). In a study of the acid phosphatase-1 (ACPH-1), alkaline phosphatase-3 (APH-3) and octanol dehydrogenase (ODH) loci, she presented data supporting a single locus, multi-allelic hypothesis for each.

To extend these studies, interstrain crosses were made using the endemic Hawaiian species D. mimica. Electrophoresis and staining of the resulting F_1 were then conducted via the methods of Steiner and Johnson (1973) to verify the genetics of five allozyme loci. These loci were ACPH-1; esterase-2 (EST-2, a beta-napthyl acetate specific, fast migrating zone of bands); phosphoglucomutase-1 (PGM-1, a medium migrating zone of bands); hexokinase-3 (HK-3, a medium migrating zone of bands) and isocitrate dehydrogenase (IDH, also a medium migrating zone of bands). The results are presented in Table 1 which confirms Rockwood's findings for

Table 1. Enzyme phenotypes of laboratory-raised females and males and their F_1 offspring.

Locus	Female phenotype	Male phenotype	No. of lines	F_1 phenotypes 33	34	44	45	55	Expected ratio	χ^2
ACPH-1	44	44	7			77			all homozygous	
	45	45	2			2	8	4	1:2:1	6.66
	44	34	3		7	13			1:1	1.80
	34	44	3		7	7			1:1	
	44	45	1			2	8		1:1	3.60
EST-2	44	44	3			27			all homozygous	
	34	44	1		1	6			1:1	3.84*
	44	45	1			5	2		1:1	1.28
	55	55	1					1	homozygous ?	
	34	34	2	2	7	6			1:2:1	2.19
PGM-1	44	45	3			14	13		1:1	0.04
	44	44	2			33			all homozygous	
	45	45	1			3	3	2	1:2:1	0.75
HK-3	44	44	2			21			all homozygous	
IDH	44	44	2			26			all homozygous	
	45	45	1			3	3	2	1:2:1	0.75
	45	44	1			8	2		1:1	3.60
	55	44	1				3		all heterozygous	
	55	45	1				4	4	1:1	
			38							

* $P < 0.05$

the ACPH-1 locus. Although limited, data for the EST-2, PGM-1, HK-3 and IDH loci are presented for the first time. The data indicate that each locus segregates in a Mendelian fashion for two or more alleles. The systems appear to be autosomal co-dominant in nature with only one mating in the EST-2 group demonstrating a significant deviation from the expected ($P = 0.05$). Additional data for PGM-1 is presented in Table 2. Here, male parental phenotypes are hypothesized and the expected ratios tested with the chi-square test for small samples. Deviations from expected here may be due to a multiple-mating or maybe a sampling effect. (see Table 2, next page.)

Comparisons of banding patterns were made visually with added controls for each gel. The allele codes comprising the phenotypes were assigned arbitrarily with the number 4 assigned to the most common and/or the most median-migrating allele.

EST-2 and PGM-1 demonstrate monomeric protein structure while the other three demonstrate a dimeric structure. Linkage relationships for these loci will be the subject of future investigations.

Table 2. PGM phenotypes of wild-caught D. mimica females and their F_1 offspring.

Female phenotypes	No. of lines	F_1 phenotypes 34	44	45	55	Male phenotypes	Expected ratio	χ^2
44	53		372			44	all homozygous	
45	1		7	3		44	1:1	1.60
55	1			4	2	45	1:1	0.66
34	2	4	9			44	1:1	1.90
44	15	40	62			34	1:1	4.32*
44	7		25	21		45	1:1	0.34
	79							

* $P < 0.05$

Acknowledgement: This work was supported by NSF grant No. GB23230 to the U.S.I.B.P. Hawaii subproject while the author was a graduate student in the laboratory of Dr. H.L. Carson. Gwen Arakaki gave technical assistance.

References: Rockwood, E.S. 1969, Univ. Tex. Publ. 6918:111-132; Steiner, W.W.M. and W.E. Johnson 1973, U.S.I.B.P./I.R.P. Tech. Report No. 30:1-21.

Tsusue, M. and C. Kitayama. Kitasato University, Sagamihara, Japan. Decomposition of eye pigment by bacteria.

Bacteria decomposes sepiapterin, eye pigment found in mutant sepia of D. melanogaster, whether they are bacilli or cocci. As sepiapterin is decomposed by the bacteria, a blue fluorescent product appears in the medium. This product was purified by combination of column chromatography using ECTEOLA-cellulose, P-cellulose and Sephadex G-25 as adsorbents. UV absorption spectrum of the product is identical with that of 2-amino-4-hydroxy-6-ethoxpteridine as shown in Figure 1. The pK values of the substance were determined to be 8.24 and 2.57 based on the changes in the absorbance at 255 nm

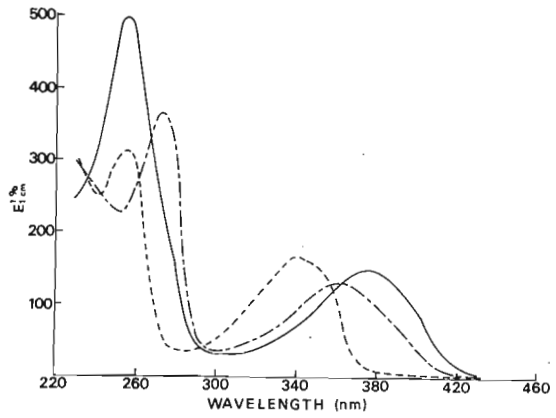


Figure 1. UV absorption spectrum of decomposition product. ————: in 0.1 N NaOH — - —: in H_2O -------: in 0.1 N HCl

and 272 nm in relation to pH. These values are also identical with those of 2-amino-4-hydroxy-6-ethoxypteridine[1], but the product is different from 6-ethoxypterin in its Rf values. On acid hydrolysis the product decomposed to yield xanthopterin. These data indicate the product is a derivative of xanthopterin. Since the first step of the decomposition of the eye pigment was not catalized by either sepiapterin reductase, sepiapterin deaminase or dihydrofolate reductase, the reaction was catalized by a new and presently uncharacterized enzyme.

References: 1. Pfleiderer, W., E. Liedek and M. Rukwied 1962, Chem. Ber. 95:755; 2. Kitayama, C. and M. Tsusue 1975, Agr. Biol. Chem. 39:903.

Kimoto, A. and C. Kitayama. Kitasato University, Sagamihara, Japan. Isolation and identification of bacteria from putrified sepia flies.

Sepiapterin is an eye pigment found in the sepia mutant of Drosophila melanogaster. This pigment is known to be a precursor of tetrahydrobiopterin, a cofactor in the enzymatic hydroxylation of aromatic amino acids. The authors found that sepiapterin is completely degraded in putrified sepia flies. From the heads of these flies three strains of bacteria were isolated and identified according to Manual for the Identification of Medical Bacteria. Based on their morphology, motility and biochemical properties, the first (K1) was identified as Proteus rettgeri, the second (K2) as Bacillus subtilis and the third as Bacillus of an unidentified species[1]. These bacteria decomposed sepiapterin when the pterin was added to the culture medium to yield a blue fluorescent product. Although the pigment did not affect appreciably the growth of these bacteria, the above described fact indicates an occurrence of enzyme catalizing decomposition of the pigment in these bacteria.

Reference: 1. Kitayama, C. and M. Tsusue 1975, Agr. Biol. Chem. 39:903.

Petříčková, V. and J.K. Benedik. J.E. Purkyně University, Brno, Czechoslovakia. The influence of genetic and environmental factors on viability of lethals.

Six lethal second chromosomes of D. melanogaster (three natural and three induced in the stock Oregon-K by 6mM solution of N-methyl-N-nitrosourea) were studied on three genetic backgrounds and in three temperatures, 22°C, 25°C and 28°C. As the background the first and the third chromosomes from the population Oregon-R, Krnov and Moravský Písek were used. 54 combinations of lethal, genetic background and temperature were made up and in each combination six repetitions from each of 17 males tested were examined.

Viability of second chromosomes was evaluated by the χ^2-test because in this manner the weighting for the number of flies counted was taken care of. The following classification of viability groups was used: No individual of standard phenotype - LETHAL; disagreement with theoretical ratio and less standards than 25% - SEMILETHAL; agreement with theoretical ratio - NORMAL; disagreement with theoretical ratio and more standard individual than 25% - SUPERVITAL.

In all lethal second chromosomes tested in this experiment in some repetition individuals of standard phenotype were found. Mann-Whitney's test did not reveal any difference between the group of natural and induced lethals as well as between particular genetic backgrounds although a generally positive effect of the background Krnov has been discovered. The main effect on the increase of frequency of non-lethal cultures showed temperature to be important, especially 28°C, where the frequency of non-lethal cultures was twice as high as those at 22°C or 25°C. This clear difference was also proved by Mann-Whitney's test (see Table).

Table. Mann-Whitney's test of differences between groups.

	Difference tested between			
Lethals	induced and natural	U = 337.5	$z < 1.96$	$P > 0.05$
Temperatures	22°C and 25°C	U = 333.0	$z < 1.96$	$P > 0.05$
	22°C and 28°C	U = 250.0	$z = 2.78$	$P < 0.01$
	25°C and 28°C	U = 250.0	$z = 2.78$	$P < 0.01$
Backgrounds	Krnov and Mor. Písek	U = 145.0	$z < 1.96$	$P > 0.05$
	Krnov and Oregon	U = 158.5	$z < 1.96$	$P > 0.05$
	Mor. Písek and Oregon	U = 153.0	$z < 1.96$	$P > 0.05$

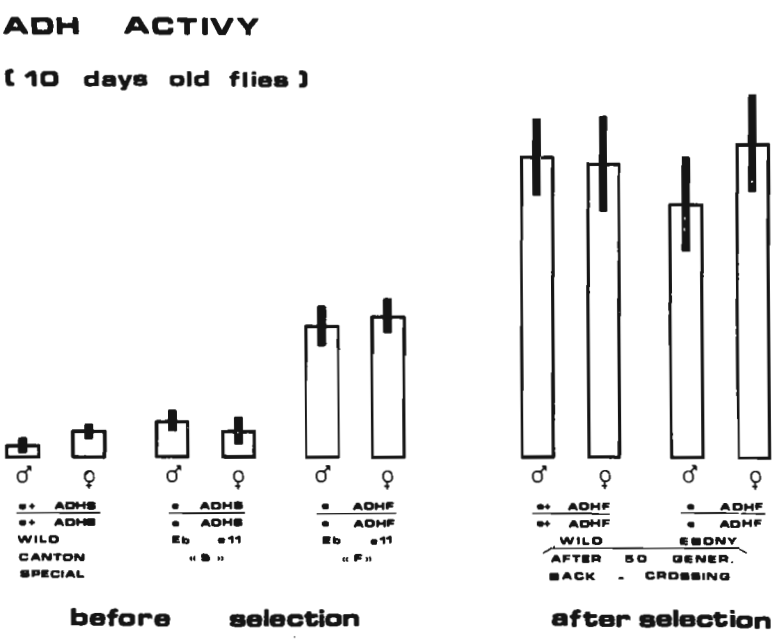
Also very interesting is the positive effect of the background of Oregon-R on the frequency of non-lethal cultures in the group of induced lethals. This clear interaction of one group of lethals with one background confirms the conclusions of Dobzhansky and Spassky (1968) on the positive role of the related genetic background in comparison with the remote ones on the viability of organisms.

References: Dobzhansky, Th. and B. Spassky 1968, Genetics 59:411-425.

Deltombe-Lietaert, C., M. Libion-Mannaert and A. Elens. Facultés Universitaires N.D. de la Paix, Namur, Belgium. Ebony gene and selective value of ADH isozyme alleles.

The present paper is concerned with the allozymes of Alcohol Dehydrogenase and the selective value of the alleles coding for it. It is known that populations of D. melanogaster contain two common ADH alleles, called "fast" (ADH^F) and "slow" (ADH^S) because of the electrophoretic mobilities of their products (Johnson and Denniston, 1964; Ursprung and Leone, 1965; Grell et al., 1965). A stock ebony e^{11}, present from many years in our laboratory, has been found polymorphic and in equilibrium, with frequencies of 0.71 for the ADH^F allele and 0.29 for the ADH^S one. Ebony males from this polymorphic stock have been crossed with wild females from the "Canton special" stock, which is homozygous for the ADH^S allele. The F_1 has been self-crossed. From the resulting F_2 progeny, ebony males have been allowed to mate with phenotypically wild females, and this process has been repeated for more than fifty generations in order to homogenize the genetical background. When screened for ADH profiles and tested for Alcohol Dehydrogenase activity, both ebony and wild substrains subsequently selected turned out to homozygous ADH^F/ADH^F and to have a very high ADH activity level (Figure 1).

How can be explained the complete elimination of the ADH^S allele, and the ADH activity level so remarkable elevation?



It is known that in natural population the ADH^F allele is more represented in cooler environments; it is in good agreement with the temperature characteristics of both isozymes, as studied in laboratory conditions (Vigue and Johnson, 1973 Day et al., 1974). Other environmental factors favor the ADH^F allele: in laboratory conditions it has been shown by Gibson that addition of alcohol in the culture medium succeeds in selecting for a higher ADH^F frequency. For Gibson such an observation may be relevant to natural populations, as there is probably a considerable variation in the amount of ethanol produced by yeasts fermentation in Drosophila habitats (Gibson, 1970).

Activity level is in general higher for the "fast" isozyme than for the "slow one. However, the range of variation is quite large, either in strains homozygous for ADH^S as in other strains homozygous for ADH^F (Birley and Barnes, 1973). Furthermore, in either kind of homozygous strains, it is possible to select artificially, in a few generations, substrains with a lower or with a higher activity level; it has been concluded that such strains "were indeed segregating for high and low modifiers of alcohol dehydrogenase activity" (Ward and Hebert, 1972).

In addition, we have to consider the prominent part played, in this selective process, by the presence of the ebony mutation. Jacobs has shown that homozygous ebony flies are deficient in getting energy through glycolysis (Jacobs, 1966). In another paper, we consider the hypothesis of a special ability of ADH^F/ADH^F homozygous flies to use ethanol as a substitute for carbohydrate food (Libion-Mannaert et al., in press). if such a special ability really exists, the ebony mutation could act as an internal selective factor favoring the ADH^F allele, as well as the polygenic modifiers, increasing its expression. Under conditions of very extreme competition, one might even consider that the ADH^S allele could be completely eliminated. However, as the heterozygotes ADH^F/ADH^S have about the same level of alcohol dehydrogenase activity as the ADH^F/ADH^F homozygotes, such a prediction micht very well be untrue. As a matter of fact, we have been able to demonstrate recently that heterozygotes ADH^F/ADH^S are sexually more active than either homozygotes, and that this selective advantage is even more pronounced in presence of alcohol. Because of that heterosis, it is most likely

that our ebony e^{11}, under normal conditions at least, would remain polymorphic; the perfect matching between the observed frequency of heterozygotes, and the theoretical value as predicted from the Hardy-Weinberg law, strongly indicates that it is in equilibrium with respect to gene frequencies.

The situation is quite different, of course, when the mutated gene ebony is in competition with its wild type allele, as in previous experiments in other laboratories (L'Heritier and Teissier, 1937; Elens, 1958), or in the present one. Since ebony flies cannot catabolize carbohydrates as efficiently as their wild competitors, an increased ability to use ethanol as an alternative source of energy would give them a definite selective advantage. In such conditions of severe competition, a complete elimination of the ADH^S allele has to be expected, and has been observed.

References: Birley, A.J. and B.W. Barnes 1973, Hered. 31:413; Day, T.H., P.C. Hillier and B. Clarke 1974, Biochem. Genet. 11:155; Gibson, J. 1970, Nature 227:959; Grell, E.H., K.B. Jacobson and J.B. Murphy 1968, Ann. N.Y. Acad. Sci. 151:441; Jacobs, M.E. 1968, J. Insect Physiol. 14:1259; Johnson, F.M. and C. Denniston 1964, Nature 204:906; L'Heritier, Ph. and G. Teissier 1937, C.R. Acad. Sci. Paris, Série Biol. 124:882; Ursprung, H. and J. Leone 1965, J. Exp. Zool. 160:147; Vigue, C.L. and F.M. Johnson 1973, Biochem. Genet. 9:213; Ward, R.D. and P.D.N. Hebert 1972, Nat. New Biol. 236:243.

Ives, P.T. and D.F. Demick. Amherst College, Amherst, Massachusetts. BHT modification of rates of induced mutagenesis.

Oregon-R males, reared on standard cornmeal-molasses food (containing methyl parasept), with or without 2 mg of BHT per g of cooked food, were aged for a day on well yeasted food and then kept in empty vials for eight hours. After being given a (one-minute) single drink of a .025 M EMS 1% sucrose solution, each male was mated to successive daily harems of six Basc females for five days and up to twenty F_1 females from each of his sperm broods were tested for X-linked lethals. One set of males, reared similarly, was exposed to 1kr of X-rays and mated singly to daily harems of six vg;se females, and their F_1 males were tested for translocations of the Y, 2, 3 chromosomes, omitting days 3 and 4 broods. The results of these tests are given in the Table with chi-square p values.

Mutation rates after EMS or X-rays in BHT-raised ♂♂

Sperm	EMS ONLY			BHT + EMS			
Brood day	Tests	X-le	%	Tests	X-le	%	p
1	1247	210	16.8	845	86	10.2	<.001
2	572	91	15.9	515	66	12.8	.150
3	316	58	18.4	428	58	13.6	.079
4	250	52	20.8	392	58	14.8	.047
5	269	59	21.9	607	74	12.2	<.001
	1 kr X-rays only			BHT + 1 kr X-rays			
Day	Tests	T's	%	Tests	T's	%	p
1 & 2	1059	22	2.1	513	3	0.6	.025
5	311	20	6.4	362	18	5.0	.42
6	210	21	10.0	198	24	12.1	.51

BHT is 3,5-di-tert-butyl-4-hydroxytoluene, an antioxidant and common food preservative. It has been found by R.B. Cumming (Genetics 64:s14, and personal communications) to reduce EMS mutagenesis, but not X-ray mutagenesis, in mice. Our data show that it also clearly reduces EMS mutagenesis in Drosophila. There is a suggestion that it may reduce the rate of X-ray induced translocations in near-mature sperm, but much more data are needed on this point.

(These data are from D.F. Demick's 1974 Senior Honors Thesis at the Webster Center for Biological Studies, Amherst College.)

Korochkin, L. and E. Beliaeva. Laboratory of Developmental Genetics, Institute of Cytology and Genetics, Novosibirsk, USSR.
The investigation of esterase isozymes in interspecific hybrids between D. virilis and D. imeretensis.

Hybrids between different stocks of D. virilis females and D. imeretensis males were produced. Using stock 160 of D. virilis with autosomes marked by the recessive mutations (2nd chromosome, broken; 3rd, gapped; 4th, cardinal; 5th, peach, 6th, glossy), we tried to localize esterase genes in D. imeretensis. For this purpose females of virilis 160 were mated to imeretensis males and then the F_1 to virilis 160 again. Analyzing electropherograms of esterases of single flies of different phenotypes, we observed that esterase genes in imeretensis are localized on the 2nd chromosome (like virilis, texana, littoralis and montana). In hybrid virilis females x imeretensis males, three fractions of β-esterase were determined - both parental and hybrid forms. The quantitative distribution of the enzyme activity between these fractions was dependent upon the proteins which were synthesized by the 4th chromosome of imeretensis.

The special slow esterase (S-esterase) was observed in many stocks of imeretensis. This S-esterase has some properties of β-esterase. The activity of the S-esterase was suppressed in interspecific hybrid virilis females x imeretensis males. It is interesting that some stocks of imeretensis which differed in their behavior are characterized by the presence or absence of S-esterase on the electropherograms.

Acknowledgement: The authors are very grateful to Prof. N. Sokoloff and Dr. M. Eugeniev for providing them with D. virilis and D. imerentensis stocks.

Loukas, M. and C.B. Krimbas. Agricultural College of Athens, Votanicos, Greece.
Some methods of detection of certain enzymes in D. subobscura.

Horizontal gel electrophoresis and connaught starch were used in all the following methods.

One gene of phosphoroglucomutase (Pgm) was detected in adults and pupae: Homogenisation in gel buffer. Tank's buffer: 0.1 M TRIS, 0.1 M Maleic acid, 0.01 M EDTA, 0.01 M $MgCl_2$ brought to pH 7.6 by 4N sodium hydroxide. Gel buffer: a dilution 1 to 10 of Tank's buffer. Staining solution: 0.25 mg/ml glucose-1-phosphate, 5mM $MgCl_2$, 0.25 mg/ml NADP, 1 EU/ml G-6-PD, 0.25 mg/ml NBT and 0.05 mg/ml PMS were added in 0.03 M TRIS-HCl buffer, pH 8.0. Four alleles (1.62, 1.00, 0.65, 0.39) were detected. Pgm was found to be located on the J chromosome with the help of chromosomal inversions as markers.

One gene of isocitric dehydrogenase (IDH) was detected mainly in adults. Buffers the same as for Pgm. Staining solution: 0.40 mg/ml isocitric acid trisodium salt, 5 mM $MnCl_2$, 0.25 mg/ml NADP, 0.25 mg/ml NBT and 0.05 mg/ml PMS were added in 0.2 M TRIS-HCl buffer, pH 8.0. Two alleles (1.04, 1.00) were detected.

Two genes of adenylate Kinase (AK) were detected in adults and pupae. Gel buffer: 0.05 M TRIS, 0.003 M EDTA brought to pH 8.5 by IN HCl. Tank's buffer: 0.05 M TRIS-HCl, pH 8.5. Staining solution: 5 mg/ml glucose, 5 mM $MgCl_2$, 0.30 mg/ml ADP, 0.25 mg/ml NADP, 1 EU/ml G-6-PD, 0.20 mg/ml Hk, 0.25 mg/ml NBT and 0.05 mg/ml PMS were added in 0.1 M TRIS-HCl buffer, pH 8.0.

One gene of α-glycerophosphate dehydrogenase (aGPD) was detected mainly in adults. Buffers the same as for Pgm. Staining solution: 0.25 mg/ml NAD, 0.6 mg/ml α-glycerophosphate 0.25 mg/ml NBT and 0.05 mg/ml PMS were added in 0.1 M TRIS-HCl buffer, pH 8.5.

Two genes of sorbitol dehydrogenase were detected with the same buffers as for Pgm. Sorb.DH-I was detected in all stages while sorb.DH-II mainly in adults. Staining solution: 5 mg/ml D-sorbitol, 0.25 mg/ml NAD, 0.25 mg/ml NBT and 0.05 mg/ml PMS were added in 0.05 M TRIS-HCl buffer, pH 8.0.

With these genes and others located previously and mapped (Loukas, et al. in press in Genetics) the following genes are available in D. subobscura.

Chromosome O: Est-6, Est-8, ODH, XDH, ME, AO, Lap
Chromosome J: Est-7, Est-3, Aph, Pgm
Chromosome U: ADH, MDH
Chromosome E: Est-9, Hk-1
Unlocated : Pt-7, Pt-8, Pt-10, Est-4c, Hk-II, aGPD, IDH, Ak-I, Ak-II, Sorb.DH-I, Sorb.DH-II

Aronshtam, A., E. Karakin, L. Korochkin and S. Svididov. Institute of Cytology and Genetics, Novosibirsk, USSR. The simple method of preparation of the antiserum against esterase-6 of D. melanogaster.

It was shown (Aronshtam et al., 1973), that the activity of esterase-6 in ductus ejaculatorius (d.e.) of males of D. melanogaster is very high. We investigated electropherograms of proteins of d.e. in males heterozygous for alleles of the gene Est-6. Two intensively staining protein fractions can be seen in region of esterase activity (Figure 1a,b). These fractions were used for preparation of the corresponding antiserum against esterase-6 of D. melanogaster. We prepared the extract from 200 d.e. of males homozygous on "S" and "F" alleles of gene Est-6, then carried out the disk electrophoresis in 5.5% polyacrilamide gel. After electrophoresis we cut out the regions of localizations of

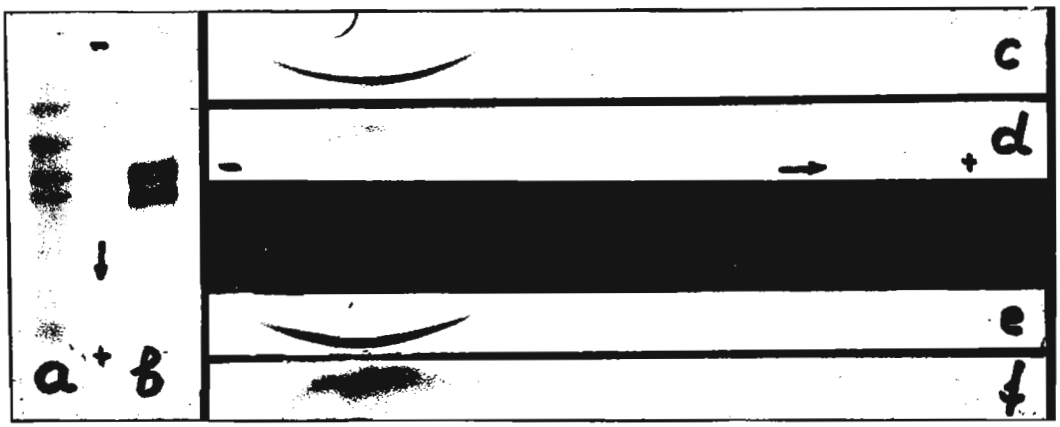


Figure 1a. The electropherogram of protein extracts from 200 d.e. of male D. melanogaster - heterozygous on locus Est-6 (amidoschwarz); b. zymograms of esterase-6 from d.e. of male heterozygous on Est-6; c. immunoelectropherogram of proteins from 100 d.e. of male D. melanogaster in agarose gel; d. immunoelectropherogram of extracts from 100 flies; e. histochemical reaction on β-esterase on immunoelectropherogram "c"; f. histochemical reaction on β-esterase on immunoelectropherogram "d".

allozymes of esterase-6. Five "disks" were macerated in deionized water and injected subcutaneously into rabbits four times with weekly intervals, then one a month later (Fritz et al., 1969). Two zones of precipitations were observed after immunoelectrophoresis of extracts of d.e. and flies of D. melanogaster, one of them was "esterase-positive" (Figure 1c,d,e,f). We can explain the existence of two zones of precipitation by the following situations: 1. We injected into rabbits two antigens with identical electrophoretical mobility and partly immunochemical identity; or 2. there is a cross reaction of our antiserum to another antigen without esterase activity. It is not clear whether or not the prepared antiserum is monospecific. But this antiserum can be used in immunochemical analysis of esterase-6 of D. melanogaster.

References: Aronshtam, A., B. Kuzin and L. Korochkin 1973, Isozyme Bull. 6:24; Fritz, P., E. Vessel, E. White and K. Pruitt 1969, Proc. Natl. Acad. Sci. 62:558.

Roberts, P.A. Oregon State University, Corvallis. The end of "terminal" deficiencies in certain wild-type strains.

Both Oregon-R and Oregon-R-C strains of D. melanogaster were described by Bridges in the 1930's as homozygous for a terminal deficiency of the right arm of chromosome 2, Df(2R)60F2-3. We have observed two faint bands distal to 60F3 in the Ore-R-C strain obtained from Cal. Tech. and bands beyond 60F5 in the Ore-R-C strain obtained from Bowling Green. These strains should no longer be considered as carrying terminal deficiencies (Genetics 77:s54); instead, the strain differences are apparently due to rapid evolution of chromomeric patterns near telomeres.

Sreerama Reddy, G. and N.B. Krishnamurthy. University of Mysore, Manasa Gangotri, India. Altitudinal gradients in the frequencies of three common inversions in Drosophila ananassae.

There is an abundant evidence to show that chromosomal polymorphism in Drosophila reflects the environmental diversity in both experimental and natural populations. Since the adaptive values of different genomes differ greatly, it appears natural that as the environment changes, the fitness of some gene arrangements either improve or deteriorate in relation to that of others in the same population.

D. ananassae exhibits a remarkably high degree of chromosomal polymorphism in its natural populations (c.f. Sreerama Reddy, 1973). It harbours a large number of paracentric inversions, some pericentric inversions and translocations. Of the several paracentric inversions known only three inversions (2LA, 3LA, 3RA) have been shown to be coextensive with the species, while the remaining are endemic. Freire-Maia (1955) has pointed out that in spite of the existence of such a vast array of chromosomal variability in this species, it has not been possible to correlate it with ecological factors. The present work was undertaken to investigate the impact of variable altitudes on the polymorphic system of ananassae.

Five populations collected at different altitudes of Nilgiri range have been used in the present study. Of these two populations collected from Gundlupet (720 m) and Gudalur (900 m) came from the western side of the hill and two populations collected from Metupalyam (390 m) and Merupalyam (1200 m) occupy the eastern side of the hill, while the population from Naduvattam (1840 m) collected at the summit of the hill is common to both sides. Since two sides of the hill differ ecologically, making the populations distinct, they have been treated separately in two groups - Western range and Eastern range.

The natural populations collected at different altitudes of the two sides of the same hill greatly vary in terms of frequencies of three common inversions (2LA, 3LA, 3RA) which constitute the polymorphic pattern of this species (Tables 1 and 2). The other gene arrangements found in the populations under study include 4 paracentric inversions (XRA, XRB, XLb, 2LA+2Le), a pericentric inversion $(2L\text{-}2R)_9$ and a translocation $(2L\text{-}3L)_8$ manifested in a very

Table 1. Widespread inversions and their observed and expected frequencies with chi-square and P. values for the populations of D. ananassae at different altitudes of Nilgiri hills (Western range).

Locality	Altitude in meters		Heterozygous inversions 2LA	3LA	3RA
Naduvattam	1840	Observed	21	31	21
		Expected	26.73	29.30	16.93
Gudalur	900	Observed	35	35	30
		Expected	36.61	40.14	23.23
Gundlupet	720	Observed	48	48	15
		Expected	40.64	44.55	25.79

$\chi^2 = 11.07$ df = (R-1) (C-1) = 4 P = 0.05 - 0.025

Table 2. Widespread inversions and their observed and expected frequencies with chi-square and P. values for the populations of D. ananassae at different altitudes of Nilgiri hills (Eastern range).

Locality	Altitude in meters		Heterozygous inversions 2LA	3LA	3RA
Naduvattam	1840	Observed	21	31	21
		Expected	20.00	37.32	25.24
Merupalyam	1200	Observed	35	47	45
		Expected	34.86	46.84	45.65
Metupalyam	390	Observed	28	34	44
		Expected	29.00	38.79	38.10

$\chi^2 = 3.74$ df = (R-1) (C-1) = 4 P = 0.5 - 0.25

low frequencies of 1 to 5% in one or two populations and hence not considered for statistical computations. Perusal of the Tables 1 and 2 indicate that the frequencies of 2LA and 3LA decrease with the increasing altitude in the Western range but not so in the populations of the Eastern range. The degree of polymorphism as measured by the mean number of inversions per larva and frequency of heterokaryotypes is found to be more at lower elevations than at higher elevations. The percentage of heterokaryotypes at the higher elevation is found to be only 58% with only 0.78 mean inversions per larva, while a higher grade of polymorphism with 69 to 79% of heterokaryotypes having 1.02 to 1.32 mean inversions per larva is noticed at the lower and intermediate elevations.

The results of the χ^2 homogeneity test indicate that the differences noted in the populations of Western range are significant while in those of the Eastern range are not significant.

Altitude, one of the ecological factors may influence the genetic structure of natural populations. The differential response of different inversions to varied altitides has been documented (Dobzhansky, 1951; Brncic, 1970). In our observation, the significant differences in the chromosomal constitutions between different altitudes has been noted only in the Western range of Nilgiri hills but not in the Eastern range. The two sides of the hill may differ ecologically making the populations to be distinct. Similarly Dobzhansky (1951) has found that Sierra Nevada Mountain Range forms a racial boundary between the populations on either side of it. The population of D. ananassae at higher altitude has low frequency of 2LA and 3LA inversions and correspondingly reduced mean inversion heterozygotes per larva. Similarly the impact of variable altitudes on different inversions has been shown in D. pseudoobscura. 3RA inversion of D. ananassae is less variable and on par with CH inversion of D. pseudoobscura. The dissimilarity in the response of inversions to altitides on either side of the hill can be accounted by the suggestion of Dobzhansky (Cf. Levine, 1969) that there is nothing inherent in a particular gene arrangement that adopts its carrier for a particular environment. It is possible that altitude and other ambient ecological factors may regulate the inversion frequencies in D. ananassae populations.

Acknowledgments: We are highly indebted to Dr. M.R. Rajasekarasetty, Professor and Head of the Department of Post-Graduate Studies and Research in Zoology, University of Mysore, Manasa Gangotri, Mysore, for his constant encouragement and valuable suggestions.

References: Brncic, D. 1970, In Essays in Evolution and Genetics, M.K. Hecht and W.C. Steere, eds, 381-399; Dobzhansky, Th. 1951, Genetics and the Origin of Species. Col. Univ. Press, N.Y.; Freire-Maia, N. 1955, DIS 29:116-117; Levine, L. 1969, Biology of the Gene. Toppan Print. Co. Ltd., Tokyo, Japan; Sreerama Reddy, G. 1973, Doc. Disser.

Miller, S., E. Berger and R. Pearcy. SUNY at Albany, New York. The temperature dependence of Km for α-GDH allozymes.

The gene-enzyme system α-glycerophosphate dehydrogenase is polymorphic in D. melanogaster populations (O'Brien and MacIntyre 1969) and shows both temporal fluctuations (Berger 1971) and spatial clines (Johnson and Schaffer 1973) in allele frequency. In general, the rarer slow allele is found at high frequency in northern latitudes and during the cooler autumn months. These patterns suggested that an analysis of enzyme activity as a function of temperature could provide a clue concerning the adaptive function of variation at this metabolically crucial enzyme locus (O'Brien and MacIntyre (1972).

Studies on pH optima or denaturation by heat revealed no important difference between semi-purified enzyme extracts from the three genotypes. Km determinations for the in vivo substrate, dihydrooxyacetone phosphate, as a function of temperature did, however, uncover clear-cut differences. Under conditions of saturating but not inhibitory cofactor concentrations (120 μM NADH) we found that at low temperatures (5^{o}-15^{o}C) the FS and SS allozymes showed a greater affinity for DHAP, that is lower Km values, than did the FF enzyme (Figure 1). At higher temperatures (>20^{o}C) the order of increasing Km was FF > SS > Fs. At about 18^{o}C the three plots intersect. At low temperatures, the FF enzyme had lower Km values at all temperatures (Figure 2), while the FS and SS patterns remained essentially identical to those in

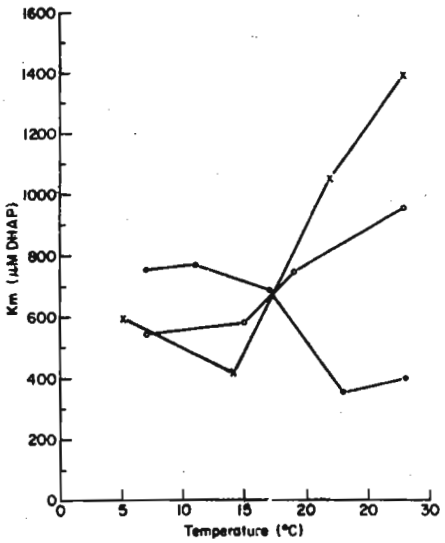


Figure 1. Temperature dependence of Km µM DHAP) determined from extracts of α-glycerophosphate dehydrogenase genotypes: FF (•), FS (x), and SS (o). Cofactor (NADH) concentration is 120 µM.

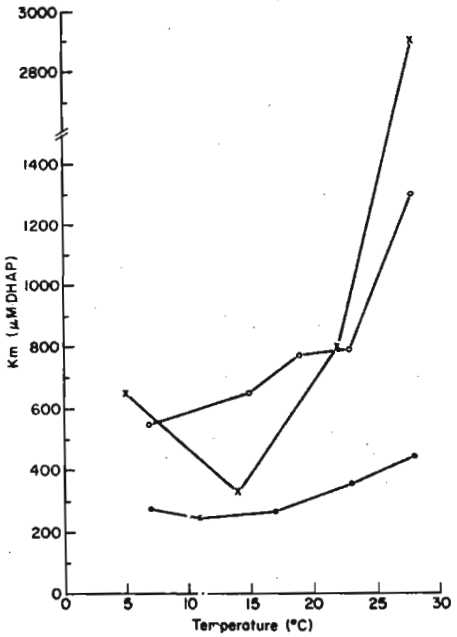


Figure 2. Temperature dependence of Km (µM DHAP) determined from extracts of α-glycerophosphate dehydrogenase genotypes: FF (•), FS (x), and SS (o). Cofactor concentration is 160 µM.

Figure 1.

If substrate affinity is an important enzyme property in nature, then the results do indicate substantial differences between the fitnesses of additional data on such properties as activation energy and Q_{10} need also be obtained. In addition some consideration of the distinct but isoallelic "larval" enzyme (Bewley, et al. 1974) must be made, since the function served by this enzyme in larval lipid metabolism is entirely distinct from the "flying" function of adult α-GDH.

Research supported by NIH (EB) and NSF (RP) grants.

References: Berger, E. 1971, Genetics 67:121-136; Bewley, G., J. Rawls and J. Lucchesi 1972 J. Insect Physiol. 20:153-165; Johnson, G. and H. Schaffer 1973, Biochem. Genet. 10:149-162; O'Brien, S. and R. MacIntyre 1969, Amer. Nat. 103:97-113; O'Brien, S. and R. MacIntyre 1972, Biochem. Genet. 7:141-161.

Gerdes, R.A. and E.D. Doyle. Texas Woman's University, Denton. Profile of lactic dehydrogenase and alpha-glycerophosphate dehydrogenese in D. melanogaster.

The enzyme profile reported by Imberski and Strommen (1972) stimulated a series of experiments to determine what, if any, differences are present in several enzyme profiles in different strains of D. melanogaster. The strains selected were Oregon-R and vestigial, both stocks were derived from Cal Tech stocks. The two strains were selected because of the developmental differences manifested in the two phenotypes. Cultures were started on sucrose-yeast media with a two hour egg laying period from 3-4 week adults and the progeny were harvested at the appropriate times. The procedure was a modification of Duke and Pantelouris (1963). The enzyme assay was the microelectrophonetic procedure of Pasteur (1972) as modified for a Joyce and Loebl apparatus.

The enzymes lactic dehydrogenase and α-glycerophosphate dehydrogenase were assigned to reflect differential gene action during the life of an organism. LDH and α-GPDH function by

supplying energy in different stages of the life cycle. Only one metabolic role has been identified for LDH while α-GPDH functions in a dual role to provide energy and to supply precursors for lipid biosynthesis.

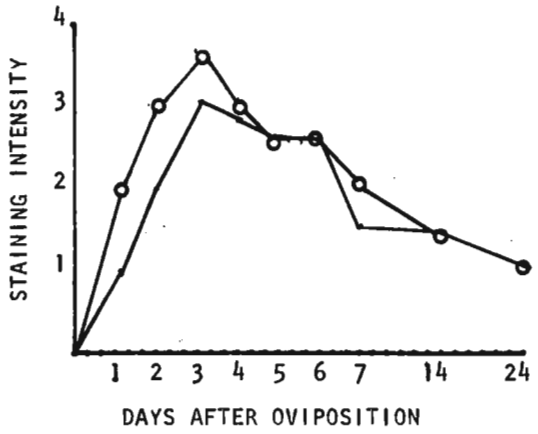


Figure 1. Staining intensity of lactic dehydrogenase in different developmental stages of Oregon-R & vestigial strains

. Oregon-R
O vestigial

Two bands of LDH activity appeared at different stages of development. One LDH band is thought to function in larval muscles and is most concentrated in the third instar stage. During pupation the level of LDH decreases and remains low throughout the life cycle. A second band which exhibits faint activity in the larval stages increases slightly in intensity in the adult

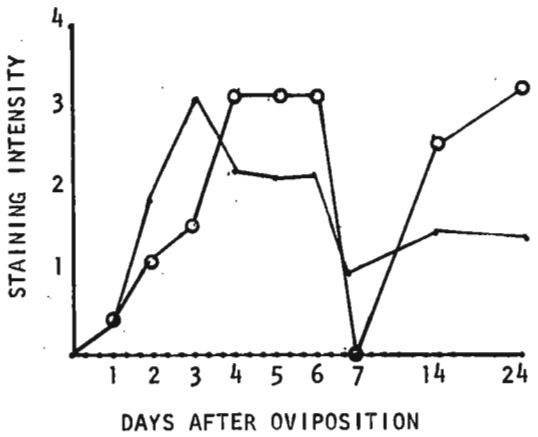


Figure 2. Staining intensity of α-glycerophosphate dehydrogenase in different developmental stages of Oregon-R & vestigial strains

. Oregon-R
O vestigial

stages. This band may be associated with energy formation in brain and nerve tissue. Figure 1 shows little difference between the enzyme profile of Oregon-R and vestigial; there is a slight increase in LDH in the early stages of vestigial development.

α-GDPH activity is present in the second and third instar when the organism is known to be storing lipids in the fat body as a source of energy during the pupal period. The activity of α-GDPH in the adult is greater than the activity of LDH in these same stages. Figure 2 shows considerable differences to be present in the profiles of α-GDPH in the development of Oregon-R and vestigial. The relation of these profiles to morphogenetic changes await additional study.

References: Duke, E.J. and E.M. Pantelouris 1963, Comp. Biochem. Physiol. 10:351-355; Imberski, R.B. and C. Strommen 1972, DIS 48:74; Pasteur, N.M. 1972, Ph.D. Disser. Univ. of Texas Southwestern Med. School, Dallas.

Chihara, C.J.[1] and J.W. Fristrom[2]. University of California, Berkeley. ([1]Dept. of Molecular Biology; [2]Dept. of Genetics). A juvenile hormone activity from extracts of D. melanogaster.

The lack of "true" juvenilizing effect of the known juvenile hormones on the larvae of Dipteran insects has prompted the search for a Dipteran JH. Since larval extracts of Drosophila showed no JH activity, we turned to a mutant whose phenotype included an hypertrophied corpora allata in the adult female, due to an abnormal ovary. The mutant is the female sterile, fes(2)B described in detail by King et al. (1966). On the rationale that the fes homozygote might be sequestering JH in the

hypertrophied corpora allata (+/+ ovaries can restore normal size) we made an extract of these females. Flies were ether extracted, methanol and ethanol extracted, and cold precipitated, after Meyer et al. (1970). The supernatant of the cold precipitation was put through a Sephadex LH 20 column 2.5 x 26.2 cm in 95% ethanol. Void volume was 40 to 60 ml. Elution of the extract was followed by spotting 10 lambda aliquots of

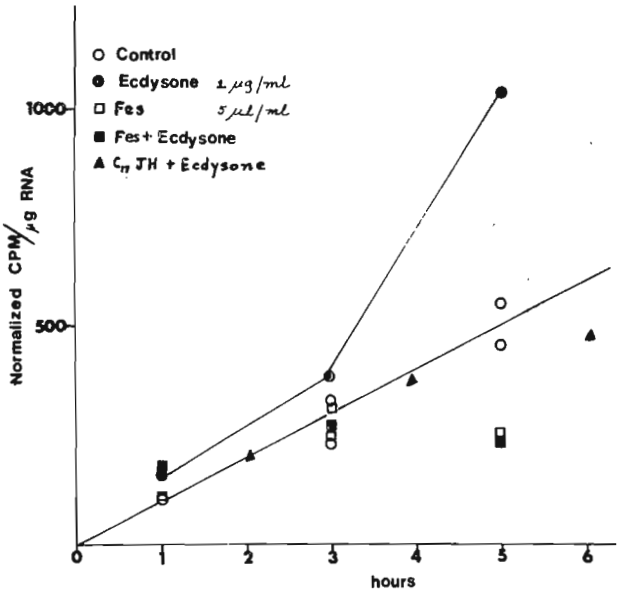


Figure 1: Cumulative incorporation of ^{3}H-uridine into acid insoluble counts in the presence of fes extract or C_{17} JH with or without β-ecdysone. Control has only ethanol.

each fraction on thin layer chromatography paper and placing it in an iodine chamber to develop color. Thin layer chromatography was run in 15% ethyl acetate in Hexane (freshly distilled) and eluted in freshly distilled tetrahydrofuran. Gas chromotography was done with a glass column packed with PDEAS 1.85% on a 3% silonized absorbant, Chromosorb,Q. The evagination-inhibition assay we used is described elsewhere (Chihara et al. 1972), as are the pro-

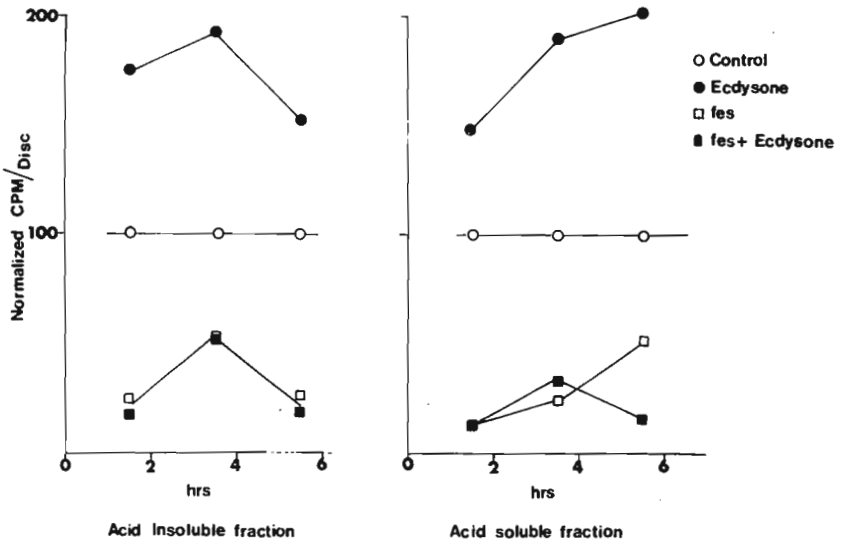


Figure 2: Pattern of incorporation of counts from ^{3}H-uridine into RNA and acid soluble material of discs treated with fes extract. "Pulse" labelled for 1/2 hour.

cedures used for precursor uptake into RNA (Raikow and Fristrom, 1971).

The original crude extract was made from fes homozygous females alone, and 10 grams of flies yielded 0.5g of crude oil. This oil was active in inhibiting the ecdysone induced evagination of imaginal discs at 2-5 µl/ml. This evagination-inhibition assay was used throughout the procedure to follow activity. A comparable inhibition of evagination is induced by 40 to 60 µg/ml of Cecropia JH (Chihara and Fristrom, 1973). In order to increase the bulk of the starting material, the entire F_1 of the starting cross was homogenized (including males and females and all heterozygotes). The resultant crude oil was active at 5 to 10 µl/ml. +/+ extracts treated the same way were inactive at up to 100 µl/ml. Thin layer chromatography of the cold methanol supernatant yielded seven bands, and the activity was present only in band 4, which was cut from an R_f of 0.25 to 0.4. Cecropia C_{17} JH had an R_f of 0.4 on the same plate. +/+ extracts cut at the same place had no activity. The active fraction was eluted from the LH 20 column at about 130 to 140 mls, at about the same position as riboflavin. Again, +/+ parallels were inactive.

It has been previously demonstrated that JH effectively inhibits the β-ecdysone induced

increase in RNA synthesis as measured by incorporation of uridine into acid insoluble material. (Chihara et al., 1972; Chihara and Fristrom, 1973). The crude fes and +/+ extracts were tested for their ability to inhibit this increased RNA synthesis. The +/+ extract had no effect up to 100 µl/ml on uptake or incorporation of uridine into RNA. The effect of the fes extract can be seen in Figure 1 compared to the effect of the C_{17} JH of Cecropia. Both the extract and the hormone decrease the β-ecdysone induced RNA synthesis back down to control levels. The leveling off of the incorporation seen in Figure 1 seems to be due to a decrease in the uptake of precursor induced by the extract. Figure 2 shows the results of uridine uptake, measured in one hour pulses, into both RNA and acid soluble material. The same pattern is seen as in the continuous labelling experiments and it is possible that the decrease of incorporation in fes treated discs is due to a decrease in uptake of precursor in the presence of the crude extract. Thus, in so far as there is no difference in incorporation of uridine into RNA by discs treated with fes extract and with fes extract + β-ecdysone, the discs respond to the extract in a way similar to their response to Cecropia JH.

Both the crude extracts and partially purified (about 8-10 peaks on GLC) extract were tested on the *galleria wax test, on a musca, and on a mosquito assay for JH. All of these tests were essentially negative. These results might be explained by the presence of inhibitors of JH in the extract which are only active in vivo. We believe that the in vitro assay on Drosophila imaginal discs, while not very sensitive, is a true indication of juvenile hormone activity on Drosophila and that therefore the extracts do indeed contain a fair amount of Drosophila juvenile hormone which, given proper facilities ought to be extractable and purifiable.

* We would like to thank Dr. J. Siddall of Zoecon for the in vivo assays.

References: Chihara, C. and Fristrom, J. 1973, Dev. Biol. 35:36; Chihara, C., Petri, W. and King, D. 1972, J. Insect Physiol. 18:1115; King, R.C., Aggarwal, S. and Bodenstein, D. 1966, J.E.Z. 161:151; Meyer, A., Hanzmann, E., Schneiderman, H., Gilbert, L. and Boyette, M. 1970, Arch. Biochem. Biophys. 137:190; Raikow, R. and Fristrom, J. 1971, J. Insect Physiol. 17:1599.

This work was supported in part by a Research Grant (USPHS GM-19937) to JWF and a predoctoral Training Grant to CJC (USPHS GM-00-367).

MATERIALS REQUESTED OR AVAILABLE II

H.J. Muller original reprints are no longer available. For xerox copies write to: Ms. Department, Lilly Library, Indiana University, Bloomington, Indiana 47401. Charges: ten cents per page for xeroxing, plus one dollar for handling and postage. Please order by title and year of publication. A List Of Works, 372 titles, is still available free.

Max Levitan, New York, would be very happy to receive adult Drosophila robusta of both sexes collected anywhere in its range, for chromosomal analysis. Kindly report date collected and locality.

Marvin Seiger has been appointed extraordinary professor of biology at Universidad Nacional, Heredia, Costa Rica. He would be grateful to receive genetics reprints and biology texts, especially in genetics, for the library. These may be sent to him in Costa Rica, or to Mr. Gerardo Lara, 1724 Westwind Way, Maclean, Virginia 22101.

F.M. Johnson, Senior Geneticist with the Research Triangle Institute, reports that the Institute is forming a research group to work in the area of population-biochemical genetics and to include mutagenesis testing with mice and Drosophila. With respect to this area of research, the Institute library is presently somewhat lacking in reference material, and a supplement in the form of a reprint collection would be most helpful. Therefore they would appreciate receiving all possible reprints in this area. (Box 12194, Research Triangle Park North Carolina 27709.)

A new Drosophila genetics laboratory has been established at the Universidad Autónoma Agraria "Antonio Narro", Buenavista, Saltillo, Coahuila, México. José Espinoza-Velázquez, former student of the genetics department of Postgraduate College at Chapingo, México, would be very grateful to receive appropriate reprints to start a laboratory library.

Fuyama, Y. Tokyo Metropolitan University, Japan. A handy olfactometer for Drosophila.

In order to assay the behavioral responses of adult Drosophila to odoriferous substances a simple olfactometer was devised. As Figure 1 shows, the olfactometer consists of three major parts, i.e., a starting tube (A: 30 mm in diameter and 70 mm in length), a choice tube (B: 30 mm in diameter and 70 mm in length) and two trap tubes (C: 25 mm in diameter and 90 mm in length), all of which are made of Pyrex glass. The upper end of the starting tube is covered with fine nylon mesh. Two connecting arms (D: 7mm diameter glass tube) which are welded to the bottom of the choice tube bend at right angles and tightly bore through #8 silicone stoppers (E). The end opening of the connecting arm protruding about 5 mm from the stopper effectively prevents the flies once trapped from going out. The larger opening of the trap tube fits the #8 stopper attached to the connecting arm, and another opening is made to take a test-tube stopper (F), through which an injector needle (G: 0.80 x 38 mm disposable needle) connected to the outlet of air delivery system pierces to provide air flow into the trap.

Just prior to assembling the olfactometer, a piece of filter paper (H: 60 x 30 mm) is inserted into each trap. Then, 0.5 ml of the odorant solution to be tested is poured onto the filter paper in one trap tube (Odor trap), whereas the same amount of distilled water (or any other solvent for the odorant) is applied to another trap (Control trap).

The air delivery system is not shown in the figure, but any device that can supply the clean air of constant humidity at the fixed rate may be used. In our experiments, air was provided into each trap at the rate of 5 ml per minute.

The olfactometry was carried out as follows: Young adult flies were deprived of food and water for some 24 hours prior to the experiment. Eighty starved flies of a given sex were

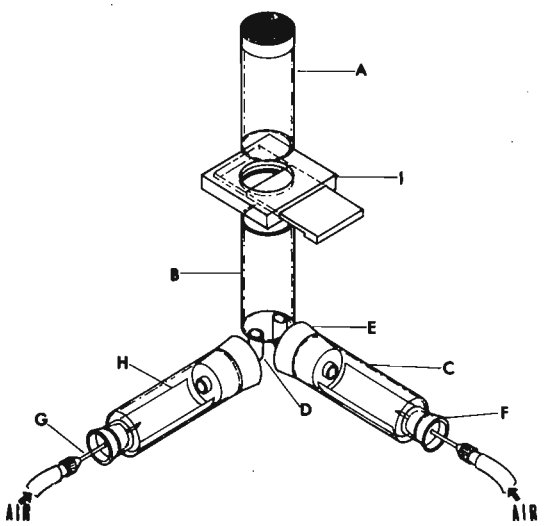


Figure 1. Diagram of the olfactometer

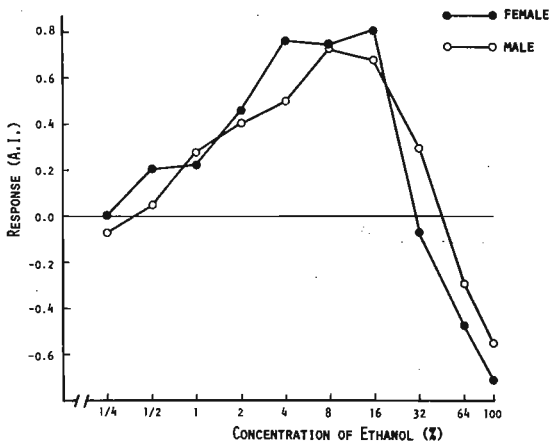


Figure 2. Response of D. melanogaster to ethanol

confined in the starting tube using a sliding shutter device (I). After flowing 25 ml of air through each trap, the shutter was opened and the flies were allowed to run freely for 30 minutes. At the end of the free running period, the number of flies in each trap was counted. The experimental methods mentioned above as well as the size of the apparatus, of course, may be altered according to the purpose of experiment or the species to be used.

The responses of flies were evaluated by an index designated as "Attractability Index" (A.I.) which was calculated as follows:

A.I. = (Number of flies in the Odor trap - Number of flies in the Control trap)/(Number of flies started)

Thus, the index varies between -1 (extreme repellency) and +1 (extreme attractancy). As an example, Figure 2 shows the responses of a wild strain of D. melanogaster to various concentrations of ethanol which is known as a potent attractant to this species. Each point in the

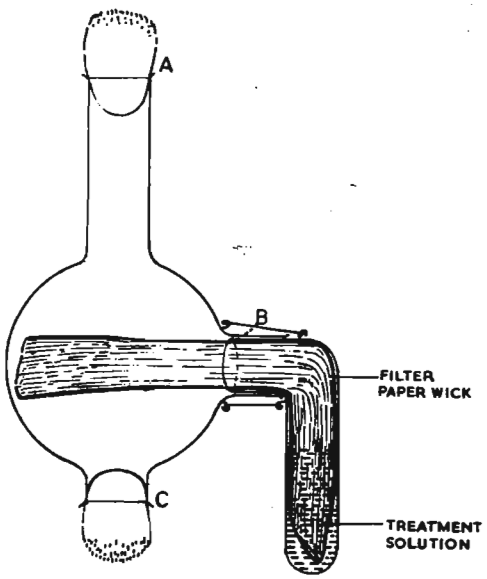
figure represents the average of four replicate experiments.

The present apparatus resembles the well-known McIndoo type olfactometer (McIndoo, 1926) in shape, but it differs in that it uses no auxiliary stimuli to facilitate the flies to move towards the traps. As the choice tube stands vertically, the flies in it tend to go away from the odor source because of their negative geotaxis, and this excludes the flies from entering the traps only by chance. In this sense, it is essentially the same as the elaborate olfactometer developed by Kikuchi (1973), however is much simpler in structure and less expensive. It takes no more than a few minutes to prepare the apparatus for each run, and the odor contamination from previous experiments can be removed merely by replacing the contaminated apparatus with another clean set. Thus, the use of several spare sets facilitates the prompt repetition of the experiment.

References: Kikuchi, T. 1973, Japan. J. Genet. 48:105-118; McIndoo, N.E. 1926, J. Econ. Entomol. 19:545-571.

Sharma, R.P. Indian Agricultural Research Institute, New Delhi. A new feeding chamber for mutagenizing Drosophila flies.

A simple and effective method of feeding chemical mutagen to Drosophila flies was proposed by Lewis and Bacher (DIS 43:192, 1968). This method has since been employed successfully by many workers. While making use of this method, we noticed that many times treatments of long duration lead to excessive killing of the flies due to drying off of the filter paper. Likewise, flies get drowned if filter paper discs have excessive moisture. If the strain to be mutagenized happens to be hyperactive, when flies have a tendency to fall and hit the base of the container, recovery of surviving males after treatment is very low. Also if sequential treatments with more than one mutagen is desired, this method of treatment is not very convenient.



We have designed and successfully employed a simple apparatus for mutagenization of Drosophila by feeding (Figure 1). It consists of (1) a 150 ml round bottomed flask with a neck opening (A). It is fitted, on one side, with a female part of ground glass joint (B) and at the bottom with glass tube of one cm length and 1.5 cm diameter. (2) a 9 cm long and 1.5 cm diameter glass test tube, of which about 3 cm length is bent at right angle and ground to fit into the opening (B) on the flask, (3) a circular filter paper wick of desired length with one end flattened and closed.

Mutagen solution of desired strength is filled in the side tube. A filter paper wick of appropriate dimensions is then inserted into the side tube in such a way that its circular end remains immersed in the solution while the flattened end is inserted into the flask through the opening (B). The length of the wick in the flask is so adjusted that it provides maximum area for the flies to feed on but in no case should touch the flask surface. Side tube is then firmly secured with the help of rubber bands hooked on to the flask. Flies to be mutagenized are released into the flask through the opening (A). Treatment can be terminated by carefully removing the side arm along with the filter paper wick. Mutagenized flies can be etherized either in the chamber itself or removed directly into etherizer.

This apparatus has also been used by us for aging the flies. Flies are released into the flask. Side tube, filled up to the brim with standard Drosophila medium, is attached to the flask. At fixed intervals, fresh medium can be supplied by replacing old tube with new one without agitating the flies. Dead flies fall to the bottom and can be easily collected through opening (C).

Mayfield, J.E. and J.R. Ellison. Carnegie-Mellon University, Pittsburgh, Pennsylvania. An improved method for isolating DNA from adult Drosophila.

Adult Drosophila are a particularly difficult subject from which to isolate DNA in good yield. The usual techniques usually produce low yields of badly nicked DNA which is deficient in the A-T rich satellites. The present method was devised to alleviate these problems.

Adult flies are frozen and weighed. Then two parts of a solution of 6 M guanidine HCl, 1% Triton X-100, and .1 M 2-Mercaptoethanol is added to one part of flies (i.e. 20 ml of guanidine HCl per 10 gm of flies). The mixture is then ground extensively with mortar and pestle. The resulting slurry is centrifuged at 12,000xg for 10 minutes. The resulting pellet and pellicle are removed and ground again with an additional 1 part (i.e. 10 ml/original 10 gm of flies) of 6 M guanidine HCl, 1% Triton X-100, and .1 M 2-Mercaptoethanol. The grinding and centrifuging are repeated 3 or 4 more times. The supernatants are filtered through cheesecloth and precipitated at room temperature by the addition of an equal volume of ethanol. The ethanol precipitates are collected by centrifugation at 12,000xg for 10 minutes. The pellets are usually purple and contain the DNA. The combined ethanol pellets are redissolved in 4 M guanidine HCl, 1% Triton X-100 and .1 M 2-Mercaptoethanol and reprecipitated from 50% ethanol. This pellet is dissolved overnight in 0.1 M pH 10.5 glycine buffer which is 2% in sodium sarkosyl. This solution is then adjusted to a density of 1.55 by the addition of .97 gm of CsCl/ml of dissolved pellet and the further addition of one twentieth volume of a 10 mg/ml solution of ethidium Br in water. The final volume should be about 18 ml/10 gm of flies. This solution is then centrifuged at 40,000xg for 48 hours in a Beckman Ti-50 rotor. As described by Firtel (Firtel and Bonner, 1972), the DNA bands free of RNA which pellets, free of polysaccharide which bands at a greater density than DNA, and free of protein which floats. The red DNA is removed with a pasteur pipet and rebanded in CsCl as before. The ethidium bromide is removed by diluting the DNA solution with three volumes of water and extracting it twice with an equal volume of isopentyl alcohol. Two volumes of 2-ethoxyethanol are then added and the DNA wound out of solution. The precipitated DNA should be soaked briefly in 95% ethanol before dissolving in the desired buffer.

The final DNA has no detectable proteins or polysaccharide contamination and is of high molecular weight. The method has been used with equal success on D. melanogaster, D. hydei, and D. virilis. Contaminating DNA from the gut flora is probably not a problem since DNA is not extracted from isolated yeast or bacteria by 6 M guanidine HCl. The approximate total yield of DNA is 100 micrograms per gram of adult D. melanogaster and about twice as much from D. hydei and D. virilis.

Reference: Firtel, R.A. and J. Bonner 1972, J. Mol. Biol. 66:339.

Binnard, R. and P.R. Lundgren. Ames Research Center, NASA, Moffett Field, California. Procedure for holding D. melanogaster imagoes in a standing position for exposure to particle radiation.

Irradiation of Drosophila melanogaster (Oregon R) with argon particles at the heavy ion linear accelerator of the University of California, at Berkeley, created the need for a nontoxic adhesive that would hold the flies in a standing position for about 1 hour. A very satisfactory solution was found in a mixture of two-thirds Mochi flour (an oriental rice flour) and one-third corn starch. Just before use, the dry ingredients are mixed and moistened with water to the consistency of a thick cream. Small dots are applied to the holding material with a fine brush, and the anesthetized flies are "perched" in the drop so that the paste adheres to the feet and legs. The paste dries in a few minutes and holds the fly in place. After irradiation, the fly, with the paste still attached to the legs, is carefully picked up with forceps and deposited on the cellucotton of the food vial. The vial usually contains enough moisture to soften and crack the paste, so that the fly can step out upon recovery from the anesthesia. Alternatively, the paste particle can be moistened with water, which results in immediate disintegration of the adhesive.

For the argon irradiation, exposure for 5 to 7 minutes to 0.5 ml metofane in a 1/2 pint anesthetizing bottle immobilized the flies for approximately 1 hour.

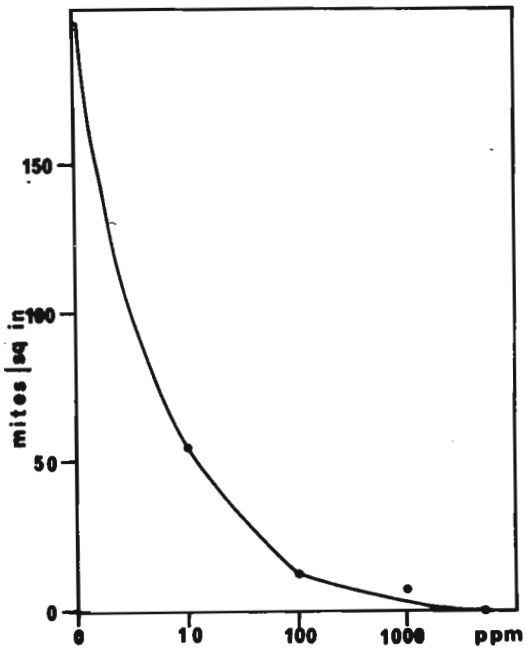
The flies were pasted directly on a lexan dosimeter in a clockface pattern, which allowed precise identification of the individual flies and recording of the dose received by each fly.

A total of 280 Drosophila were anesthesized and pasted without a single instance of death or injury.

Ashburner, M. and T. Littlewood, University of Cambridge, England. Tedion - an effective control of anoetid mites.

In 1973 this laboratory was afflicted by an unusually bad infestation of mites. Two groups of mite were present in our cultures, a mesostigmatid mite of the genus Proctolaelaps and two species of anoetid mite, Histiostoma feroniarum (Dufour) and Histiostoma sapromyzarum (Dufour). The mites were identified by Dr. D.A. Griffiths of the Pest Infestation Laboratory, Slough. The Proctolaelaps sp. is especially dangerous since this mite is a predator of Drosophila eggs. The anoetid mites possess a facultative hypopus stage in their development which attaches itself to adult flies often in such numbers as to cause their death.

Mr. Roger Lemon, of the Research Laboratories of Fisons Ltd. at Great Chesterford near Cambridge was kind enough to screen a number of acaricides of low insecticidal activity for use as control agents. At his suggestion we made further tests on a compound called Tedion and the results are satisfactory enough to warrant, we believe, a wider notice. As we will detail below, Tedion is a very effective agent for the control of anoetid



Number of mites per square inch of bottle wall vs. concentration of tedion solution in which bottle papers were soaked.

mites and does not, as far as we can judge, harm Drosophila. Neither it, nor any other acaricide tested was effective against Proctolaelaps sp. although Cantelo and Boswell (DIS 50:163) recommended the use of oxythioquinox (6 methyl-2-oxo-1, 3 dithiolo (4,5,b) quinoxaline) against Proctolaelaps hypudaei (Oudemans).

Tedion, also known as Tetradifon, is the "common name" for 2,4,5,4'-tetrachlorodiphenyl sulphone or, in U.S. usage, S-p-chlorophenyl 2,4,5,trichlorophenylsulfone. Its acaricidal properties were first described by Huisman, H.O. et al (Nature (Lond.) 1955, 176:515) and it was introduced, in 1954, by N.V. Philips-Duphar under the code number V-18 and the trade name 'Tedion-V-18' (British Crop Protection Association, Pesticide Manual 1968). Its solubility in water is 0.02% at 50°C, its vapour pressure is 2.4×10^{-10} mm Hg at 20°C and it is non-corrosive. For mammals its acute oral LD_{50} is 10,000 mg/kg (Rabbits). "Rats fed two months on a diet containing 500 ppm suffered no ill effects and the offspring of rats fed for two months on a diet containing more than 1000 ppm were normal" (loc. cit. p. 411).

For use against anoetid mites we dilute the commercial preparation, obtained from Duphar-Midox, Smardon, Kent, England, (which is an 8% w/v emulsion) to the required final strength in acetone. Either Watmann No. 1 7 cm. diameter filter papers or our normal bottle or vial paper towels are soaked in the solution and then allowed to thoroughly air dry. The papers are then introduced into the cultures or used in place of the regular paper towels.

The graph shows the number of anoetid mites (all stages of development being counted) within a square inch of the bottle surface (using 200 ml. milk bottles and regular medium) immediately above the food in cultures provided with 'tedion papers' from inception. The cultures were mass Canton-S and were three weeks old when scored for mites. It will be observed that while in the control, untreated cultures, very high densities of mites were reached (in excess of 190/square inch) cultures with papers soaked in tedion solutions of 1000 ppm or 5000 ppm were mite free. The few mites in the 1000 ppm cultures were all dead. No discernable effect of the viability, developmental rate or fecundity of Drosophila from cultures treated with 5000 ppm 'tedion papers' could be discerned. A small scale Muller-5 run, of 200 chromosomes, gave no indication that Tedion was mutagenic at 5000 ppm.

We completely eliminated anoetid mites from this laboratory within one generation by

using 5000 ppm 'tedion papers' in all cultures. We are informed of its successful use in at least two other laboratories. Tedion, or any other acaricide, should not be used prophylactically: not only have its long term effects in Drosophila cultures not been studied but Tedion resistant mites would, if we can be forgiven, be tedious. Tedion resistant spider mites are known.

We would welcome any further reports of the use of tedion against mites in fly laboratories.

We are very grateful to Dr. D.A. Griffiths and Mr. Roger Lemon for their gracious and essential help.

Spieth, H.T. University of California, Davis. Rearing method for some fungal feeding Drosophila species.

In their native habitats Drosophila flavopinicola Wheeler, D. pinicola Sturtevant, D. subquinaria Spencer and D. melanderi Sturtevant use various soft bodied fungi as ovipositional and larval substrates. All four species have been difficult to rear and maintain on traditional laboratory media, but I find that females of these species will readily oviposit in the laboratory on decomposing commercial mushrooms and the larvae will undergo normal development. The adults will feed upon decomposing mushroom, but young flies cannot mature and produce ova unless they also have an additional food source such as laboratory media. However, if a chunk of commercial mushroom is inserted into or placed upon the surface of laboratory media, it usually develops a growth of mould on its surface which seriously interferes with oviposition and prevents eggs that are oviposited from hatching.

The following method has proved successful for the rearing of the four species: One quart, or preferably half gallon, self-sealing mason jars are used as rearing containers for mass cultures. The flat metal lid is discarded, but the screw top ring is retained. Washed and sterilized dry sand to the depth of about three inches is placed in the bottom of the jar and then well moistened, but not soaked, with distilled water. A fresh mushroom or a piece thereof is then dropped onto the surface of the wet sand where it will decompose without becoming infested with mould. The food for the adults is suspended in a container above the sand. I use short 19 mm (3/4 inch) sections of plexiglass tubing I.D. 19 mm (3/4 inch). Three of these sections are cemented transversely onto a rectangular strip (115 mm x 13 mm x 3 mm) of sheet plexiglass. A mass of standard Drosophila media is pressed into the tubular sections. The "food stick" is then suspended in the rearing jar by means of a 7-8 cm long strip of Labtex Tape. One end of the tape is attached to the "food stick" and the other end is lapped over the top edge of the jar.

An 11 x 11 cm piece of thin cloth, such as cheesecloth but preferably bolting silk which can be washed and reused, is placed over the top of the jar and secured by the screw top ring. It is imperative that this cloth cover allow ventilation since the decomposing mushroom releases volatile products which, if not dispersed, are lethal to the flies.

The adult flies are introduced into and removed from the jar by means of an aspirator. It is helpful to have a cloth with a small hole in the center through which the aspirator can be inserted and which can be substituted for the regular cloth lid when removing or introducing the flies. Once every 5-7 days a fresh food stick is introduced and the old one removed if its surface has become mouldy. Every 3-4 weeks the adults are removed and placed in a fresh jar.

The flies should be reared at 17-18^{o}C. This reduces desiccation and the chance of mould forming on the adult food. Such a temperature is mandatory for pinicola and flavopinicola whose females are not able to develop mature eggs at temperatures above 19^{o}C.

At 18^{o}C. pinicola and flavopinicola have long life cycles. The adults do not reach sexual maturity until 12-15 days after eclosion, the females begin to lay eggs about 3 days later and the F_1 emerge 17-23 days later. D. subquinaria has a shorter life cycle but melanderi females do not reach sexual maturity until they are about 30 days old, and each generation takes about 60 days.

Burychenko, G.M. Institute of Developmental Biology, Academy of Sciences of the USSR, Moscow. The modification of the technique of the treatment of insects with chemical mutagenes in gaseous phase.

The technique of treatment of Drosophila flies with chemical mutagenes in gaseous phase is rather simple and efficient (Rapoport, 1965); however, it has not provided the constant-rate dose of mutagenes for one run as mutagenes were dosed by number of drops and treatment conditions varied. We have modified the technique which is briefly as follows (Burychenko, 1968): a constant number of vials with the same number of Drosophila flies are put into the dessicator, the vials containing the nutrient medium and flies covered with gauze. A certain definite volume of mutagene is introduced into the dessicator. The temperature of treatment is normally 25°C. The liquid mutagene or solution of any mutagene is put on a strip of filter paper with the help of a simple device, shown on Figure 1. A micropipette (1) attached to a rubber bulb (2) is used to measure the volume of the mutagene, a screw (3) regulating the volume of the mutagene introduced. The filter paper strip is rapidly attached to the dessicator lid and the dessicator

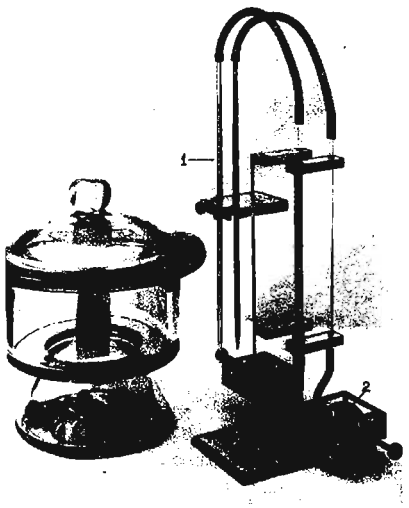


Figure 1. The device for treatment of insects with mutagenes in gaseous phase. (1) micropipette; (2) rubber bulb; (3) screw.

is hermetically sealed. The duration of the treatment is determined experimentally. While studying the sensitivity of different spermatogenesis stages, it must be considerably shorter than the duration of the shortest spermatogenesis stage. Otherwise, the mutations in germ cells could increase, being affected by the mutagene throughout a number of stages. In this case we cannot determine the sensitivity of a particular stage. The prolonged treatment of flies results in almost complete killing of spermatocytes (Burychenko, 1968). The technique described here has been employed in the experiments on silkworms (Abeleva, Burychenko, Gmukhadze, 1973).

References: Abeleva, E.A., Burychenko, G.M., Gmukhadze, N.F. 1973, Genetica USSR IX, N3: 63-68; Burychenko, G.M. 1968, Genetica USSR IV, N 10:77-84; Rapoport, J.A. 1965, Genetica USSR N 1:142-152.

Bos, M. University of Groningen, Haren (Gn), The Netherlands. Extension of life span by low temperature storage.

Sometimes it may be useful to mate particular genotypes of a Drosophila melanogaster stock with flies of different generations. Therefore one needs flies with a duration of life of several months.

In this note I will report on virgin flies from a Kaduna cage population. Males and females were separately stored at 15°C and storage vials were refreshed each fortnight. After 89 days almost all the flies were still alive. A sample of these flies (591 flies) was stored for a further period. They reached a mean age of 162.3 days, that is about 5 months (April - September). The maximum age was 207 days.

A fertility test was done with the 89 day old flies: 10 vials, each with 4 pairs of flies, were cultured at 25°C. They all gave progeny: the mean number of offspring was 7.4 flies per female. As a comparison: 13 day old flies of a Groningen cage population kept at 25° mated at the same time produced a mean offspring of 15 flies per female (no live yeast was added).

Another fertility test was done at the age of 157 days: no offspring were produced.

So, it appears possible to store male and female flies for at least 2 or 3 months with the maintenance of a 50% fertility. No tests have been done to find the optimal storage temperature (or other conditions), but it must be easy to improve the storage method.

Perondini, A.L.P., F.M. Sene and L. Mori. University of São Paulo, São Paulo, Brazil. Xerox copies: an easy way to keep records of zymograms.

The analysis of electrophoretic variants of enzymes and non-catalytic proteins is used extensively in determining the genetic constitution of populations, in many species. In these studies, a large number of analyses are usually done, making the permanent recording of the zymograms a tedious and/or elaborate and costly procedure. Ordinarily, the recording involves a drawing or a photograph of the stained gel.

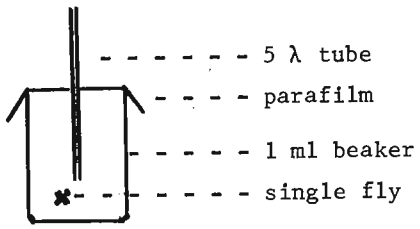
In electrophoresis done in our laboratories the records of the experiments are being kept by taking xerox copies of the stained gels. The gel, after fixation, is wrapped in transparent plastic film (like Zapp or Saran Wrap), taking care to completely smooth the film on to the surface of the gel. The wrapped gel is then placed in the xerox copier and covered with a white paper, on which identification or any other observation about that gel is noted.

The xerox copies, being an exact reproduction of the electrophoretic pattern, allow further measurements and analysis. When the histo-chemical reactions produce different colors (for example the α and β esterases), the xerox copies are labeled with color pencils. This procedure saves considerable time and expense and helps to have a precise and permanent record of the zymograms.

Valencia, R.M. and L. Mansfield. University of Wisconsin, Madison. A method for measuring liquid consumed by individual flies.

When chemical solutions are fed to Drosophila, such as in experiments to test for mutagenicity of environmental substances, it is often important to have at least a rough measure of the amount consumed, so as to compare the treatment with human exposure levels. We have devised a direct feeding method which is quite successful.

Jean M. Clarke (DIS 37:139, 1963) found that flies just beginning to recover from etherization would drink large amounts of fluid presented in a capillary tube. This technique, however, proved to be impracticable for treating large numbers of flies and measuring the amount consumed. Further experimentation with capillary feeding led to the following method.



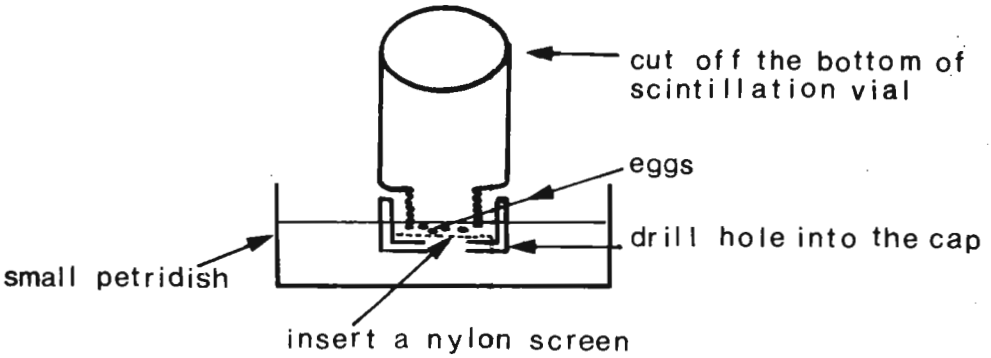
Individual flies, which have been kept in empty dry vials for about four hours, are placed into 1 ml beakers covered with parafilm. A 5 λ disposable micro-pipette (Drummond "Microcap"), filled with the feeding solution, is inserted into the beaker through the parafilm (see diagram). We use 1% glucose as a carrier for our chemicals and as a control solution. The beakers are placed in a closed container with wet filter paper in the bottom, to produce a high humidity and thus reduce evaporation from the tubes. Some beakers without flies are included in order to determine an evaporation factor, which is used to correct all other readings.

Readings are taken at the desired time by laying the capillary tube on or beside a finely marked ruler, having calculated the amount of liquid (fraction of λ) per unit of tube length (fraction of mm). Measurements of consumption within a determined period of time, such as 24 hours, are fairly simple. It is much more difficult to attempt to feed each fly a pre-determined amount, since this entails frequent observation of the flies, which vary extremely in consumption rate and amount, and also the evaporation correction varies with time. It is, of course, possible to feed a large number of flies and choose for study only those which happened to consume the desired amount of liquid. When 10 Canton S males were fed 1% glucose for 24 hours, the average amount consumed (corrected for the evaporation factor of 0.38 λ) was 0.56 λ, with a range from 0 to 1.16 λ for individual males.

Widmer, B. and W.J. Gehring. Biozentrum der Universität Basel. A method for permeabilization of Drosophila eggs.

A method for permeabilization of Drosophila eggs has recently been published by Limbourg and Zalokar (Develop. Biol. 35:382, 1973). Independently we have developed a similar but somewhat simpler method in our laboratory, which proved to be suitable for labelling DNA, RNA and proteins using tritiated thymidine, uridine or amino acids, respectively. Nucleic acids also have been labelled with ^{32}p-orthophosphate. Furthermore, it has been used successfully for introducing metaphase arresting agents (e.g. colchicine) into eggs. Survival of eggs and embryos is greater than 85%. Compared to Limbourg and Zalokar's method our procedure has the advantage that after the permeabilization, the eggs need not be kept in a special medium, provided that the humidity is high enough, and the labelling can be done in water or buffer solution.

Equipment:



nylon monofilament cloth about 50 µ pore size
Tobler, Ernst & Traber, Inc.

Solutions:

Zephiran chloride (K&K Laboratories)
Stock solution 17%; use 0.8 ml stock solution/100 ml H_2O

Sodium hypochlorite 3% (or 50% clorox)

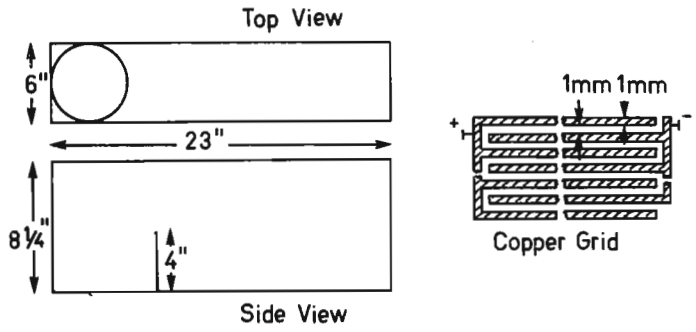
Ether in 70% alcohol:
Prepare freshly each time.
Shake ether with H_2O in separatory funnel to remove peroxides.
To 80 ml of ethanol (70%) add 20 ml of ether.

Procedure: at room temperature

1) Wash eggs with H_2O on nylon screen
2) Wash eggs in zephiran chloride for 3 min.
3) Wash with sterile H_2O dist.
4) Dechorionate with sodium hypochlorite for 2 min.
5) Wash with 100 ml of sterile H_2O dist.
6) Treat with ether-alcohol solution for 2 min. Pipet ether constantly through the nylon screen
7) Wash with 100 ml of sterile H_2O dist.
8) Treat for 2 min. with zephiran chloride again with pipetting
9) Wash with 100 ml of sterile H_2O dist.

Sheppard, D.E.* University of British Columbia, Vancouver, Canada. A selective procedure for the separation of flightless adults from normal flies.

In order to select for mutants that lack the ability to fly, a plexiglass box was constructed according to the following diagram. The main feature of the box consists of a six inch diameter cylinder which opens into the main compartment of the box at the top. A copper grid surrounds the inside of the cylinder and is connected to a conventional electrophoresis power pack. When a potential of 120 volts is placed across the copper grid, any fly that attempts to escape by walking up the sides of the cylinder will receive an electric shock sufficient to cause it to fall back. Under these conditions, only adults capable of flight can escape from the cylinder. In a typical screen, three to five thousand F_1 male progeny derived from EMS-treated male parents were placed into the cylinder following light anesthetization with CO_2 gas. At periodic intervals, the box was shaken to excite the flies. Those flies that escaped to the main compartment were removed by quickly placing a lid onto the top of the cylinder, gassing the main chamber with CO_2 and removing the anesthetized adults by vacuum. Efficiency of separation is increased if the adults are removed from the main chamber at least twice during a two to three hour interval.



Approximately 10-20 percent of the initial population remained in the cylinder at the end of a run, of which approximately half exhibited visible wing defects and were discarded. All of the normal appearing males were mated singly to y f:= females. Each clone was then individually screened for flight ability by visual inspection. Of approximately 20,000 male flies screened in this manner for flightless behavior at 22°C, six mutants were obtained that exhibited a partial or complete loss of flight ability. Stocks of these mutants were made homozygous and characterized quantitatively by placing populations of 250 individuals into the cylinder and determining the retention of the population within the cylinder after thirty minutes. These results are shown in the Table. Complementation analysis indicates that each of these mutations is defective in a different gene.

Retention of adult flies exhibiting partial or complete loss of flight behavior

Strain	Number of populations tested	Arcsin* (transformed from % retention)
301	3	90.0
306	3	88.3 ± 4.1
307	3	83.6 ± 0.5
312	3	90.0
321	5	54.8 ± 3.1
326	3	75.7 ± 2.8
Oregon-R	4	25.8 ± 1.2
striped	2	90.0

* An Arcsin of 90 is equivalent to 100 percent retention of the population.

In a second screen, approximately 46,000 flies were examined for flightless behavior at 29°C. From 3,058 single pair matings, fifty-one putative mutants were detected, none of which exhibited a clear restoration of flight activity at 22°C. These mutants should prove useful in a genetic dissection of the Drosophila flight mechanism. This screen also yielded other types of mutants including a series of rudimentary and Hyperkinetic mutants and a cold-sensitive, bang-sensitive mutant.

Research supported by NIH Grant 1-F03-GM-55,084-01 to D.E.S. and NRC A-1764 and NCI 6051 of Canada grants to D.T. Suzuki.

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Montijn, C., F.R. van Dijken, M.H. den Boer and W. Scharloo. University of Utrecht, The Netherlands. Apparatus for a measurement of locomotor activity in Drosophila.

To measure the behavioral trait of locomotor activity in D. melanogaster, it was felt necessary to construct an apparatus in which it would be possible to handle hundreds of flies at one time. We started from the principle of the apparatus used by Barton Brown and Evans

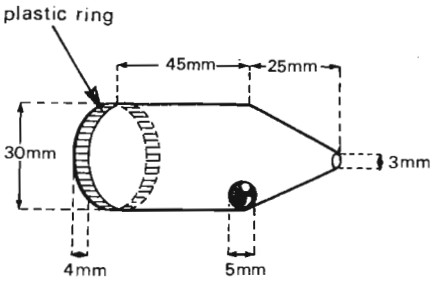


Figure 1

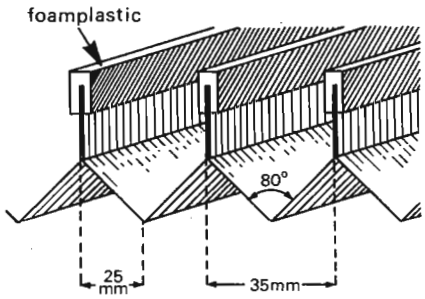


Figure 2

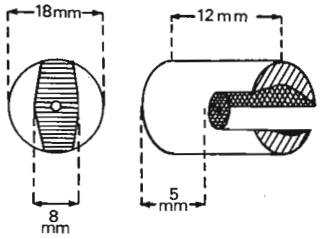
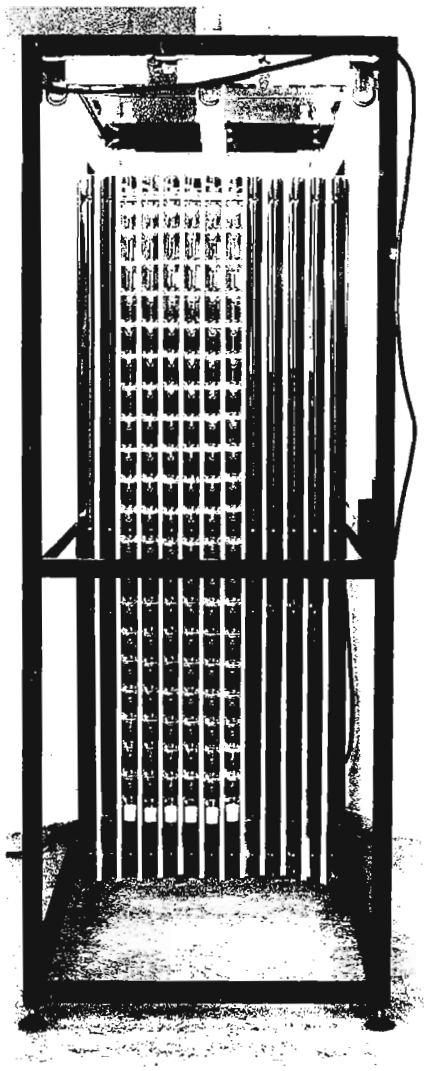


Figure 3



(1960) to measure locomotor activity in the blowfly. This apparatus was adapted for Drosophila by Ewing (1963). With his apparatus it was only possible to select for flies out of the first and last compartments. In order to use locomotor activity as a quantitative trait, it is necessary to obtain a more detailed separation of different levels of activity. Therefore we looked for a possibility of locking all compartments simultaneously. Our apparatus consists of a series of glass tubes (see Figure 1), each of which contains a small stainless iron ball. These tubes lie in a gutter made of a stainless iron sheet (high finish) and are fixed in their places by a layer of plastic foam (ethafoam, Dow Chemicals (R.)) on both sides of the gutter (see Figure 2). The apparatus consists of 13 of these gutters with a length of 119 cm, so that it is possible to have 20 tubes in a row. The plane in which the gutters lie can be turned on an axis perpendicular to the gutters in a horizontal and a vertical position, fixed with a touch latch. In vertical position the holes at the ends of the tubes are closed by the little balls; in horizontal the holes are open and the balls lie in the position as shown in Figure 1, because there is a tiny deviation from the horizontal axis.

To collect flies out of the tubes, we use a magnet which fixes the ball in the exit of the tube (see Figure 3). Then it is possible to remove the flies from the tube by only light shaking. As light source we use three vita-lite tubes (fluorescent 40 W each), which are placed 63 cm above the tubes with a mutual distance of 21 cm.

References: Barton Browne, L. and D.R. Evans 1960, J. Inst. Physiol. 4:27-37; Ewing, A.W. 1963, Animal Behav. XI:369-378.

Figure 4: Photograph of apparatus in vertical position.

Candido, E.P.M. and D.L. Baillie. University of British Columbia Medical School, Vancouver, Canada. Labelling of Drosophila with ^{35}S and ^{32}P.

In the course of studies on Xanthine dehydrogenase from Drosophila, we have worked out a cheap and effective procedure for labelling flies with ^{35}S. Incorporations of $5x10^6$ cpm per fly or per third instar larva have been obtained. Baker's yeast is grown in a sulfate-free medium (R. Graham and W.M. Stanley, Jr., Anal. Biochem. 47:505, 1972) containing 1mCi/ml of carrier-free $^{35}SO_4^=$ overnight at 30^0 without aeration. Incorporation into the cells is 50-70%. The yeast is collected by centrifugation and stored at -20^0 until used. Flies may be labelled by feeding second or third instar larvae on a paste of this yeast plus 1% sucrose. For larger preparations, the labelled yeast may be added to the surface of ordinary Oak Ridge food, to a maximum of 5 mCi per half pint culture; the incorporation into flies is still reasonable. We have found that a lower level of incorporation may be achieved by simply adding 1-5 mCi of $^{35}SO_4^=$ to ordinary cultures when they have reached second instar, and then it is advisable to transfer the labelled larvae or adults to an unlabelled live yeast medium for a few hours prior to use.

The labelling achieved by this simple procedure is sufficient to allow the autoradiography of proteins in extracts from single flies, after electrophoresis in polyacrylamide gels. The use of $^{35}SO_4^=$ renders the process much cheaper than would be the case if ^{35}S-methionine were used. Incorporation of ^{35}S from labelled yeast into flies ranges from $5x10^5$ to $5x10^6$ cpm per fly, and when $^{35}SO_4^=$ is added directly to the food, levels of $5x10^4$ to $5x10^5$ cpm per fly can be expected.

A useful low phosphate medium for labelling Baker's yeast with ^{32}P for use in the above procedure is described by G.M. Rubin, J. Biol. Chem. 248:3860, 1973. Levels of greater than 10^5 cpm per fly can be expected. Labelling by these procedures is not recommended for first instar larvae, since mortality is very high. For obvious reasons, the use of flightless strains such as vg is recommended.

Koliantz, G. and J. Darwishpur. Teachers' Training College and Institute of Tadj Pahlavi, Tehran, Iran. A culture medium for D. melanogaster.

Mostashfi's culture medium (1957) is suitable for wild types and many mutants of D. melanogaster. It contains dry yeast and vinegar which is suspended by hydrophile cotton. Because of different hydrophility rates of cottons sometimes the medium becomes dry or wet. In both cases it makes trouble for weak mutants.

In 1968 one of us (K.G.) prepared a new culture medium with the following constituents: Yellow corn meal 30 gr., agar 8 gr., sugar 22.5 gr., water 570 ml. Propionic acid was used as bacteriocide.

This medium was used only for laboratory stocks, because it was found that propionic acid (or sometimes a moldex) are unable to precent the high fungal and bacterial infections carried by flies captured from nature.

To change these troubles we prepared a new medium which shows successful results in culturing some wild types, namely Gayaneh, Java, Oregon, Tehran and many mutants. (Details for mutants are available upon request). This medium, tabled below, is used for some classical crosses done by college students and also used for flies collected directly from nature. Because of low pH of grape vinegar, neither moldex nor bacteriocide are necessary.

Materials	Amounts
Dry yeast*	20 gr.
Agar**	8 gr.
Vinegar***	150 ml.

* Suggested the mark Cenovis
** Suggested the mark Merck, Art 1615
*** Standardized grape vinegar

To prepare culture: Weigh dry yeast and agar, mix them and pour into a pyrex flask, then add vinegar. After shaking well, autoclave them for 15 minutes in 120^oC. Still warm, distribute to sterile test tubes, flasks or half pint bottles. The medium in the test tubes need be only 3 cm deep, (slanting is desirable), and in other vials approximately 2 cm deep is enough. Stopper the tubes and vials with cotton and after hardening store in refrigerator. Before using stand in laboratory temperature for an hour.

Sederoff, R. and R. Clynes. Columbia University, New York. A modified medium for culture of Drosophila cells.

For biochemical experiments, we have been culturing Drosophila melanogaster cells in spinner cultures. These cells, derived from Schneider's line 2 by Garen, will grow on a variety of media, however, we have cultured them routinely on Schneider's medium which is commercially available (Grand Island Biological). For large scale culture the cost of commercial medium became prohibitive and we began to prepare our own medium. That lowered the cost greatly, but the cost of the medium was still much greater than the cost per liter for mammalian cell medium. Moreover, the medium was quite complex and we felt that some simplification would be useful. We have therefore carried out a series of modifications which attempted to make Schneider's medium cheaper, easier to make in large quantity (10 liters) and more chemically defined. A chemically defined medium for cell growth has not yet been achieved, but some progress has been made in that direction. Many of the modifications we describe simply involve leaving out compounds.

Cells grown in suspension on our modified medium (Reduced Schneiders) divide approximately every 48 hours. They saturate at a density of 8×10^6 cells per ml; we routinely dilute them when they reach 6 to 8×10^6 cell per ml. The cells are diluted two fold in fresh medium. We grow suspension cultures either in standard closed spinner flasks or in screw-cap erlenmeyer flasks with a teflon spinner bar at the bottom. Suspension cultures are started from one or more confluent monolayer bottles (250 ml Falcon plastic T flask with 10 mls of medium) diluted with 100 mls of medium for each T flask. A karyotype analysis of our cells indicates that a large fraction of the cells have normal or nearly normal chromosome composition.

When we began growing our cells, we added various supplements to Gibco's Schneiders medium including bactopeptone, NCTC vitamins, indicator and 15 percent fetal bovine serum. Simply running low on money forced us to eliminate the vitamins and to reduce the serum level to 5% (or even 2%). We found that bactopeptone also could be eliminated with no apparent effect on growth rate in spinner culture.

One major problem with Schneider's medium is that of insoluble material which exists or is formed during preparation. We have improved the situation in the following way. Cystine, which is added to the medium and is very insoluble, has been left out. It seemed unlikely that cystine would be essential since cysteine is also added to the medium. Leaving out cystine, again had no noticeable effect. Another insoluble compound in the medium was tyrosine, which was added in amounts exceeding its solubility. We reduced the amount of tyrosine added to a level just below the solubility at $25^{\circ}C$.

Four organic acids present in the original medium have been left out at Imogene Schneider's suggestion. The cells show no difference in growth, but the pH of the medium during preparation is greatly changed. As a result, less NaOH is needed to neutralize the medium when adjusting the pH, and the Na^+ ion concentration is not as greatly distorted as it had been with the organic acids present. However, a strict protocol for preparation must be observed or insoluble precipitates will result.

Trehalose, although the major sugar in hemolymph, is not required by the cells if glucose is present. We have doubled the amount of glucose added and deleted the trehalose. The trehalose is rather expensive and this modification was motivated only by an attempt to save money.

Phenol red, the traditional indicator of mammalian cell culture is not as suitable for Drosophila cells. The pK of phenol red is 7.9 and it is not very sensitive to pH changes at 6.7. A more suitable indicator seemed to be bromthymol blue with a pK at 7.3. Bromthymol blue is yellow at acid pH, green at neutral pH and blue at alkaline pH. We have been using bromthymol blue instead of phenol red and our cultures are therefore green. The indicator is in fact very useful since pH changes occur with biological or chemical contamination or when mistakes are made in the preparation of the medium. The medium as we now prepare it has a dark green hue which lightens somewhat when serum and cells are added.

As a result of these modifications, we have reduced the cost of the chemicals to about two dollars a liter. Including serum at 5 percent, the cost is comparable to that of mammalian cell cultures, approximately five dollars per liter. We were previously spending $14.00 per liter when we prepared the medium ourselves. Using Schneiders in solution from Gibco raises the costs to about $50.00 per liter.

At this point only two components are undefined, serum and yeast extract. We expect that serum may be reduced, dialysed or possibly eliminated, since some serumless lines have been developed by Echalier. We hope to be able to reduce serum or eliminate it with this modified

medium. Yeast hydrolysate (yeastolate) is more of a problem.

Yeastolate is absolutely essential for growth of our cells and the hope of a completely defined medium may require that we learn what is supplied by yeast hydrolysate. This problem can be approached by adding defined ingredients or by fractionating yeast hydrolysate to attempt to define the essential components. Two available vitamin mixtures, NCTC (Gibco) and Eagles vitamins have been tested and they will not eliminate the need for yeast hydrolysate.

We have been concerned about these undefined components because of radioactive labelling experiments. For labelling cells with ^{3}H thymidine, we found that standard medium with serum, yeast hydrolysate and bactopeptone greatly reduced the efficiency of labelling. By preparing modified medium without yeast hydrolysate and serum (now bactopeptone is always left out) we have increased our incorporation in short labelling (1 to 4 hours) by an order of magnitude.

We have decided to call this medium Reduced Schneiders medium because the previous recipe was already called modified Schneiders. Most chemicals we use are purchased from Sigma or Calbiochem. Yeastolate is purchased from Difco. Heat treated serum and other cell culture products are purchased from Gibco.

A medium somewhat simpler than Schneiders has been developed by Echalier and Ohanessian (1970) called D20. It has 3 undefined components, lactalbumin hydrolysate, yeast hydrolysate and serum. We chose not to use it because some lines of Schneider (1972) would not grow on it. However, our line of suspension-adapted cells, derived from line 2 of Schneider by Garen, called line E, grew on both media equally well. One advantage of Reduced Schneiders over D20 medium is the absence of lactalbumin hydrolysate. Reduced Schneiders has no added vitamins and no added organic acids. We hope that future studies will bring us closer to a defined medium for cultured fly cells.

References: Schneider, I. 1972, J. Embrol. Exp. Morph. 27:353; Echalier, G. and A. Ohanessian 1970, In Vitro 6:162.

Ingredients for 10 liters of medium
All measurements are given in grams unless otherwise specified.

	Schneiders	Reduced Schneiders
cystine	1	none
tyrosine	5	4.2
NaCl	21	21
KCl	16	16
KH_2PO_4	4.5	4.5
Na_2HPO_4	7	7
$NaHCO_3$	4	4
cysteine	0.6	0.6
histidine	4	4
leucine	1.5	1.5
arginine	4	4
β alanine	5	5
glycine	2.5	2.5
serine	2.5	2.5
proline	17	17
phenylalanine	1.5	1.5
lysine	16.5	16.5
glutamic acid	8	8
aspartic acid	4	4
glutamine	18	18
tryptophane	1	1
methionine	8	8
threonine	3.5	3.5
isoleucine	1.5	1.5
valine	3	3
glucose	20	40
trehalose	20	none
yeast hydrolysate (yeastolate Difco)	20	20
$CaCl_2$	6	6
$Mg\ SO_4 \cdot 7H_2O$	37	37
alpha-ketoglutaric acid	2	none

	Schneiders	Reduced Schneiders
fumaric acid	1	none
succinic acid	1	none
malic acid	1	none

Supplements	Previously used with Schneiders	Now used with Reduced Schneiders
FBS	10 to 15%	5% or 2%
bactopeptone	50	none
phenol red (indicator)	10 mgs	none
bromthymol blue	none	100 mgs
NCTC vitamins	1 to 2%	none

Procedure for making 10 liters of reduced medium:

Step 1: 10 liters distilled water in large container which can be continuously stirred at room temperature. Add 4.2g tyrosine and stir, heat gently to dissolve or wait patiently. Then add all other amino acids.

Step 2: Weigh out all other ingredients on list except serum. Add to solution and stir for 30 minutes. Solution should finally be clear. Some light particles of unknown origin may still be visible.

Step 3: Cool to 15°C and raise pH while stirring to 6.7 with 5N NaOH (about 5 mls). A transient precipitate will form as the NaOH is added.

Step 4: Sterile filter. Medium can be stored in this form for several months at 4°C.

To Use: Before use, add Fetal Bovine Serum (heat inactivated) to final concentration of 5% (or 2%). If required, antibiotics can be added. We use Gibco antibiotic antimycotic mix at 1%.

Recently we have successfully reduced the serum requirement for our cells in monolayer to 0.5 percent and we anticipate growing these cells serum free within the next few months. Lengyel and Penman (Cell Biology Abstracts for 14th Annual Meeting Abstract no. 381) have adapted Schneiders line 2 to grow on Dulbecco's medium including lactalbumin hydrolysate. Cells grown on Dulbecco's medium no longer need yeast hydrolysate. The combination of yeast hydrolysate independent medium and serum independent medium would finally result in a defined medium for Drosophila cells.

Brent, M.M. and I.I. Oster. Bowling Green State University, Bowling Green, Ohio.

Nutritional substitution - a new approach to microbial control for Drosophila cultures.

A sine qua non of raising Drosophila is that the flies introduced initially and/or their progeny should comprise the only living organisms in the culture containers (with the possible exception of those formulations which call for seeding with live yeast. On the other hand, because the medium ordinarily used for Drosophila melanogaster (and many other species) is relatively rich in nutrients, a wide variety of unwanted but ubiquitous organisms may find such surroundings ideally suited for multiplication. In addition to introducing another variable (whose undesirability would be compounded by the fact that it would tend to be inconstant from culture to culture and experiment to experiment) certain molds and bacteria are capable of exerting deleterious effects on stocks, particularly on heavily-marked flies and/or on those with single mutants which are phenotypically extreme.

Often cultures which are heavily infested with mold (e.g., Penicillium) will become totally covered by a mat of hyphae which can prevent larvae and adults from feeding adequately and females from laying eggs. Moreover, the surface of such cultures will become drier than is desirable in those cases where the covering of mold is sufficiently luxuriant to entrap the water normally interspersed in the gel-like Drosophila culture medium. Various bacterial intruders in or on the medium can also pose serious problems for the unwary Drosophilid (and by extension, his keeper). The growth of some bacterial contaminants is such that they may cause the entire surface of the culture to take on a slimy consistency. Just as in the case of the

hyphae of molds, a slimy mat-like cover may interfere with the feeding of the larvae and adults and the egg-laying of the females. In addition, its sticky nature often poses mechanical problems - not allowing the weaker and/or smaller larvae to burrow freely and miring the adults.

In connection with any discussion of microbial infection of fly cultures it cannot be overemphasized that periodic washing with soap and water followed by swabbing of etherizers, counting plates, desk tops, and the hands (not neglecting the fingernails) of those who come into contact with the cultures with 70% ethyl alcohol goes a long way in minimizing the establishment of foci of contamination. Based on our own experience (attested to by the fact that the Mid-America Drosophila Stock Center and its associated laboratory areas have been maintained in a completely mold- and mite-free condition since its inception in 1959 without our even having to resort to propionic acid as an inhibitor) we would recommend that washing of utensils with soap may be carried out at regular weekly intervals but swabbing with alcohol should be carried out on a daily basis. Needless to say, in order to be effective, hand-washing is necessary prior to the start of one's activities in the "fly-room" and should be repeated as necessary (e.g., after tea or coffee breaks, lunch, other "natural" contingencies, etc. - depending on how often the particular individual must undertake other endeavors outside of the "clean areas" represented by the fly-rooms).

Up to the present time all laboratories which have been engaged in Drosophila work have tried to rely on the addition to the medium of agents which would inhibit the growth and/or destroy any microbial contaminants. These compounds have included Nipagin, Tegosept (methyl parahydroxybenzoate), propionic acid, streptomycin, other antibiotics, etc. While these agents have enjoyed some success, being essentially toxic, their incorporation into the medium may result in the retardation of the development, and in some cases destruction, of the flies. As a matter of fact, doses of some compounds which are distinctly efficacious in controlling microbial contamination are often unusable because of their untoward effects on the flies!

Recently, notwithstanding our most diligent and scrupulous efforts, a number of experimental cultures containing heavily marked stocks became infested with a microbial contaminant which exacted a heavy toll by over-growing the medium. This microbe was characterized by a very sticky slime-like habit; cultures with a heavy infection just barely yielded offspring because the slime posed both mechanical and physiological problems for the developing larvae and flies. The offending culprit was isolated using standard microbial techniques and was identified as Leuconostoc. Prior to this, it might be mentioned, "shot-gun" treatment of the cultures with a whole host of antibiotics, including streptomycin, penicillin, tetracycline, erythromycin, etc., proved to be singularly ineffective in checking the infection (at least in the doses utilized).

It then occurred to us that it might be possible to approach the problem from a different angle. Since it is known that Leuconostoc secretes a gummy capsule composed largely of dextran polysaccharides and it is this feature which makes it such an undesirable contaminant, we decided to attempt to eliminate the production of the encapsulating layer. In order to synthesize the dextran-containing capsule, Leuconostoc must be grown on sucrose-containing substrates. Since this bacterium possesses the enzyme sucrose transglucosylase, which catalyzes the sucrose to dextran conversion, it seemed logical for us to develop a sucrose-free medium. After numerous trials we found that the following formula works admirably well under the conditions prevalent in our laboratory (temperature of either 64°F, 74°F or 80°F and humidity of 40-60% - for those laboratories where there is a different mix of temperature and humidity it may be necessary to determine empirically whether slightly different proportions, particularly of water and agar, are better):

Corn meal	1400	grams
Brewer's yeast	280	grams
Agar	80	grams
Dextrose	2270	grams
Water	15,400	cc
Benzyl benzoate	350	cc (1 1/2% solution in 95% ethyl alcohol)
Tegosept	250	cc (10% solution in 95% ethyl alcohol)

It is prepared in the same manner as the standard corn meal-molasses medium. Actually the chief departure from the commonly-used medium consists of the substitution of dextrose for sucrose (or a sucrose-source as in the case of molasses). Yields and handleability of this medium are equal, if not superior, to several food-formulae in use in our laboratory and elsewhere.

In addition to controlling a particular microbe (while Leuconostoc may still infest the

cultures it is incapable of producing its undesirable slime layer on dextrose) the method described affords a new approach for the maintenance of vigorous cultures of Drosophila - that is, by substituting in the diet of the potentially-infective microbial contaminant some essential nutrient without at the same time subjecting the Drosophilids to undue stress and thereby compromising their development.

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Beeson, V.S.* Oregon State University, Corvallis, Oregon. Mouth aspirator for use in mutant screening.

Mouth aspirators have been used in behavior experiments for transferring flies without etherization. In addition, they have been found to be extremely useful in experiments in which the progeny of large numbers of single pair crosses are to be screened. The aspirator (Figure 1) is made from a Pasteur pipette by breaking off the long bill to give an opening large enough for an adult fly to be drawn through without damage. The other end of the pipette is loosely plugged with cotton and a piece of flexible tubing with a mouthpiece (of the type used for hematology).

Crosses set in vials are the easiest to handle but half pint culture bottles also can be used. When the culture is inverted, the flies walk upward toward the medium. The plug can then be removed or merely held loosely in place while specific flies are selected from the sides of the vial and drawn into the aspirator by mouth suction. If necessary these can be checked under the dissecting microscope or a magnifying lens while confined in the aspirator. This technique has been found to be considerably faster than the etherization of a culture. In addition to eliminating the chance of over-etherization, the flies seem to mate and oviposit sooner making this a particularly useful technique when one wants progeny as soon as possible, or for use in a teaching lab.

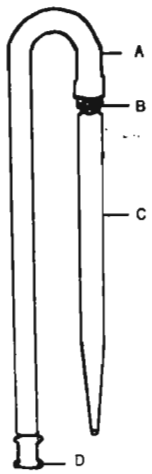

Figure 1. Apparatus for transfer of Drosophila melanogaster. (A) Tubing, (B) Cotton plug, (C) Pasteur pipette, (D) Mouthpiece.

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ANNOUNCEMENTS

Bibliography on the Genetics of Drosophila Part VI, edited by I.I. Herskowitz, covering the years 1968 - 1972, and containing 4821 references, has been published by Macmillan Company. It costs $17.95. (Please do not order copies from DIS). Prof. Herskowitz has updated that bibliography with a special section in this issue of DIS.

Handbook of Genetics, in five volumes, edited by Robert C. King, Northwestern University, is being published by Plenium Publishing Corp., New York. Volumes 1 (Bacteria, Bacteriophages and Fungi) and 2 (Plants, Plant Viruses and Protists) were released in late 1974.

Two volumes of H.J. Muller's essays have been published by State University of New York Press, 99 Washington Avenue, Albany, New York 12210. Man's Future Birthright ($6.95) contains Muller's humanistic and social essays on such topics as eugenics, coexistence, human freedom, science fiction and the evolution of values. An introductory essay by Bentley Glass evaluates Muller's contributions and life. The Modern Concept of Nature ($8.95) contains essays on genetics and evolution, especially gene theory, neo-Darwinism, genetic load, radiation genetics, and mutation. Both volumes should be of interest to geneticists and students of genetics. The articles are complete, with their bibliographies, and the editing, by E.A. Carlson, includes a chronological synopsis of Muller's career.

MacIntyre, R. Cornell University, Ithaca, New York. Phenotypically identical but genotypically unique Drosophila "unknown" stocks for genetics laboratory courses.

At Cornell, all biological sciences majors are required to take a course in general genetics which includes a mandatory 2 hour laboratory component. As part of the laboratory, each student is required to genetically analyze an unknown strain of D. melanogaster and to write a detailed report on his analysis. This is a six-seven week project and, accordingly, contributes substantially to his grade in the course. Unknowns have generally 3 - 6 mutant genes and are crossed to the following marker stocks: (1) Wild type, to identify the genes and their individual phenotypic effects and to localize them to the X or to autosomes; (2) Cy/Pm;Sb/Ubx to place autosomal genes on the 2nd, 3rd or 4th chromosome; (3) BL L/Cy;Ly Sb or Gl Sb/LVM;f B, to map genes on the 2nd, 3rd and X chromosomes respectively. Most students work very hard on this project, and it is not surprising that the "fruit fly experiment" has a widespread, rather notorious reputation. I have always felt, however, that the hard work put in by the students was justified by and, in fact, enhanced the tutorial value of the genetic analysis.

Recently, it came to my attention at a wedding reception in a fraternity house, that there were extensive collections of old Drosophila reports at several places on and around the campus. The inebriated fraternity member bragged to me how valuable these collections were to the genetics "students" seeking short cuts to high grades. Usually by the F_1 generation there would be enough information to enable the short-cutter to find an already complete analysis of his unknown in one of these collections. Because of the obvious unfairness of this practice to the serious, hard working student and because of the gamesmanship involved, I decided to construct a set of "unknowns" which would force all students to complete the analysis. After examining several combinations, I found one which involves no difficult phenotypic interactions with the marker stocks. This is a combination of orange eyes, black bodies and incomplete wing veins. In the stocks I've constructed so far, orange eyes are due to the interaction of car; st, car; kar, pn; st, pn; kar, v car or pn v. Body color is due to b or e and wing veins to shv or ve. Thus, I have 24 genotypically unique but phenotypically identical or at least very similar stocks (b and e as well as shv and ve show different phenotypes to the trained eye). I plan to multiply this number by incorporating s and rb or ca as additional body color and brown eye mutants in the near future. If anyone would like subcultures of these stocks, please request them during the summer months.

MATERIALS REQUESTED OR AVAILABLE III

Request for lethal mutants: Year after year, large numbers of lethal mutants are produced by Drosophilists and after time-consuming selection procedures, most of them land in the waste basket. I am interested in obtaining this waste. Why? (1) to save myself work, and (2) to rummage among them for mutants with malignant neoplasms. You may not be aware of the fact that Drosophila develops real, malignant neoplasms of genetic origin. The genetic bases of these neoplasms makes it a choice animal for studying the genetics of the neoplastic transformation process. You will be informed about each successful find and your participation as a co-author will be noted in any possible forth-coming publications. Your cooperation is gratefully appreciated. Please send material to Elizabeth Gateff, Biological Institute I, University of Freiburg, 9-11 Schänzlestr., 78 Freiburg i.Br., West Germany.

Lee Ehrman, Division of Natural Sciences, State University, Purchase, New York 10577, requests reprints, etc. of published and in-press descriptions of drosophilid mating behaviors for a chart to be included in the planned chapter, "Sexual Behavior" in The Genetics and Biology of Drosophila, Vol. 2, edited by T. Wright and M. Ashburner.

Bruce Wallace, Genetics, Development and Physiology, Cornell University, Ithaca, New York 14850, has lost his A_1 B_{18} (CyL-Ubx)/(Pm-Sb), stock. This is a stock which by virtue of both translocations and inversions permitted the manipulation of both second and third chromosomes as one unit. He wishes to learn if anyone is still maintaining this stock of flies.

Irwin H. Herskowitz, Editor

D. = Drosophila D.m. = Drosophila melanogaster

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Faccio Dolfini, S. Milan Italy
Falk, R. Jerusalem, Israel
Farish, D.J. Columbia, Missouri
Farmer, J.L. Provo, Utah
Félix, R. Mexico City, Mexico
Fenner, H. Santiago, Chile
Feuerbach-Mravlag, H. Vienna, Austria
Finnerty, V.G. Storrs, Connecticut
Fontdevila, A. Barcelona, Spain
Fourche, J. Lyon, France
Fowler, G.L. Eugene, Oregon
Fox, D.P. Aberdeen, Great Britain
Frankel, A. Iowa City, Iowa
Franklin, R. Sydney, Australia
Frei, H. Geneva, Switzerland
French, W.L. Baton Rouge, Louisiana
Frías, D. Santiago, Chile
Friedman, T.B. Bethesda, Maryland
Fritz-Niggli, H. Zürich, Switzerland
Fuchs, M.S. Notre Dame, Indiana
Fujii, H.M. Fukuoka, Japan
Fukatami, A. Sakado-Machi, Japan
Futch, D.G. San Diego, California
Fuyama, Y. Tokyo, Japan
Gale, J.S. Birmingham, Great Britain
Gall, J.G. New Haven, Connecticut
Ganetzky, B. Seattle, Washington
García, P. Barcelona, Spain
Gardner, E.J. Logan, Utah
Garen, A. New Haven, Connecticut
Garen, S. New Haven, Connecticut
Gartner, L.P. Baltimore, Maryland
Gatti, M. Rome, Italy
Gehring, W. Basel, Switzerland
Gelbart, W. Storrs, Connecticut
Gerresheim, F. München, Germany
Gersh, E.S. Philadelphia, Pennsylvania
Gethmann, R.C. Catonsville, Maryland
Ghysen, A. Pasadena, California
Gilbert, R.I.F. Swansea, Great Britain
Gill, K.S. Ludhiana, India
Gingeras, T. New York, New York
Girard, P. Paris, France
Glätzer, K.H. Düsseldorf, Germany
Gloor, H. Geneva, Switzerland
Godoy, R. Santiago, Chile
Gonzales F., H. Lima, Peru
González, R. Barcelona, Spain
Goux, J.M. Paris, France
Grabicki, E. New Haven, Connecticut
Graf, U. Zürich, Switzerland
Green, M.M. Davis, California
Grigliatti, T. Vancouver, Canada
Gross, J.E. Chicago, Illinois
Grossman, A. Jerusalem, Israel
Guest, W.C. Fayetteville, Arkansas
Guevara P., M. Lima, Peru
Guillet, C. Lyon, France
Gupta, A. Cambridge, Massachusetts
Gupta, A.K. Ludhiana, India
Gupta, J.P. Varanasi, India
Gupta, P. Calcutta, India
Guzman, J.M.S. Mexico City, Mexico
Hackman, W. Helsinki, Finland
Hadorn, E. Zürich, Switzerland
Haendle, J. München, Germany
Hägele, K. Bochum-Querenburg, Germany
Halfer, C. Milan, Italy
Hall, J.C. Waltham, Massachusetts
Hall, L. Cambridge, Massachusetts
Hammerschmidt, H. München, Germany
Hampel, H. Vienna, Austria
Hanna, P.J. Clayton, Australia
Hannah-Alava, A. Turku, Finland

Hara, K. Fukuoka, Japan
Hardy, D.E. Honolulu, Hawaii
Hardy, R. Los Angeles, California
Hardy, R.W. La Jolla, California
Harrison, B.J. Norwich, Great Britain
Hartl, D.L. West Lafayette, Indiana
Hartmann-Goldstein, I.J. Sheffield, Britain
Hauschteck-Jungen, E. Zürich, Switzerland
Hay, D.A. Bundora, Australia
Hedrick, P.W. Lawrence, Kansas
Hennig, W. Tübingen, Germany
Herforth, R.S. Minneapolis, Minnesota
Herskowitz, I.H. New York, New York
Hess, C. Zürich, Switzerland
Hess, O. Düsseldorf, Germany
v. Heuduck, A. München, Germany
Hexter, W.M. Amherst, Massachusetts
Hiroyoshi, T. Osaka, Japan
Hoekstra, R.F. Haren, Netherlands
Holden, J. Basel, Switzerland
Hollander, W.F. Ames, Iowa
Hollingsworth, M.J. London, Great Britain
Holm, D. Vancouver, Canada
Holmgren, P. Umeå, Sweden
Homyk, Th. Vancouver, Canada
Honda, Y. Nagasaki, Japan
Hoorn, A.J.W. Utrecht, Netherlands
Hotta, Y. Tokyo, Japan
Hubby, J.L. Chicago, Illinois
Ikeda, H. Matsuyama, Japan
Ikeda, K. Duarte, California
Imberski, R.B. College Park, Maryland
Inagaki, E. Hiroshima, Japan
Inocenio, B. Purchase, New York
Ish-Horowicz, D. Basel, Switzerland
Istock, N. Rochester, New York
Iturra, P. Santiago, Chile
Ives, P.T. Amherst, Massachusetts
Iyama, S. Misima, Japan
Jacob, G. Tübingen, Germany
Jacob, R. Tübingen, Germany
Jain, H.K. New Delhi, India
Janning, W. Münster, Germany
Jarry, B. Vancouver, Canada
Jarry, B.R. Marseille, France
Jeffery, D.E. Honolulu, Hawaii
Jeffery, D.E. Provo, Utah
Jinks, J.L. Birmingham, Great Britain
Johannisson, R. Düsseldorf, Germany
Johnson, K. London, Great Britain
Johnson, W.W. Albuquerque, New Mexico
Jones, G.H. Birmingham, Great Britain
Jong, G. de Utrecht, Netherlands
Jordan, B.R. Marseille, France
Jungen, H. Zürich, Switzerland
Kaji, S. Kobe, Japan
Kale, P.G. Varanasi, India
Kalisch, W.-E. Bochum-Querenburg, Germany
Kambysellis, M.P. New York, New York
Van der Kamp, K. Haren, Netherlands
Kamping, A. Haren, Netherlands
Kaneko, A Tokyo, Japan
Kaneshiro, K.Y. Honolulu, Hawaii
Kang, Y.-J. Seoul, Korea
Kang,(Song), S.-J. Seoul, Korea
Kang, Y.S. Seoul, Korea
Kankel, D.R. New Haven, Connecticut
Kaplan, W.D. Duarte, California
Karlik, A. Vienna, Austria
Karlsson, B-M. Umeå, Sweden
Katoh, S. Sakado-Machi, Japan
Kaufman, T.C. Vancouver, Canada
Kawanishi, M. Misima, Japan
Kearsey, M.J. Birmingham, Great Britain
Kelly, L. Brighton, Great Britain
Kiefer, B.I. Middletown, Connecticut
Kiger, J.A. Davis, California
Kikkawa, H. Osaka, Japan
Kimura, M. Misima, Japan
Kirschbaum, W.F. Buenos Aires, Argentina
Kitagawa, O. Tokyo, Japan
Kobayashi, Y. Hiroshima, Japan
Kobel, H.R. Geneva, Switzerland
Köhler, W. Berlin, Germany
Korge, G. Hünchen, Germany
Kortselius, M.J.H. Leiden, Netherlands
Kosuda, K. Sakado-Machi, Japan
Kramers, P.G. Leiden, Netherlands
Krause, E. South Orange, New Jersey
Kress, H. München, Germany
Krider, H. Storrs, Connecticut
Krimbas, C. Athens, Greece
Krishnamurthy, N.B., Mysore, India
Krivshenko, J.D. Rochester, New York
Kroeger, H. Saarbrücken, Germany
Kroman, R.A. Long Beach, California
Kubli, E. New Haven, Connecticut
Kunz, W. Düsseldorf, Germany
Kuo, G. New Haven, Connecticut
Kuroda, Y. Misima, Japan
Lachaise, D. Gif-sur-Yvette, France
Laird, C. Seattle, Washington
Lakhotia, S.C. Ahmedabad, India
Lakovaara, S. Oulu, Finland
Lamb, M.J. London, Great Britain
Lambert, D. Brisbane, Australia
Lambertsson, A. Umeå, Sweden
Lande, R. Cambridge, Massachusetts
Lankinen, P. Oulu, Finland
Lawrence, M.J. Birmingham, Great Britain
Lee, C.C. Seoul, Korea
Lee, T.J. Seoul, Korea
Lee, W.R. Baton Rouge, Louisiana
Lefevre, G. Northridge, California
Legay, J.M. Lyon, France
Leibenguth, F. Saarbrücken, Germany
Leibold, Ch. Bochum-Querenburg, Germany
Leigh, B. Leiden, Netherlands
Leister, F. Baltimore, Maryland
Lemeunier, F. Gif-sur-Yvette, France
Leonard, J. Purchase, New York
Leoncini, O. Tübingen, Germany

Levins, R. Chicago, Illinois
Levitan, M. New York, New York
Lewis, E.B. Pasadena, California
Lewontin, R. Cambridge, Massachusetts
L'Helias, C. Gif-sur-Yvette, France
Lindsley, D.L. La Jolla, California
Link, B. Tübingen, Germany
Llorens, A. Barcelona, Spain
Lokki, J. Helsinki, Finland
Lommerse, R. Leiden, Netherlands
Long, T.C. Chapel Hill, North Carolina
Louis, M. Gif-sur-Yvette, France
Loukas, M. Athens, Greece
Loverre, A. Rome, Italy
Lowy, P.H. Pasadena, California
Lucchesi, J.C. Chapel Hill, North Carolina
Lüers, H. Berlin, Germany
Lumme, J. Oulu, Finland
Lüning, K.G. Stockholm, Sweden
MacBean, I.T. Bundoora, Australia
MacIntyre, R. Ithaca, New York
Maddern, R.H. Leiden, Netherlands
Magalhães, L.E. de São Paulo, Brazil
Magnusson, J. Stockholm, Sweden
Maher, E.P. Aberdeen, Great Britain
Mainx, F. Vienna, Austria
Majumdar, S.K. Easton, Pennsylvania
Mandal, S. Calcutta, India
Manna, P.K. Calcutta, India
Manosalva B., J. Lima, Peru
Mansfield, L. Madison, Wisconsin
Margulies, L. New York, New York
Marques, E.J. Mato Grosso, Brazil
Martinez I., N. Lima, Peru
Martinez, R.M. Hamden, Connecticut
Maruyama, T Misima, Japan
Mather, K. Birmingham, Great Britain
Mather, W.B. Brisbane, Australia
Matsubara, T. Sakai, Japan
Matsuzaki, Y. Tokyo, Japan
Mayfield, J.E. Pittsburgh, Pennsylvania
Mazar Barnett, B. Buenos Aires, Argentina
McCarron, M. Storrs, Connecticut
McCrady, W.B. Arlington, Texas
McKechnie, S. Bundora, Australia
Megna, F. Naples, Italy
Mendelson, D. Leiden, Netherlands
Mercader, J. Mexico City, Mexico
Merriam, J.R. Los Angeles, California
Mettler, S. DeKalb, Illinois
Meyer, G.F. Tübingen, Germany
Micheli, A. Rome, Italy
Michinomae, M. Kobe, Japan
Miglani, G.S. Ludhiana, India
Mikasa, K. Sakado-Machi, Japan
Miklos, G.L. Canberra, Australia
Milkman, R. Iowa City, Iowa
Miller, D.D. Lincoln, Nebraska
Miller, O.L.Dr. Charlottesville, Virginia
Minamori, S. Hiroshima, Japan
Minato, K. Misima, Japan
Mindek, G. Zürich, Switzerland
Mitchell, H.K. Pasadena, California
Miyamoto, D.M. Durham, North Carolina
Moffitt, S. Oxford, England
Mollet, P. Zürich, Switzerland
Momma, E. Sapporo, Japan
Monclús, M. Barcelona, Spain
Montalenti, G. Rome, Italy
Montelius, I. Stockholm, Sweden
Montell, I. Umeå, Sweden
Montijn, C. Utrecht, Netherlands
Moree, R. Pullman, Washington
Mori, S. Nagasaki, Japan
Moriwaki, D. Misima, Japan
Mortensen, M. Copenhagen, Denmark
Moskwinski, T. Notre Dame, Indiana
Mourad, A.M. Alexandria, Egypt
Muckenthaler, F.A. Bridgewater, Massachusetts
Mukherjee, A.S. Calcutta, India
Mulley, J C. Sydney, Australia
Muñoz, E.R. Buenos Aires, Argentina
Murakami, A. Misima, Japan
Murnik, M.R. Macomb, Illinois
Murray, N.D. Bundoora, Australia
Nag, A. Calcutta, India
Nakai, S. Osaka, Japan
Nakao, Y. Hiroshima, Japan
Nakashima-Tanaka, E. Sakai, Japan
Narise, S. Sakado-Machi, Japan
Narise, T. Sakado-Machi, Japan
Nash, W.G. Bethesda, Maryland
Navarro, J. Santiago, Chile
Nawa, S. Misima, Japan
Nigon, V. Lyon, France
Nirmala Sajjan, S. Mysore, India
Nöthel, H. Berlin, Germany
Nöthiger, R. Zürich, Switzerland
Nouaud, D. Paris, France
Novitski, E. Leiden, Netherlands
Nygren, J. Umeå, Sweden
O'Brien, S.J. Bethesda, Maryland
Oelshlegel, F.J. Ann Arbor, Michigan
Ogaki, M. Sakai, Japan
Agita, Z. Osaka, Japan
Ohba, S. Tokyo, Japan
Ohta, T. Misima, Japan
Oikarinen, A. Oulu, Finland
Oksala, T.A. Turku, Finland
Okubo, S. Osaka, Japan
Olivieri, G. Rome, Italy
Olvera, O. Mexico City, Mexico
Oram, G Birmingham, Great Britain
Orevi, N. Jerusalem, Israel
Osborn, R. Berkeley, California
Oshima, C. Misima, Japan
Ouweneel, W.J. Utrecht, Netherlands
Paik, S.G. Seoul, Korea
Paik, Y.K. Honalulu, Hawaii
Pak, W.L. West Lafayette, Indiana
Palabost, L. Paris, France
Parisi, G. Naples, Italy

Parkash, R. Ludhiana, India
Parsons, P.A. Bundoora, Australia
Pavan, C. São Paulo, Brazil
Pavlovsky, O. Davis, California
Paz, C. Buenos Aires, Argentina
Pelecanos, M. Patras, Greece
Pereira, S.M.F. Mato Grosso, Brazil
Pérez-Salas, S. Caracas, Venezuela
Periquet, G. Paris, France
Persson, K. Umeå, Sweden
Petit, C. Paris, France
Petitpierre, E. Barselona, Spain
Petrella, L. Milan, Italy
Petrovich, S.B. Catonsville, Maryland
Pilares G., L. Lima, Peru
Pinsker, W Tübingen, Germany
Plus, N. St. Christal les Alès, France
Poodry, C.A. Santa Cruz, California
Portin, P. Turku, Finland
Postlethwait, J.H. Eugene, Oregon
Potter, J.H. College Park, Maryland
Poulson, D.F. New Haven, Connecticut
Powell, J.R. New Haven, Connecticut
Prakash, S. Rochester, New York
Preuss, V. Tübingen, Germany
Prevosti, A. Barcelona, Spain
Pruzan, A. Purchase, New York
Pulvermacher, Ch. Berlin, Germany
Purnell, D.P. Swansea, Great Britain
Puro, J. Turku, Finland
Quinn, W.G. Princeton, New Jersey
Rae, P. New Haven, Connecticut
Rahman, R. Calcutta, India
Rai, K.S. Notre Dame, Indiana
Rajasekarasetty, M.R. Mysore, India
Ramel, C. Stockholm, Sweden
Ranganath, H.A. Mysore, India
Ransom, R. Utrecht, Netherlands
Rapport, E. Burnaby, Canada
Rasmuson, B. Umeå, Sweden
Rasmuson, M. Umeå, Sweden
Ratty, F.J. San Diego, California
Ray-Chaudhuri, S.P. Calcutta, India
Razzini, A. Milan, Italy
Relton, J.M. Sheffield, England
Remondine, D.J. Houghton, Michigan
Reuterwall, C. Stockholm, Sweden
Ribó, G. Barcelona, Spain
Rick, J.T. Sheffield, Great Britain
Rickoll, W. Durham, North Carolina
Rinehart, R.R. San Diego, California
Ripoll, P. La Jolla, California
Rivera, M.L. Barcelona, Spain
Rizki, R.M. Ann Arbor, Michigan
Rizki, T.M. Ann Arbor, Michigan
Robbins, L. East Lansing, Michigan
Roberts, D.B. Oxford, England
Roberts, P.A. Corvallis, Oregon
Robertson, A. Edinburgh, Great Britain
Robertson, F.W. Aberdeen, Great Britain
Rockwell, R.F. Kingston, Canada
Roehrdanz, R.L. Chapel Hill, North Carolina
Rokop, S. La Jolla, California
Rosenfeld, A. Seattle, Washington
Rosset, R. Marseille, France
Rudden, N. Oxford, Great Britain
Ruderer-Doschek, E. Vienna, Austria
Rungger, E. Geneva, Switzerland
Russell, R. Bundoora, Australia
Rutherford, P. Aberdeen, Great Britain
Ryman, N. Stockholm, Sweden
Sakaguchi, B. Fukuoka, Japan
Sakoyama, Y. Osaka, Japan
Salceda S., V.M. Chapingo, Mexico
Sanders, T.G. Princeton, New Jersey
Sandler, L.M. Seattle, Washington
Sang, J.H. Brighton, Great Britain
Sankaranarayanan, K. Leiden, Netherlands
Santos, E.P. dos São Paulo, Brazil
Sarkar, D.N. Varanasi, India
Saura, A. Helsinki, Finland
Savontaus, M.-L. Turku, Finland
Schabtach, E. Eugene, Oregon
Schäfer, U. Düsseldorf, Germany
Schalet, A. Leiden, Netherlands
Scharloo, W. Utrecht, Netherlands
Scheid, W. Münster, Germany
Schmid, D. Tübingen, Germany
Schmolesky, G. Madison, Wisconsin
Schouten, S.C.M. Utrecht, Netherlands
Schubiger, G. Seattle, Washington
Schweizer, P. Zürich, Switzerland
Schwinck, I. Storrs, Connecticut
Schwochau, M. Düsseldorf, Germany
Sederoff, R. New York, New York
Seecof, R.L. Duarte, California
Seki, T. Osada, Japan
Sene, F.M. Honolulu, Hawaii
Sene, F.M. São Paulo, Brazil
Sernau, R. Madison, Wisconsin
Seyffert, W. Tübingen, Germany
Shafer, S.J. Oakdale, New York
Sharma, A.K. Ludhiana, India
Sharma, R.P. New Delhi, India
Shearn, A. Baltimore, Maryland
Shields, G. Brighton, Great Britain
Shima, T. Sapporo, Japan
Shiomi, T. Nagasaki, Japan
Shorrocks, B. Leeds, Great Britain
Sick, K. Copenhagen, Denmark
Siddaveere Gowda, L. Mysore, India
Sillans, D. Lyon, France
Simmons, J.R. Logan, Utah
Singh, B.N. Varanasi, India
Singh, R. Cambridge, Massachusetts
Slatis, H.M. East Lansing, Michigan
Slizynska, H. Edinburgh, Great Britain
Smith, B.R. Aberdeen, Great Britain
Smith, D.A Birmingham, Great Britain
Smith, J. Canberra, Australia
Sobels, F.H. Leiden, Netherlands
Södergren, A. Umeå, Sweden

Sofer, W. Baltimore, Maryland
Soll, D.G. New Haven, Connecticut
Somero, M.G. La Jolla, California
Søndergaard, L. Copenhagen, Denmark
Sorsa, M. Helsinki, Finland
Sorsa, V. Helsinki, Finland
Sparrow, J. York, Great Britain
Speers, L. Ottawa, Canada
Sperlich, D. Tübingen, Germany
Spiers, G. Edinburgh, Great Britain
Spofford, J.B. Chicago, Illinois
Springer, R. Vienna, Austria
Sreerama Reddy, G. Mysore, India
Ståhl, G. Stockholm, Sweden
Stennek, A. Stockholm, Sweden
Stern, C. Berkeley, California
Stewart, B. Los Angeles, California
Stewart, D. Waltham, Massachusetts
Strøman, P. Copenhagen, Denmark
Suchopová, N. Brno, Czechoslovakia
Suchowersky, O. Vancouver, Canada
Sulerud, R.L. Minneapolis, Minnesota
Suomalainen, E. Helsinki, Finland
Suzuki, D.T. Vancouver, Canada
Suzuki, S. Vancouver, Canada
Svahlin, H. Umeå, Sweden
Svensson, I. Umeå, Sweden
Takaya, H. Kobe, Japan
Takikawa, S. Sagamihara, Japan
Tantawy, A.O Alexandria, Egypt
Targa, H.J. São Paulo, Brazil
Tener, G. Vancouver, Canada
Thalmann, G J Oakdale, New York
The, D. Sydney, Australia
Thirtle, B. Ann Arbor, Michigan
Thongmeearkom, P. Brisbane, Australia
Thörig, G E W. Utrecht, Netherlands
Throckmorton, L H. Chicago, Illinois
Tigerstedt, P. Helsinki, Finland
Tiivola, A. Helsinki, Finland
Tobari, Y.N. Tokyo, Japan
Tokunaga, C Berkeley, California
Tokuyasu, K. La Jolla, California
Tonomura, Y. Tokyo, Japan
Traut, A. Münster, Germany
Traut, H. Münster, Germany
Trippa, G Rome, Italy
Trout, L. San Diego, California
Trout, W E. Duarte, California
Tsacas, L. Gif-sur-Yvette, France
Tsakas, S. Athens, Greece
Tsuno, K. Sakado-Machi, Japan
Tsusue, M. Sagamihara, Japan
Tsutiyama, S. Fukuoka, Japan
Tuinstra, E J Utrecht, Netherlands
Twardzik, D.R. Bethesda, Maryland
Ulrich, H. Zürich, Switzerland
Urbischek, E.F. Swansea, Great Britain
Ushioda, Y. Kobe, Japan
Vaidya, V.G. Poona, India
Valadé, E. Santiago de Compostela, Spain
Valencia, J.I. Madison, Wisconsin
Valentin, J. Stockholm, Sweden
Van Deusen, E. Basel, Switzerland
Van Herrewege, J. Villeurbanne, France
Van Valen, L. Chicago, Illinois
Vepsäläinen, K. Helsinki, Finland
Verburgt, F.G. Leiden, Netherlands
Viinikka, Y. Turku, Finland
Vilageliu, L. Barcelona, Spain
Vogel, E. Leiden, Netherlands
Wada, R. Osaka, Japan
Waddington, C.H. Edinburgh, Great Britain
Waddle, F.R. Fayetteville, North Carolina
Wallace, B. Ithaca, New York
Wallace, H. Birmingham, Great Britain
Ward, C.L. Durham, North Carolina
Wargent, J.M. Sheffield, Great Britain
Warn, R. Oxford, Great Britain
Watanabe, TlK. Misima, Japan
Watson, W.A.F. Aberdeen, Great Britain
Wehner, R. Zürich, Switzerland
Weideli, H Basel, Switzerland
Welshons, J. Ames, Iowa
Welshons, W.J. Ames, Iowa
Wensink, P. Waltham, Massachusetts
Westerman, J. Bundoora, Australia
Westerman, M. Bundoora, Australia
White, B N. Kingston, Canada
White, K New Haven, Connecticut
Whittinghill, M. Chapel Hill, North Carolina
Whittle, J.R.S Brighton, Great Britain
Whitty, R.W. Canberra, Australia
van der Wielen, A W Leiden, Netherlands
Wieschaus, E. Basel, Switzerland
Wildeboer-du Pui, M.L.L Haren, Netherlands
Williamson, R Duarte, California
Wimber, D. Eugene, Oregon
Winkel, E.-U. Berlin Germany
Wöhrmann, K. Tübingen, Germany
Wong, P. Duarte, California
Wright, E.Y Charlottesville, Virginia
Wright, T.R.F. Charlottesville, Virginia
Würgler, F.E. Zürich, Switzerland
Wyl, E. von Zürich, Switzerland
Wyman, R.J. New Haven, Connecticut
Yakoumi, G. Athens, Greece
Yamada, M.A Misima, Japan
Yamaguchi, O. Tokyo, Japan
Yamazaki, H I. Tokyo, Japan
Yamazaki, T. Misima, Japan
Yanders, A F. Columbia, Missouri
Yannopoulos, G Patras, Greece
Yeh, F. Halifax, Canada
Yokokawa, C Sagamihara, Japan
Yoshikawa, I Nagasaki, Japan
Yost, H.T Amherst, Massachusetts
Ytterborn, K.H Stockholm, Sweden
Zacharopoulou, A. Patras, Greece
Zárate, E. Santiago, Chile
Zimm, G La Jolla, California
Zouros, E Halifax, Canada