# DROSOPHILA

## Information Service

# 38

## December 1963

Material contributed by
DROSOPHILA WORKERS

and arranged by
E. NOVITSKI



Prepared at the
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EUGENE, OREGON

# DROSOPHILA INFORMATION SERVICE

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The attention of all Drosophilists is urgently directed to the note by Profs. Stalker and Carson on page 96

Research Notes

It will be evident after a glance at this DIS that stock lists notes are no longer arranged alphabetically, but rather higgeldy-piggeldy. This is unfortunate; the convenience of the alphabetical listing helped make DIS the useful document it is. Now one must go to the table of contents to find a specific note, by name of author, or a stock list, by city.

The reason for this is that contributions are now typed in order of receipt, so that much of DIS is completed before the official deadline. Further, the number of contributions is now so great that the cut-off point for a given issue actually comes before the official deadline, and must await the next issue. Another reason is that we must try to position, properly, on the page, those notes for which reprints have been ordered, and this disrupts any simple alphabetical arrangement.

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Below is an alphabetical list of the basic mutant symbols that have appeared in DIS subsequent to the publication of Bridges and Brehme. This list is meant to help those who wish to name mutants but who do not have DIS' available to check to see if a symbol which seems appropriate to them, has, in fact, been used before. This list also can be used to check names of mutants since the symbols should bear a close relationship to the names. In the event that the appropriate DIS' are not available locally (the compilations do not include the "new mutants" sections), it may be necessary to get the appropriate DIS on inter-library loan, or if you write to the editor of DIS, he will supply you with a photocopy of the pages in question. Note that the same symbol has been used for different names in about ten per cent of all cases.

As,31:80; abab,24:54; abe,33:82; abt,33:83; Ac,22:53; acc,32:67; Ae,20:63; agl,24:60; alan,21:66; alo,32:67; α,34:51;35:46; als,20:63; 21:66; amb,32:67;32:67; amy,34:51; ang,24:61; Antp,30:76; apb,24:59; apr,30:71; apx,33:83; Asy,24:62;25:75; at,18:45;22:53; At,23:64;28:74; Ata,29:73; aw,24:54;25:77; awu,33:100

bal,32:82; bar,24:58; bashed,18:40; bc,20:66; ber,32:67; bis,32:67; Bit,23:58; bk,33:83; Bk,23:58; bkl,33:83; bla,32:67; Ble,33:97; blu,21:71; bp,29:72; bran,18:41;18:40; brb,33:83; brc,32:68; bre,32:68; brw,33:83; bsc,32:68; bsd,19:45; Bt,24:54;25:77; bur,23:60;34:52; bwl,33:83; bz,32:68

Cb,19:47; cbf,20:64; Cbx,28:76; ccw,32:68; Cd,29:69; Ch,17:51; che,25:71; chi,21:66; cho',29:75; clf,32:68; clm,32:68; co,18:44; coi,34:48; cop,32:68; cor,21:66; Cor,20:66; corr,38:31; cpl,33:83; cpw,32:68; cq,28:74;32:83; crc,19:45; crk,32:69; crm,25:78;32:69; crt,33:84; csk,32:69; ctt,33:84; Cu,22:53;32:80; Cur,26:66; ckrl,18:42; cvw,32:69; Cyg,35:47; cyl,32:82

da,33:84;28:73; der,32:69; dfa,33:84; dft,33:84; dfw,33:84; Din,29:70; dkl,33:85; dlv,33:85; dor,32:88; dg,30:73; dr,23:60; drb,25:75; dsh,33:85; dss,33:85; dta,32:69; dtv,32:69; du,24:57; dvw,33:85; dwf,33:85; dwg,33:85; dwu,33:85; dyb,33:85

eb,33:86; Eb,17:50; ef,33:86; Elp,34:50; elr,34:49; en-bx,33:96; en-w,31:81; er,17:50; er-w,24:60; exi,32:70; exr,32:70; ext,32:82

fas,24:54;25:77; fb,32:70; fd,24:54;25:77; fd adp,34:48; fes K,31:81; ff,33:86; fft,28:78; fg,34:49;35:45; fil,33:86; fin,33:86; fla,32:70; fm,33:86; fnc,33:86; Fo,34:52; fs,26:61; fs,25:72;34:49; fs adp,35:45; fs 1, 36:37; ful,21:67

ge,32:70; gli,32:70; Go,22:55; gr,33:86; gs,31:85; gv,20:65

ha,32:70; hdp,32:70; hpa,33:86; hs,17:51

im,33:86; Ind,21:72; ix,32:83

ke,33:87; kf,33:87; Ki,32:80; kno;32:70; krb,20:68

lac,33:87; lf,33:87; Lg,24:55; lgh,33:87; lix,34:50; lme,33:87; loz,17:48; lyp,34:50

Mas,33:98; Mc,18:40; mc-like,32:71; mch,33:87; mdg,32:71; meg,32:71; mel,32:71; mf,32:71; mgt,32:71; mib,33:88; Mio;32:84; mis,32:71; mk,32:71; mn,33:88; mo,32:72;33:99; ms,32:72; 33:97;24:60;25:69; mtb,33:88; mumac,34:51; mur,31:81; mwh,25:70; mwi,32:72

ne,20:66; ne,21:67; neu,31:80; ni-s,21:67; nr,36:38; nrs,33:88; nub,23:61; obl,32:72; Oce,32:72; Of,31:85;30:79; omm,32:72; oo,21:67; opa,21:67; opa,18:43; opb,33:88; or,20:68; or,24:61; or,19:45; osh,32:73; ot,32:73; otri,19:45; Outspan,24:62; ovi,32:73; pa,27:56; pa,23:61; pat,32:73; pbx,28:76; Pc,21:69; Pl,25:75; plw,33:88; pol,27:59; pop,32:73; pra,33:88; prn,21:67; Ps,33:95; pta,33:88; pte,32:73; pub,24:55; pun,32:73; pvt,33:88; Qd,33:99

rab,24:55; rai,27:55; rbc,24:59; rc,34:50; rd,32:74; rdb,33:89; rdm,33:89; rdt,33:89; rea,32:73; re-b,28:78; red,28:77; ref,33:89; ref,27:59; re-o,23:63; ret,32:73; rey,22:55; rgt,33:89; rk,22:53; rm,32:74; rm,33:89; roe,38:31; Roi,26:65;30:72; rr,29:73; rsc,31:85; rsd,19:46;21:69; rsi,33:89; Rst,32:87; Rst,32:87; rta,33:89; rv,33:89; rw,29:70; rwg,32:74;

saw,26:60;26:61; sb,24:54; sbs,33:90; sbt,33:90; Sc,20:67; Scar,23:65; sch,33:90; Sco,33:96; scrp,36:38; sct,34:49; sfc,32:74; sg,23:57; sge,32:74; Sh,23:61;23:62; shb,33:90; shf,24:55; shl,33:90; shm,33:90; sho,26:65; shp,32:74; sht,33:90; shv,21:69;33:90; shw,33:90 siw,32:74; sla,32:74; slb,32:74; slc,33:90; sld,33:91; slm,32:75; sln,33:91; slw,32:75; sma,32:75; smb,33:90; smd,32:75; sme,33:91; smh,33:91; sml,24:56; smn,33:91; smp,33:91; smt,32:75; so,20:63; so',20:64; so,20:69;21:67; som,32:75; sple,18:43; splw,32:75; sps,25:73 spt,20:65;33:91; sts,33:92; stt,33:92; stu,33:92; su-poi,18:43; Su-Cy,38:28; Su-y,24:62; su-Hw,23:59; Su-ss,24:59; su-v,26:63; Su-Pm,33:95; Su-tu,28:74; swa,33:92; swb,34:49; swy,33:92

ta,23:61;27:56; Tac,32:80; taw,32:75; tc,32:76; tdd,32:76; te,32:76; ten,32:76; tft,33:99; Tft,26:68; Tg,38:32; tha,33:93, thb,32:76; thl,33:93; thm,32:76; tht,33:93; thv,32:76; tmc,32:76; tmo,24:62; tms,33:93; tnt,33:93; tny,33:93; tom,25:73;32:83;32:76; tpw,32:76; tra,18:44; trb,33:93; tre,33:93; Tu,21:70;17:49;23:57;31:83;25:69; tuf,22:56; twg,33:93; twl,28:74; twt,33:94

Ubx,26:66; uex,36:39; unc,34:49; unp,33:94; up,32:77; ups,32:77; uq,32:77; us,33:94

vac,32:77; vao,33:94; vd,20:64; ves,32:77; vi,20:64;21:68; vnl,25:77; vst,18:40; vta,20:64;21:68

wa,24:56;32:77; wd,18:44; wgv,33:94; wh,38:29; wi,38:31; wl,20:66; wm,38:32; wr,33:100; ws,32:77; wtw,32:77; ww,32:78; wxt,31:81

ylt,31:81

z,33:82

## Editor's Comments

The yearly expense of issuing DIS is spiralling upwards. Not only are there more contributors, but each now sends in more material, with respect to both number of notes and their length. Many of the notes now submitted are clearly meant as publications, for which there is no better repository than DIS.

It is necessary for DIS to become more self-sufficient and, I hope, completely so in the not-too-distant future. Therefore, the following policy will be adopted starting with DIS 40, to appear a year from now. Each worker will be given gratis the equivalent of a half page of DIS, for his contributions, either singly or as a co-author. Any space in excess of that amount will be charged at the rate of $20 per page or fraction thereof. This is more than the actual cost per page, but will support the other sections (stock lists, new mutants, directory, etc.) for which individual charges will not be levied. It might be noted that DIS now costs about $5,000 per year.

Accompanying each bill for page costs will be a statement avowing lack of research funds for payment, which the contributor may sign and return in lieu of payment. This will be meant particularly for contributors from outside the U. S., where research funds may not be quite as available as in the U. S., or where currency exchange problems make payment difficult. It is hoped that all Drosophila workers in the position to do so will look upon these charges as a welcome means of making DIS self-supporting.

E. Novitski

## AMES, IOWA: IOWA STATE UNIVERSITY
### Department of Genetics

The following are additions to the Ames (Genetics Department) list of DIS 34, which otherwise remains the same.

Chromosome 1

5a dow/FM6, $y^{31d}$ $sc^{8}$ dm B
9a $pn^{2}$ $w^{sat}$ $ras^{2}$ v f
9b Qd
25a y w cv $rux^{60d}$ sn
28a $y_{2}$ v r f/Basc
28b $y^{2}$ v ma-l

Chromosome 2

31a B1 $L^{2}$/SM5
36a ds dp
36b l(2) hst/Gla
38a Sp J L Pin/Gla
38b Sp J L Pin/SM1
38c Su-Cy/SM1
* Su-Cy in 68b

Chromosome 3

39a $bar\text{-}3_{nd}$
43a $e^{s}$ $ca^{nd}$/In(3LR) $ca^{vPl}$
44a $p^{p}$ cu
44b ri $p^{p}$/T(Y;2;3) F, st
48a ss cu th
48b ss e tx
49a st
51a W

Chromosome 4

52a ci gvl bt $Cat$/$ey^{D}$
52b ci gvl spa

Multichromosomal

64a g; dp; by
64b M-5; SM1/$ds^{33k}$ dp b Pm; $Ubx^{130}$ $e^{s}$/C Sb; pol
66a dp; $tx_{33k}$
68a SM1/$ds^{33k}$ dp b Pm; $Ubx^{130}$ $e^{s}$/C Sb
68b SM1/$ds^{33k}$ Su-Cy Pm; $Ubx^{130}$ $e^{s}$/C Sb
68c $Ubx^{130}$ $e^{s}$/$bx^{34e}$; pol

Attached-X

71a sc $m^{D}$ f · f v sc

Closed-X

88 $X^{c2}$, ec f/y f:=

Inversions

* Ins(1)$sc^{1L}$, S, $sc^{8R}$ in 28a, 64b
* Ins(1)FM6 in 5a
* In(2)Gla in 36b, 38a
* Ins(2)SM1 in 38b, 38c, 64b, etc.
* Ins(2)SM5 in 31a
* Ins(2)Pm in 64b, 68a, 68b
* Ins(3)$Ubx^{130}$ in 64b, 68a, 68b, 68c
* In(3LR)Cx in 92

Translocations

89 T(Y;2)dp/SM1
* T(Y;2;3)F in 44b
90 T(2;3)$bw^{V4}$/SM1
91 T(2;3)B, ru h D ss $e^{2}$/Payne
92 T(3;4)e/In(3LR)Cx $bx^{34e}$ $e^{4}$
93 T(3;4)86D,

## EAST LANSING, MICHIGAN: MICHIGAN STATE UNIVERSITY
### Department of Zoology, Biology Research Center

Wild Stocks

Canton-S
Crimea
Samarkand
Swedish-B
Urbana

Chromosome 1

br
cm
cy
f
g
$m^{D}$
$M^{D}$/FM3
$rb_{sc^{8}}^{S1}$ B In $w^{a}$ $sc^{8}$ (muller-5)
$sc^{8}$·Y/y B/y f:=
$w^{a}$
$w^{e}$

y
w m
w m f
y Hw $sn^3$/y w f:=
$X^{c1}_S$, y $v^+_L$/y w f:=
Y w $y^+$·Y y /0/ y w:=

Chromosome 2

bw
dp
vg

b pr
cn bw
Cy/Pm
pk cn
SD-5

Chromosome 3

ca
st
se

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

Multichromosomal

b; se h
bw; st
Cy/Pm; H/In(3R)Mo, Sb sr
v; bw
y f:p; bw; e; ci $ey^R$
y $sc^{S1}$ $In^{49}msc^8$; bw; st $p^p$

## LEXINGTON, KENTUCKY: UNIVERSITY OF KENTUCKY

### Wild Stocks

Lexington, Kentucky

## WALTHAM, MASSACHUSETTS: BRANDEIS UNIVERSITY
### Department of Biology

BL/L/CyII
bw
bw/st
Cy/Pm/D/2b
dk
e
e, se
ey
l(2) gl cn bw/Cy
l(2) gl cn bw/SM5, $al^2$ Cy $lt^r$ $sp^2$
LS wild type
se
se ss
st
vg
$w_a$
w
w/vg
XX
y
$y_{Lc}$ v ♂ & y f:=
$Y^3_L$/lz f·$Y^s$ ♂ & y v f:=
$Y^L$/f· $Y^s$ ♂ & sc v f:=
y w mi
Y/BS/M8/YNS
$Y^2$/NF/car

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

Wild Stocks

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

Chromosome 1(X)

14 B
15 B-reverted
16 bi
17 bi $ct^6$ $g^2$
18 car
19 $ct^6$
20 dor/ClB
21 dor/FM4
22 fa
23 fu/ClB
24 $fs^{NasA}$/M-5
25 g/ClB
26 $g^{50e}$

27 $g^{50e}_{496}$/y f:=
28 $Hw^{496}$/M-5
29 l(1)48J/M-5
30 $lz^{50e}$
31 na/FM3, $y^{31d}$ $sc^{8}$ dm B l
32 $pn^{2}$
33 sc ec v g f/ClB
34 sc ec cv $ct^{6}$ v g f/ClB, v
35 $sc^{S1}$ ec v g f
36 $sc^{S1}$ B, In-S, $w^{a}$ $sc^{8}$ (M-5)
37 $sn^{3}$/ y f:=
38 $sn^{3}$ v
39 $sn^{4}$ v B
40 $sn^{3}$ oc $ptg^{3}$/+:=
41 v
42 w
43 w ec
44 w m f
45 $w_{a}$ spl
46 $w_{a}$
47 $w_{a}$ v B
48 $w_{bf}$ v B/In(1)AM
49 $w_{bf}$
50 $w_{bl}$/FM4
51 $w_{e}$
52 w
53 $y_{2}$ sc $w^{a}$
54 y sc $w^{a}$ ec/FM4, $y^{31d}$ $sc^{8}$ dm B (triploid)
55 $y^{2}_{2}$ sc $w^{a}$ ec/y f:=
56 $y_{2}$ v f
57 $y_{2}$ $w_{a}$ cv v f/M-5
58 $y_{2}$ $w_{a}$ cv $sn^{55a}$ v f/M-5
59 $y_{a}$ w $w_{496}$/y f:=
60 $w_{2}$ $fw^{a}$ /y f:=
61 $y^{2}$ w m f

## Chromosome II

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

## Chromosome III

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

## Chromosome IV

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

## Multichromosomal Stocks

112 $ct^{45e}$ v; bw; e; $ey^{2}$(1;2;3;4)
113 g; cn(1;2)
114 v; bw(1;2)
115 v; bw; e(1;2;3)
116 v; bw; e; $ey^{2}$(1;2;3;4)
117 $sc^{S1}$ B In-S $w^{a}$ $sc^{8}$; In SM1, $al^{2}$ Cy $sp^{2}$/ dp b Pm $ds^{33k}$; C Sb/$Ubx^{130}$ $e^{s}$(1;2;3) (H-40)
118 v;e(1;3)
119 w;e(1;3)
120 $w_{e}^{a}$ v; e(1;3)
121 w ;cn(1;2)
122 $y_{2}$w; ant(1:2)
123 $y^{2}$ v f; bw(1;2)
124 bw;e(2;3)
125 bw;st(2;3)
126 cn bw; e(2;3)
127 cn; se(2;3)
128 dp;e(2;3)

129 Pm, dp b/Cy $sp^2$;Sb/CxF (ru h ca?)(2;3)
130 pr;$ey^2$ (2;4)
131 e;$ey^2$ (3;4)

Closed-X

132 $X^c$, y/y $f^{vc}$:=
133 In($X^{c2}$)$w^{vc}$ f/dl-49, y w $lz^s$

Deficiencies

Chromosome I(X)

134 Df(1)$g^1_{4.2}$ f B/In(1)AM
135 Df(1)$L_8$/M-5
136 Df(1)$N_{45e}$/y Hw $m^2$ $g^4$, dl-49
137 Df(1)$N_{264-40}$/y Hw $m^2$ $g_2$, dl-49
138 Df(1)$N_{258-11}$/y Hw $m^2$ $g_2$, dl-49
139 Df(1)$w_{258-21}$, y/y Hw $m_4$ g, dl-49
140 Df(1)$w_{258-42}$/y Hw $m^2$ $g_2$, dl-49
141 Df(1)$w_{258-45}$, y/y Hw $m^{31d}$ $g_8$, dl-49
142 Df(1)$w_{258-48}$/FM4, $y^2$ $sc_4$ dm B
143 Df(1)$w_{m4L}$, y/y Hw $m^{3R}$ g, dl-49
144 Df(1)$w_{551}$, rst /y Dp(1;3)$^{a7}_{49}$
145 Df(1)$w^{33.2}$ spl/In(1)$sc^{S1}$, dl-49, v f B
146 Df(1)$w^{56d\ 10.1}$ spl/In(1)$sc^{S1}$, dl-49, $y^{59K13}$ f B
147 Df(1)$w^{59k13}$ spl($sn^3$)/Dp(1)$w^V$, cb
148 Df(1)$w^{59k13}$ spl/In(1)$sc^{S1}$ dl-49, v f B
149 Df(1)$y^2$ sn $w^a$ $w^{56l\ 10.2}$/In(1)$sc^{S1}$ dl-49, v f B
150 Df(1)y sc/M-5 (Vogt)

Chromosome II

151 Df(2)$bw^5$ $sp^2$/Xa
152 Df(2)$bw^{VDe2L^R}$ sp/SM1, $al^2$ Cy $sp^2$
153 Df(2)$bw^2$ $Cy_2$/Gla
154 Df(2)$Px^B$ bw $sp_4$/Cy, $al^2$ $lt^3$ $L^4$ $sp^2$
155 Df(2)$vg_C$/Cy, $L_4$ $sp^2$
156 Df(2)$vg_D$/Cy, $L_4$ $sp^2$
157 Df(2)$vg_S$/Cy, $L^4$ $sp^2$
158 Df(2)vg cn/Cy, $al^2$ $lt^3$ $L^4$ $sp^2$

Duplications

159 Dp(1;1)Co

Inversions

see 20, 21, 31, 36, 48, 78, 79, 80, 96, 97, etc.

## LONG BEACH, CALIFORNIA: LONG BEACH STATE COLLEGE
### Department of Biology

Wild Stocks

Oregon-R
Canton-S

Chromosome 1

1 bo
2 bo cx
3 cm
4 $ec_{n3}$ cv
5 $f^2$
6 $pn^2_3$
7 $sn^3$
8 spl
9 $v_a$
10 $w_{58k29}$ $bo^9$
11 $w_{58l12}$
12 $w^{RM}$
13 $w_{a2}$
14 $w_{a4}$
15 w
16 y ac spl bo cx
17 $y_2w^a$
18 $y^2$ cv

Chromosome 2

19 b
20 Bl $L^2$/SM5, Cy $lt^v$ $sp^2$
21 bw
22 cl
23 cn
24 cn bw
25 dp
26 ltd
27 pr
28 px
29 vg

Chromosome 3

30 $cu_{11}$
31 $e^3$
32 $gl^3$
33 Gl Sb/LVM
34 $kar^2$
35 Ly Sb/LVM
36 $p^b$
37 se
38 ss
39 st

Chromosome 4

40 ci $ey^R$

Multichromosomal

41 $w^a_{a2}$; bw
42 $w_2$; $bw_a$ $a$ $w^{ch}$ spl;
43 $y^2$ su-$w^a$ $w^a$ Cy Ubx/Xa
44 $v^1$; st bw

Translocation

45 T(2;3) Xa/Sb $Ubx^{130}$

Attached-X

46 y f x $pn^2_2$ $w^{a2}$
47 y f x $pn^2_{a2}$ spl $sn^3$
48 y f x $w^{a2}_{55c26}$ $rb^5$
49 y w f x $w_{a2}$ $g^2$
50 y w f x $w_{a3}$ rb cx
51 y w f x $y^2$ su-$w^a$ $w^a$

## CLEVELAND, OHIO: FENN COLLEGE

### Wild Stocks

1 Oregon-R

### Chromosome 1

2 $f^{36a}$
3 f B/y f:=
4 M(1)n/FM6, $y^{31d}$ $sc^{8}$ dm B

5 oc $ptg^{3}$/ClB
6 y w $sn^{3}$ $f^{36a}$
7 w

### Chromosome 2

8 Bl/In(2LR) dp
9 $hk$
10 $L^{2}$
11 vg

### Chromosome 3

12 H/In(3R)hp, hp
13 Pr/In(3R)C, $e_{101}$
14 Sb/In(3LR)Ubx
15 ss

### Chromosome 4

16 $ey^{2}$
17 $sv^{n}$

### Special Stocks

18 f B M(1)n/+
19 f B/f B; $ey^{2}/ey^{2}$
20 f B/f B; $L^{2}/L^{2}$
21 f BB/f BB; $ey^{2}/ey^{2}$
22 f BB/f BB; $L^{2}/L^{2}$
23 f(Bar reverted)
24 $f^{36a}$ B

## JOHNSON CITY, TENNESSEE: EAST TENNESSEE STATE UNIVERSITY
### Department of Biology

### Wild Stocks

1 Collected near Johnson City and maintained in mass cultures since November 26, 1962.
2 Stock from pair-mating matings from mass culture above.

## EMPORIA, KANSAS: KANSAS STATE TEACHERS COLLEGE

### Wild Stocks

Oregon-R
Canton-S

### Chromosome 1

y $ct^{6}$ $ras^{2}$ f
$w_{a}$
$w_{h}$
w $sn^{3}$ m
$lz^{37h}$

$ras^{2}$
v
m
Muller-5

### Chromosome 2

al b c $sp^{2}$
dp
b
b vg
cn

vg
$L^{2}$
bw

### Chromosome 3

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

### Chromosome 4

$ey^{2}$
pol

### Multi-chromosomal

v; bw
Cy/Pm; $Ubx^{130}$/Sb

## CARBONDALE, ILLINOIS: SOUTHERN ILLINOIS UNIVERSITY
### Department of Zoology

Note: All stocks originated from Department of Biological Sciences, Purdue University, Lafayette, Indiana. Numbers in parentheses represent old stock numbers.

### Wild Stocks

1. E [W-1] I-9 [55i] (W-101)

### Chromosome 1

2. y w (A-4)
3. sc cv v f (A-9)
4. w (A-12)
5. v (A-18)
6. B (A-20)

### Chromosome 2

7. vg (C-16)
8. bw (C-19)
9. b (C-80)
10. B (C-82)

### Chromosome 3

11. se (D-4)
12. $st^{11}$ (D-7)
13. $e^{s}$ (D-12)

### Chromosomes 1, 2

14. v; bw (H-6)

### Chromosomes 1, 3

15. w; se (H-9)

### Chromosomes 2, 4

16. Cy/Pm; pol (C-60)

Wild Stocks

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

Normal X Chromosome Stocks

b-1 B/y f:=
b-2 car bb
b-3 Co/y w f
b-4 $cx^{a}$ $t^{tg}$/FM1, $y^{31d}$ $sc^{8}$ $w^{s}$ $lz^{s}$ B
b-5 dow/FM6, $y^{31d}$ $sc^{8}$ dm B
b-6 ec dx
b-7 f
b-8 f BB/y f:=
b-9 f fu/ClB
b-10 fa
b-11 fa $fa^{no}$ $sn^{3}$
b-12 fa $N^{J24a}$ $Hw\ m^{2}$/In(1)dl-49, y
b-13 fa $N^{J24c}$ $sn^{3}$/In(1)dl-49, y Hw $m^{2}$
b-14 fa rb
b-15 fa spl $sn^{3}$
b-16 $fa^{no}$
b-17 $fa^{no}$ spl
b-18 $fu^{59}$/y f:=
b-19 l(1)J1 $sc^{J1}$/Del(1)24(Muller)
b-20 l(1)J1 $y^{259}$ y/S-5/$y^{+Y}$
b-21 m f car/y w f:=
b-22 $ma\text{-}l^{bz}$
b-23 ma-l
b-24 $N^{264\text{-}40}$/In(1)dl-49, y $Hw\ m^{2}\ g^{4}$
b-25 $N^{264\text{-}109}$/In(1)dl-49, y $Hw\ m^{2}\ g^{4}$
b-26 $N^{Co}$/In(1)dl-49, y Hw $m^{2}$
b-27 nd
b-28 nd rb
b-29 $ptg^{3}$ v $m\ g^{2}$ sd f/y f:=
b-30 pn
b-31 $ras_{2}dy$
b-32 rst/FM1, $y^{31d}$ $sc^{8}$ $w^{a}$ $lz^{s}$ B
b-33 rux/FM6, $y^{31d}$ $sc^{8}$ dm B
b-34 s
b-35 sc cv v f B/y f:=
b-36 sc ec $cy\ ct^{6}$ v $g^{4}$/In(1)dl-49, y Hw $m^{2}$ g
b-37 sc ec cv $ptg^{3}$ v/y v f car
b-38 $sc^{s}$ z ec ct
b-39 $sn^{3}$
b-40 sp-w
b-41 spl
b-42 spl $cho^{2}$
b-43 spl dm/y f:=
b-44 spl rb
b-45 sw
b-46 v
b-47 v f $su^{w}$-f
b-48 $w^{a}$
b-49 $w^{a}$
b-50 $w^{a}$ fa
b-51 $w^{a}$ fa rb
b-52 $w^{a}$ fa spl
b-53 $w^{a}$ $fa^{no}$ rb
b-54 $w^{a}$ $fa^{no}$ spl
b-55 $w^{a}$ $fa^{no}$ spl rb/y f:=
b-56 $w^{a}$ nd rb
b-57 $w^{a}$ spl
b-58 $w^{ch}$ spl rb
b-59 $w^{e}$ rb/y f:=
b-60 $w^{e}$ $bb^{1}$/y f:=/$B^{S}Y$
b-61 $w^{t}$ dy/y w f
b-62 $w^{t}$ fw
b-63 y
b-64 y ac sc pn/y f:=
b-65 y ac sc pn w rb $cm\ ct^{6}$ $sn^{3}$ $ras_{2}$ $y^{S1}$ dy $g^{2}$ f car/Ins(1)$sc^{S1}$, dl-49, $sc^{S1}$ v f car/$B^{S}Y$
b-66 y B/y f:=
b-67 y $bb^{13a}$/y w/$y^{+Y}$
b-68 y $bb^{174}$/y w/$y^{+Y}$
b-69 y $bb^{1158}$/y w/$y^{+Y}$
b-70 $y_{+}\ bb^{1452}$/$y^{2}$ su-$w^{a}$ $w^{a}$ bb/$y^{+Y}$
b-71 $y_{+}\ bb^{1456}$/$y^{2}$ su-$w^{a}$ $w^{a}$ bb/$y^{+Y}$
b-72 y cv v f
b-73 y cv v f car
b-74 y $f_{451}$
b-75 y l/FM6, $y^{31d}$ $sc^{8}$ $dm^{S1L,8R}$ B
b-76 y Hw/Ins(1)$sc^{S1+8}$,S,$sc^{8}$, $sc^{264\text{-}47}$ $w^{a}$ B (RM)
b-77 y $N^{264\text{-}103}$/In(1)dl-49, y Hw $m^{2}\ g^{4}$
b-78 y $N^{264\text{-}107}$/In(1)dl-49, y Hw $m^{2}\ g^{4}$
b-79 y $sc_{3}w^{col}$ spl f/In(1)$rst^{3}$, rst f
b-80 y w $bb^{no}$
b-81 y w $fa^{no}$ $sn^{3}$
b-82 y w fa spl
b-83 y $w_{a}$ spl $sn^{3}$
b-84 y $w^{a}$
b-85 y $w^{a}$ m f car
b-86 $y_{2}w^{a}$ spl rb
b-87 $y_{2}$ $cho^{2}$
b-88 $y_{2}$ cv v f
b-89 $y^{2}$ spl
b-90 $y_{2}$ v $ma\text{-}l^{bz}$
b-91 $y_{2}$ $w^{a}_{cf}$ w/y f:=
b-92 $y_{2}$ w
b-93 y

II Chromosome Stocks

c-1 a px or
c-2 al b c $sp^{2}$
c-3 al dp b pr c px sp
c-4 al dp b pr Bl c $px^{rev}$ sp/SM1, $al^{2}$ Cy $sp^{2}$
c-5 b cn c bw
c-6 b pr c px sp
c-7 Bl $L^{v}$/SM5, $al^{2}$ Cy $lt^{v}$ $sp^{2}$
c-8 $bw_{D}$
c-9 bw
c-10 cn bw
c-11 d
c-12 da/Ins(2L+2R)Cy, $Cy^{txI}$
c-13 dp Sp lys rc pr/dp Cy, Ins 0 pr $cn^{2}$
c-14 lt $stw^{3}$
c-15 lys
c-16 lys $rc_{2}$
c-17 $lys_{2}$ $rc^{2}$/$dp^{txI}$ Cy, Ins 0 pr $cn^{2}$
c-18 M(2)S10/Ins(2L+2R) Cy, Cy pr $Dp(2;2)41^{2}$
c-19 ms cn bw/$dp^{txI}$ Cy pr Bl lt $cn^{2}$ $L^{4}$ $sp^{2}$
c-20 net al ex ds S ast shv hq rub/SM1, $al^{2}$ Cy $sp^{2}$
c-21 $nw^{D}$/Ins(2LR)$Px^{4}$
c-22 $Pin_{Yt}$/Ins(2L+2R)Cy, Cy
c-23 $Pu^{2}$/SM1, $al^{2}$ Cy $sp^{2}$
c-24 px sp $Pin^{2}$/SM1, $al^{2}$ Cy $sp^{2}$
c-25 rc pr
c-26 rc J/Ins(2L+2R)Cy, Cy Bl L
c-27 $sp^{2}$ bs
c-28 Sp J Pin/SM5, $al^{2}$ Cy $lt^{v}$ $sp^{2}$
c-29 Sp $lys_{2}d$/SM1, $al^{2}$ Cy sp
c-30 Sp lys d J/Ins (2L+2R)Cy, Cy Bl L
c-31 Sp lys rc J/SM1, $al^{2}$ Cy $sp^{2}$
c-32 Sp $rc^{2}$/Ins(2L+2R) Cy, Cy Bl L
c-33 $sp^{2}$ $bs_{2}$ L
c-34 Sp $rc^{2}$/Ins(2L+2R) Cy, Cy Bl L

c-35 spd$^{fg}_{3}$
c-36 stw$^{2}$ c
c-37 Tft/SM1, al$^{2}$ Cy sp$^{2}$
c-38 vg$_{U}$
c-39 vg$^{2}$/In(2L)t + In(2R) Cy, Roi bw sp or

III Chromosome Stocks

d-1 Bd$^{G}_{3}$/In(3R)C, 1(3)a
d-2 bx$^{34e}$
d-3 ca
d-4 ca$^{K}$-pn
d-5 ca$^{nd}$/In(3LR)Ubx$^{130}_{s}$, M(3)1 Ubx$^{130}$ e$^{s}$
d-6 ca$^{nd}$/TM3, Sb Ser
d-7 cu kar
d-8 cy-c sbd$^{2}$
d-9 D$^{3}$ H/In(3L)P, Mé
d-10 e$^{s}$
d-11 e$^{s}$
d-12 Gl Sb/LVM
d-13 h$_{2}$th st ry$^{3}$ ss$^{a}$
d-14 H$^{2}$/Tp(3R)Vno, Vno
d-15 jvl
d-16 p$^{p}$ bx sr e$^{s}$
d-17 p$^{p}$ Ki
d-18 Pr$^{K}$/In(3R)C, e
d-19 Pr$^{K}$ Dp/Ins(3L+3R)P
d-20 R Ly/In(3L)P, gm
d-21 red
d-22 ru
d-23 ru h th st cu sr e$^{s}$ ca
d-24 ru h th st cu sr e$^{s}$ Pr ca$_{2}$/TM1, Mé ri
d-25 ry$^{2}$
d-26 se
d-27 se ss k e$^{s}$ ro
d-28 sr$^{a}$ gl
d-29 ss$^{a}$
d-30 st
d-31 st C3G ca/In(3LR)Ubx 130, Ubx$^{130}_{s}$ e$^{s}$
d-32 st in$_{2}$ri p$^{p}$
d-33 st ry$^{2}$ sr$^{s}$ e$^{s}$
d-34 st$_{2}$sbd e$^{s}$ ro ca
d-35 su$^{2}$-Hw bx bxd/TM1, Mé ri
d-36 ve h th

IV Chromosome Stocks

e-1 bt$^{D}_{2}$
e-2 bt$^{D}_{2}$/ci$^{D}$
e-3 Ce/spa$^{Cat}$
e-4 ci ey$^{R}$
e-5 ci gvl ey$^{R}$ sv$^{n}_{Cat}$
e-6 ci$^{D}$ gvl$^{D}$ spa/ci$^{D}$
e-7 ci$^{n}$/ey$^{D}$
e-8 sv$^{n}$
e-9 spa
e-10 pol

Multichromosomal Stocks

f-1 v/Y$^{bb}_{st}$; (1;Y)
f-2 br$^{3}$ dx$^{m4}$; ed Su$^{2}$-dx (1;2)
f-3 In(1)w$^{a}$; E-Var7/Ins(2L+2R)Cy, Cy (1;2)
f-4 v; In(2R)bw$^{VDel}$/Ins(2LR)SM1, al$^{2}$ Cy sp$^{2}$ (1;2)
f-5 v f; In(2R)bw$^{VDel}$/SM1, al$^{2}$ Cy sp$^{2}$ (1;2)
f-6 y/y f:=; dp lys rc pr (1;2)
f-7 FMA2, y$^{2}$/+; b pr Elp/SM1, al$^{2}$ Cy sp$^{2}$ (1;2)
f-8 y$^{2}$ cho$^{2}$; rc$_{2}$ (1;2)
f-9 In(1)AM, y$^{2}$/FM6, y$^{31d}$ dm B; SM1, al$^{2}$ Cy sp$^{2}$/Bl; Tp(3R)Vno, Vno/In(3LR)Ubx$^{130}$, Ubx$^{130}_{s}$ e$^{s}$ (1;2;3)
f-10 spl$_{4}$rb; Ins(2LR) SM1, al$^{2}$ Cy sp$^{2}$/In(2LR)Pm, al$^{130}$ ds$^{33k}$ lt$^{-}$ bw$^{V1}$; C Sb/In(3LR) Ubx$^{130}$, Ubx$^{130}_{s}$ e$^{s}$ (1;2;3)
f-11 y; Ins(2L+2R)Cy, Cy/In(2LR)Pm, al$^{4}$ ds$^{33k}$ lt$^{-}$ bw$^{V1}$; Sb/In(3L)D, D(1;2;3)
f-12 FM6, y$^{31d}$ sc$^{8}$ dm B/In(1)AM, y$^{2}$; pr Bl/SM1, al$^{2}$ Cy sp$^{2}$; Tp(3R)Vno, Vno/In(3LR)Ubx$^{130}$, Ubx$^{130}_{s}$ e$^{s}$; pol (1;2;3;4)
f-13 y/y f:=; bw; st; pol (1;2;3;4)
f-14 v; In(2R)bw$^{VDel}$/SM1, al$^{2}$ Cy sp$^{2}$; M-4/ey$^{D}$ (1;2;4)
f-15 y$^{2}$ v; bw; ci$^{D}_{D}$/ey$^{D}$ (1;2;4)
f-16 y$^{2}$ v; bw; ey$^{D}$/simulans-4 (1;2;4)
f-17 Ins(1)sc$^{7}_{L}$AM, sc$^{7}_{R}$/y f:=; bx sr e (1;3)
f-18 Ins(1)sc$^{S1L}$, S, sc$^{S1+8}_{8R}$, sc$^{S1+8}$ w$^{a}$ B/y f:=/Y; In(3R)112, bx (1;3)
f-19 Ins(1)sc$^{8L}_{+}$, dl-49, sc$^{S1R}_{+}$, y$^{31d}$ sc$^{-}$ v f/y w$^{a}$ N$^{Nic}$/y Y w$^{+}$; ca K-pn (1;3)
f-20 +/y; Tp(3R)Vno, Vno/In(3LR)Ubx$^{130}$, Ubx$^{130}_{s}$ e$^{s}$ (1;3)
f-21 Ins(1)sc$^{Sl}$, dl-49, sc$^{Sl}$ v f car/y f:=; bx sr e$^{s}$; pol(1;3;4)
f-22 b pr Bl/SM1, al$^{2}$ Cy sp$^{2}$; Tp(3R)Vno, Vno/In(3LR) Ubx$^{130}$, Ubx$^{130}_{s}$ e$^{s}$ (2;3)
f-23 bw; st (2;3)
f-24 Ins(2L+2R)Cy, Cy/In(2LR)Pm, al$^{4}$ ds$^{33k}$ lt$^{-}$ bw$^{V1}$; Sb/In(3LR)DcxF, D$^{rev}_{2}$ (2;3)
f-25 SM1, al$^{2}$ Cy sp$^{2}$/In(2LR)Pm, dp b ds$^{33k}$; Sb/Ins(3LR)Ubx$^{130}$, Ubx$^{130}_{s}$ e$^{s}$ (2;3)
f-26 bw; ci sv$^{n}_{D}$ (2;4)
f-27 bw; sim-4/ci$^{D}$ (2;4)

Inverted Chromosomes

g-1 In(1)AB, y f
g-2 In(1)AM/T(1;3)65, y
g-3 In(1)B$^{M1}$, cm/y f:=
g-4 In(1)Cl, sc l t$^{2}$ v sl B (ClB)
g-5 In(1)dl-49, y fa$^{n}$
g-6 In(1)dl-49, y Hw m$^{2}$
g-7 In(1)dl-49, y Hw m$^{2}$ g$^{4}$
g-8 In(1)dl-49, y v f car/y f:=
g-9 In(1)dl-49, ty-l bb$^{1}$/y v f car
g-10 In(1)dl-49, y w B
g-11 In(1)dl-49, y w lz$^{s}$/y f:=
g-12 In(1)dl-49, y w lz$^{s}$ bb/In(1)sc$^{L8}$, sc$^{L8}$ car m w$^{a}$
g-13 In(1)EN, y/y f:=

g-14 In(1)EN, y bb/sc$^{8}$·Y
g-15 In(1)rst$^{3}$, rst$^{3}$
g-16 In(1)rst$^{3}$, w$^{48-3}$ rst$^{3}$ v f/y f:=
g-17 In(1)rst$^{3}$, y rst$^{3}$ car bb?/RM, Ins(1) sc$^{S1L}$,S,sc$^{8R}$,y$^{31d}$ sc$^{S1+8}$ w$^{a}$ B (=M-5)
g-18 In(1)sc$_{4}$, y sc$_{4}$
g-19 In(1)sc$_{4}$, y sc$_{4}$ cv v f
g-20 In(1)sc$_{4L}$, y sc$_{8R}$ cv v B/y f:=/B$^{S}$Y
g-21 In(1)sc$_{4L}$, sc$_{8R}$, y sc$_{4+8}$ cv v f/y f:=
g-22 In(1)sc$_{7}$, sc$_{7}$, y sc$_{4+8}$ cv v B/y f:=/B$^{S}$Y
g-23 In(1)sc$_{8}$, sc$_{8}$
g-24 In(1)sc$_{8}$, sc$_{8}$
g-25 In(1)sc$_{8}$, sc$_{8}$ v f car
g-26 In(1)sc$_{8}$, sc$_{8}$ cv y f/In(1)dl-49, y Hw m$^{2}$ g$^{4}$
g-27 In(1)sc$_{8}$, y$^{31d}$ sc$^{8}$ w$^{a}$
g-28 In(1)sc$^{L8}$, sc$^{L8}$ w$^{a}$ m car/In(1)dl-49, y w lz$^{s}$ bb
g-29 In(1)sc$^{L8L}$,sc$^{S1R}$, sc$^{L8}$ w$^{a}$ m car/In(1)dl-49, y w lz$^{s}$ bb
g-30 In(1)sc$_{S1L}$,sc$_{4R}$, sc$_{S1}$ cv v B/y w/sc$^{8}$·Y
g-31 In(1)sc$_{S1L}$,sc$_{8R}$, sc$_{S1+8}$ y f B/y f:=/B$^{S}$Y
g-32 In(1)sc$_{m4}$,sc$_{m4}$, sc$_{m4}$ v B/y f:=/B$^{S}$Y
g-33 In(1)w$_{m4L}$, w$_{m4L}$
g-34 In(1)w$^{N}$$^{264-84R}$, y sn/FM3, y$^{31d}$ sc$^{8}$ dm B l/B$^{S}$Y y$^{+}$
g-35 In(1)w$_{m4L}$,rst$_{3P}$, y w$^{-}$ rst$^{3}$
g-36 In(1)y$_{4}$, y$_{4}$ B$_{3P}$
g-37 In(1)y$_{4}$, y$_{4}$
g-38 In(1)65, y f/B$^{S}$Y
g-39 In(1)481(12E-F;14B), y bb$^{l481}$/FM6
g-40 In(1H)59(3-4), y l$^{59}$/y w/sc$^{8}$·Y
g-41 In(1H)132(4E), y l$^{132}$/y w/sc$^{8}$·Y
g-42 In(1H)227(1F), y l$^{227}$/y w/sc$^{8}$·Y
g-43 In(1H)231(1C-D), y l$^{231}$/y w/sc$^{8}$·Y
g-44 Inp(1)sc$^{V1}$, v·sc$^{V1}$ y$^{+}$/Ins(1)sc$^{8}$, dl-49, y$^{31d}$ v f B
g-45 Inp(1)sc$^{V1}$, y v·sc$^{V1}$ y$^{+}$/y$^{2}$ su-w$^{a}$ w$^{a}$ bb/ sc$^{8}$·Y
g-46 Inp(1)139, w$^{m139}$ rst$^{m}$ l$^{139}$/y w f/Y/Y
g-47 Ins(1)dl-49,B$^{M1}$ sc$^{4L}$,S,sc$^{8R}$ v sc$^{4+8}$ w$^{a}$ B (S-5)/
g-48 Ins(1)sc$^{8}$ l$^{259}$ y/sc$^{8}$·Y
g-49 Ins(1)sc$_{8}$,AM, sc$_{7}$
g-50 Ins(1)sc$_{8}$,dl-49, y$^{31d}$ sc$^{8}$ w$^{a}$ lz$^{s}$ B (FM1)
g-51 Ins(1)sc$_{8}$,dl-49, sc$_{8}$ v f/y f:=
g-52 Ins(1)sc$_{8}$,dl-49, y$^{31d}$ sc$^{8}$ v f B
g-53 Ins(1)sc$_{8}$,dl-49,3C-4EF, w dm f (FM4)/y f:=
g-54 Ins(1)sc$_{8}$,dl-49,3C-4EF, y$^{31d}$ sc$^{8}$ dm B (FM4)
g-55 Ins(1)sc$^{8}$,dl-49,3C-4EF,15DE-20, y$^{31d}$ sc$^{8}$ dm B (FM6)
g-56 Ins(1)sc$_{8}$(c.o. X J-3),S, y$^{-}$ ac$^{-}$ sc$^{8}$ w$^{a}$/sc$^{8}$·Y
g-57 Ins(1)sc$^{8L}$,dl-49,sc$^{S1R}$, y sc$^{-}$ v B f (Biny)/ l$^{259}$ y w$^{m}$ f/sc$^{8}$·Y
g-58 Ins(1)sc$_{S1L}$,dl-49,sc$_{4R}$ y$^{S1}$ f car
g-59 Ins(1)sc$_{S1L}$,AB,sc$^{8R}$,sc$_{S1+8}$ w$^{a}$ car/y f:=
g-60 Ins(1)sc$_{S1L}$,dl-49,sc$^{8R}$,sc$_{S1+8}$ v B car/y f:=
g-61 Ins(1)sc$^{S1L}$,S,sc$^{8R}$, sc$^{S1+8}$ w$^{a}$ B (M-5)
g-62 Ins(1)sc$^{S1L}$,S,sc$^{8R}$, sc$^{S1+8}$ w$^{a}$ B bb$^{1}$/y f:=
g-63 Ins(1)sc$^{S1L}$,S,sc$^{8R}$, y$^{2}$ sc$^{-}$ w$^{a}$ B
g-64 Ins(1)y$^{3PL}$,S,sc$^{S1R}$, y$^{19i}$ ac$^{-}$ sc$^{-}$; Cy/sc$^{8}$
g-65 In(2LR)lt$^{m3}$(60D)/SM5
g-66 In(2LR)lt$^{m12}$(60D)/SM5
g-67 In(2LR)Pm$^{2}$/Df(2)M33a$^{p}$
g-68 In(3LR)sep, sep ri p$^{p}$
g-69 In(3R)18/Xa
g-70 In(3R)112
g-71 Tp(3R)Vno, Vno/H$^{2}$

Translocated Chromosomes

h-1 T(1;B$^{S}$Y$_{S}^{L}$)3C no. 107
h-2 T(1;B$^{S}$Y$_{S}^{L}$)3C no. 111
h-3 T(1;B$^{S}$Y$_{S}^{L}$)3C no. 114
h-4 T(1;B$^{S}$Y$_{S}^{L}$)3C no. 128
h-5 T(1;B$^{S}$Y$_{S}^{L}$)3C no. 140
h-6 T(1;B$^{S}$Y$_{S}^{L}$)3C no. 164
h-7 T(1;B$^{S}$Y$_{S}^{L}$)3D no. 104
h-8 T(1;B$^{S}$Y$_{S}^{L}$)5D no. 108
h-9 T(1;B$^{S}$Y$_{S}^{L}$)6E no. 149
h-10 T(1;B$^{S}$Y$_{S}^{L}$)7D no. 102
h-11 T(1;B$^{S}$Y$_{S}^{L}$)7D no. 156
h-12 T(1;B$^{S}$Y$_{S}^{L}$)9F no. 124
h-13 T(1;B$^{S}$Y$_{S}^{L}$)11A no. 129
h-14 T(1;B$^{S}$Y$_{S}^{L}$)12A + T(1;3)3F;69C no. 143
h-15 T(1;B$^{S}$Y$_{S}^{L}$)13A no. 152
h-16 T(1;B$^{S}$Y$_{S}^{L}$)13E no. 142
h-17 T(1;B$^{S}$Y$_{S}^{L}$)14A no. 240
h-18 T(1;B$^{S}$Y$_{S}^{L}$)15A no. 112
h-19 T(1;B$^{S}$Y$_{S}^{L}$)15D no. 125
h-20 T(1;B$^{S}$Y$_{S}^{L}$)16A no. 106
h-21 T(1;B$^{S}$Y$_{S}^{L}$)16E no. 118
h-22 T(1;B$^{S}$Y$_{S}^{L}$)18A no. 159
h-23 T(1;B$^{S}$Y$_{S}^{S}$)3F no. 150
h-24 T(1;B$^{S}$Y$_{S}^{S}$)6E no. 131
h-25 T(1;B$^{S}$Y$_{S}^{S}$)7B no. 155
h-26 T(1;B$^{S}$Y$_{S}^{S}$)8F no. 115
h-27 T(1;B$^{S}$Y$_{S}^{S}$)8F no. 147
h-28 T(1;B$^{S}$Y$_{S}^{S}$)11B no. 145
h-29 T(1;B$^{S}$Y$_{S}^{S}$)12E + T(1;3)19E;62A no. 134
h-30 T(1;B$^{S}$Y$_{S}^{S}$)14D + T(1;4)9C;101 no. 116
h-31 T(1;B$^{S}$Y$_{S}^{S}$)19E no. 101
h-32 T(1;B$^{S}$Y$_{S}^{S}$)19E no. 133
h-33 T(1;B$^{S}$Y$_{S}^{S}$)19E no. 141
h-34 T(1;B$^{S}$Y$_{S}^{S}$)19F no. 103
h-35 T(1;B$^{S}$Y$_{S}^{S}$)19F no. 105
h-36 T(1;B$^{S}$Y$_{S}^{S}$)19F no. 119
h-37 T(1;B$^{S}$Y$_{S}^{S}$)19F no. 132
h-38 T(1;B$^{S}$Y$_{S}^{S}$)19F no. 137
h-39 T(1;B$^{S}$Y$_{S}^{S}$)19F no. 151
h-40 T(1;B$^{S}$Y$_{S}^{S}$)20A no. 122
h-41 T(1;B$^{S}$Y$_{L}^{+}$;3)6F;86D no. 121
h-42 T(1;y$^{+}$Y$^{L}$)1F no.21

h-43 T(1;$y^+Y^L$)3E no. 10
h-44 T(1;$y^+Y^L$)4C no. 16
h-45 T(1;$y^+Y^L$)11A no. 4
h-46 T(1;$y^+Y^L$)11A no. 20
h-47 T(1;$y^+Y^L$)11D + T(1;3)14F;72 no. 5
h-48 T(1;$y^+Y^L$)16F no. 1
h-49 T(1;$y^+Y^S$)17A no. 19
h-50 T(1;$y^+Y^S$)2C;19F no. 9
h-51 T(1;$y^+Y^S$)5E;11F;19F no. 2
h-52 T(1;$y^+Y^S$)3E no. 3
h-53 T(1;$y^+Y^S$)11D no. 6
h-54 T(1;$y^+Y^S$)14F + T($y^+Y^L$;2)36C no. 7
h-55 T(1;$y^+Y^S$)19E no. 22
h-56 T(1;$y^+Y^S$)19F no. 14
h-57 T(1;2)459, y $l^{459}$/FM6, $y^{31d}$ $sc^8$ dm B
h-58 T(1;2)B1d/ClB
h-59 T(1;2)$sc^{19}$/y f:=; fes $sc^{19i}$ b pr/Cy dp$^{Th}$ pr
h-60 T(1;2) + T(2;3)Dinky/y f:=
h-61 T(1;2H)25(20), y $l^{25}$/FM6
h-62 T(1;2LH)150(16-17), y $l^{150}$/FM6
h-63 T(1;2LH)219(10A), y $l^{219}$/FM6
h-64 T(1;2RH)75(20), y $l^{75}$/FM6
h-65 T(1;2RH)135(18-19), y $l^{135}$/FM6
h-66 T(1;2;$y^+Y^L$)7B;39 no. 17
h-67 T(1;2;$y^+Y^S$)7B;50 no. 12
h-68 T(1;2H?;$B^SY^L$)3C no. 109
h-69 T(1;2;3)220(14A;50A;75), y $l^{220}$/FM6
h-70 T(1;2;3;4)454, y $l^{454}$/FM6
h-71 T(1;3H)361(20), y $l^{361}$/FM6
h-72 T(1;3H)453(12D, y $l^{453}$/FM6
h-73 T(1;3H)463(20), y $l^{463}$/FM6
h-74 T(1;3LH)163(17A-B), y $l^{163}$/FM6
h-75 T(1;3LH)455(3C), y $l^{455}$/FM6
h-76 T(1;3RH)3(3-4), y $l^3$/FM6
h-77 T(1;3RH)129(18B), y $l^{129}$/FM6
h-78 T(1;3;$B^SY^L$)15E;74 no. 144
h-79 T(1;3;$B^SY^S$)17A;94 no. 161
h-80 T(1;3;$B^SY^S$)10A;97A no. 154
h-81 T(1;3;$B^SY^S$)19F;85 no. 127
h-82 T(1;4)A7, y w/$y^2$ su-$w^a$ $w^a$ bb
h-83 T(1;4)A13(18C5)
h-84 T(1;4)A17(8A2)/y f:=
h-85 T(1;4)A17(8A2), y cv/y f:=
h-86 T(1;4)A19
h-87 T(1;4)A20/y f:=
h-88 T(1;4)$B^S$(16A1), $B^S$/y f:=
h-89 T(1;4)$B^S$(16A1), $B^2$ car/y f:=
h-90 T(1;4)$B^S$(16A1), $y^2$ cv v $B^S$ car/y f:=
h-91 T(1;4)e15
h-92 T(1;4)h4
h-93 T(1;4)h6
h-94 T(1;4)11(15A), y $l^{11}$ car/$sc^8$·Y
h-95 T(1;4)$w^{m5}$(3C3), $w^{m5}$
h-96 T(1;4)(4C3)/y f:=
h-97 T(1;4)(13B8-9)/y f:=
h-98 T(XY$^L$·$Y^S$;4)$B^S$(16A1), $X^D$,$B^SY^L$·$Y^S$/y v bb:=/0
h-99 T($Y^S$;4)
h-100 T(2;3)$bw^{V4}_{V5}$, $bw^{V4}_{V5}$/Cy
h-101 T(2;3)$bw^{V4}_{VDe3}$, $bw^{V4}_{V5}$/Cy
h-102 T(2;3)$bw^{m7}$ Ubx bxd/In(3R)C
h-103 T(2;3)$lt^{m7}$(98C), $lt^{m7}$/SM5, $al^2$ Cy $lt^v$ $sp^2$
h-104 T(2;3)$S^M$, $S^M$ Cy C3G Sb Ubx/st$^{130}$ C3G ca
h-105 T(2;3)Xa/In(3LR)Ubx$^{130}$, Ubx$^{130}$ $e^s$
h-106 T(2;4)$ast^v$/In(2L+2R)Cy, Cy
h-107 T(3;4)A2/$ci^D$
h-108 T(3;4)e/$ci^D$
h-109 T(3;4)86D, $bx^{34e}$ $e^4$
h-110 T(3;4)89E, ss bs bxd/$ey^D$
h-113 Ti(3;1)05/y f:=/TM3/Tp(3R)Vno, Vno
h-114 Ti(3;1)05, D/y f:=/$B^SY^{m5}$
h-115 Ts(1;4)$w^{m5}$, 9A, $w^{m5}$/FM4, $y^{31d}$ w dm B
h-116 Ts(1;4)$w^{m5}$, $B^S$, w v $B^S$/FM4, $y^{31d}$ w dm f

Closed X Chromosomes

i-1 $X^c$, y/y f:=/y Y (examined cytologically September, 1962)
i-2 $X^{c2}$ (ET), +/M-5
i-3 $X^{c2}$ (KOA), +/M-5
i-4 $X^{c2}$, $w^{col}$ 491/In(1)d1-49, y Hw $m^2$ $g^4$
i-5 $X^{S1}$ $w^{spont}$ $v^a$ f/RM, Ins(1)$sc^{S1L}$,S,$sc^{8R}$, ss $w^a$ $sc^8$/Y
i-6 $X^{c2}$, y B/y f:= (examined cytologically September, 1962)
i-7 $X^{c2}$, $y^{49}$/RM, y w/$y^+$Y (examined cytologically September, 1962)
i-8 $X^{c2}$, In(1)$w^{vc}$ (stable), $w^{vc}$/Ins(1)$sc^7$, $sc^{AM}$
i-9 $X^{c2}$, In(1)AB, +/y f:=

X Chromosomes with A Y Arm Attached

j-1 X·$Y^L$(A-2), y w·$Y^L$/y/Y"
j-2 X·$Y^L$(C-2), y cv v f car bb$^-$·$Y^L$/RA, (ND-27) v f/Y"
j-3 X·$Y^L$(C-2), y w bb$^-$·$Y^L$/y/Y"
j-4 X·$Y^L$(U-8e), y w·$Y^L$/y/Y"
j-5 X·$Y^L$, y cv v f car·$Y^L$/y/Y"
j-6 X·$Y^L$, y v f bb(bb$^+$)$Y^L$/y f:=/$sc^{V1}$·$Y^S$
j-7 X·$Y^L$, In(1)$sc^{8L}$,$EN^R$, $y^+$ car f v cv y·$Y^L$/y/Y"
j-8 X·$Y^L$(K-7), In(1)$sc^{8L}$,$EN^R$, $y^+$ f v cv y·$Y^L$/y/Y"
j-9 X·$Y^L$(P-8b), In(1)$sc^{8L}$,$EN^R$, $y^+$ f y·$Y^L$/y/Y"
j-10 X·$Y^S$(A-3), y w·$Y^S$/y v f/$Y^{Lc}$
j-11 X·$Y^S$(U-8C), y w·$Y^S$/y v f/$Y^{Lc}$
j-12 X·$Y^S$(P-8b), In(1)$sc^{8L}$,$EN^R$, $y^+$ f y·$Y^S$/y v f/$Y^{Lc}$
j-13 $Y^S$X·(FR-1), $Y^S$ y cv v f/y f:=/Y
j-14 $Y^S$X·(FR-1$^L$,Inp(1)$sc^{V1R}$), $Y^S$ y cv v f ·$y^+$/$Y^{Lc}$

j-15 $Y^S X\cdot$(FR-1), $Y^S$ y m f car/y v f:=/$Y^{Lc}$
j-16 $Y^S X\cdot$(P-0), In(1)EN, $Y^S$ y/y w/Y
j-17 $Y^S X\cdot$(P-7), In(1)EN, $Y^S$ y f/y v f/Y
j-18 $B\,Y^S X\cdot$(FR-1, $3a^R$) $B\,Y^S$ y cv v f·/ y v f/$Y^{Lc}$
j-19 $y^+ Y^S X\cdot$(FR-1, $3a^R$) $y^+ Y^S$ y cv v f·/ y f:=/Y?
j-20 $y^{31d} Y^S X\cdot$(FR-1, $3a^R$) $y31d\,Y^S$ y cv f·/ y f:=/Y

Compound X Chromosomes

k-1 RA, l(1)J1 $sc^{J1}$--In(1)$sc^8$·/$XY^L\cdot Y^S$, l(1)$J1^{259}$ y w $Y^L\cdot Y^S$/y $sc^8\cdot Y$
k-2 RA (ND-27), $sc^8$ v f--In(1)$sc^8$, f v $sc^8$·/$X\cdot Y^L$(C-2), $y^+$ cv v f car bb·$Y^L$/Y"
k-3 RA, y--In(1)$sc^8$·/$Y^S X\cdot Y^L$, In(1)EN, $Y^S$B y·$Y^L$/$y^+$ $ac^+\cdot Y^L$
k-4 RA, y ac sc pn--In(1)$sc^8$·
k-5 RA, y--In(1)$sc^{8L}$, $EN^R\cdot Y^L$/$Y^S X\cdot Y^L$, In(1)EN, $Y^S$B y·$Y^L$/$y^+$ $ac^+\cdot Y^L$
k-6 RA, In(1)AB, y--In(1)$sc^8$·/$Y^S X\cdot Y^L$, In(1)EN $Y^S$B y·$Y^L$/$y^+$ $ac^+\cdot Y^L$
k-7 RA (Muller), In(1)dl-49, y w f--In(1)$sc^8$? f $sc^8$·/$XY^S$,y w·$Y^L$/$Y^L$·bb $ac^+$ $y^+$
k-8 RA (Muller), $In(1)dl\text{-}49^a$, $y\,w\,f\text{--}In(1)sc^{8L}$?, $f\,w\,sc^{8R}$·Dp(1;2R)$w^{51b7}$ $/Y^L/In(1)w^{m4L}$, $rst^{3R}$
k-9 RA, $y^2$ $su\text{-}w^a$ $w^a$ --$Ins(1)sc^{8L}$,S,$sc^{8R}$, $B_L w^a$ $sc^8\cdot Y^S X(Y^L\cdot)$, $Y^S y(Y^L\cdot bb^+)/Y$
k-10 RA·$Y^L$, +--In(1)$sc^{8L}$,EN, y·$Y^L$ $y^+$/$Y^S X\cdot Y^L$, In(1)EN, $Y^S$B y·$Y^L$/$y^+$ $ac^+\cdot Y^L$
k-11 RM, pn/$Y^{ee}$ x $Y^S X\cdot Y^L$, y B/Y ♂♂
k-12 RM, y/$X\cdot Y^L$(A-2), y w·$Y^L$/Y"
k-13 RM, y v bb
k-14 RM, y v f/$X\cdot Y^S$,y w·$Y^S$/$ac^+$ $y^+\cdot Y^L$
k-15 RM, y v f car
k-16 RM, y w/$X\cdot Y^S$, y w·$Y^S$/$Y^L$ $bb^+$ $ac^+$ $y^+$
k-17 RM, $y^2$ $su\text{-}w^a$ $w^a$ bb/$X\cdot Y^S$, y w·$Y^S$/$Y^L$ $bb^+$ $ac^+$ $y^+$
k-18 RM, Ins(1)$sc^{8L}$,S,$sc^{8R}$, $sc^8$ $w^a$ $sc^8$/$X^L$,$w^a$ $v^a f$ $sc^{c2}$ spont
k-19 RM (13-0-15=$XY^L\cdot X$), $y^2$ $su\text{-}w^a$ $w^a$ bb $Y^L$/$y^2$ $su\text{-}w^a$ $w^a$ bb? $bb^+$/$Y^S X\cdot Y^L$, In(1)EN, $Y^S$B y·$Y^L$
k-20 RM (15-DRP=$XY^L\cdot Y^L X$), $y^2$ $su\text{-}w^a$ $w^a$ bb $Y^L$/$y^2$ $su\text{-}w^a$ $w^a$ bb? Y $bb^+$/$Y^S X\cdot Y^L$, In(1)EN, $Y^S$B f v y·$Y^L$ $y^+$
k-21 RM, In(1)EN, $Y^S$ y/0/$Y^S X\cdot Y^L$, Ins(1)EN, d1-49, $Y^S$ car f v y·$Y^L$
k-22 RM (TAX), $In(1)y^a$, w $y^a$/y v f/$Y^S X\cdot Y^L$, In(1)EN, $Y^S$B y·$Y^L$/0
k-23 TM (hw f), originally y Hw v f·$y^+$ cv f $y^+$/X·Y,y B

Attached XY Chromosomes

m-1 $XY^L\cdot Y^S$ (2-10T13 Parker), $y^2$ $su\text{-}w^a$ $w^a$ $Y^L\cdot Y^S$/y/Y
m-2 $XY^L\cdot Y^S$ (2-10T15 Parker), $y^2$ $su\text{-}w^a$ $w^a$ $Y^L\cdot Y^S$/y/Y
m-3 $XY^L\cdot Y^S$ (10809 Parker), $y^2$ $su\text{-}w^a$ $w^a$ $Y^L\cdot Y^S$/y v bb/0
m-4 $XY^L\cdot Y^S$ (112-17 Parker), $y^2$ $su\text{-}w^a$ $w^a$ $Y^L\cdot Y^S$/y v bb/0
m-5 $XY^L\cdot Y^S$ (127-29 Parker), $y^2$ $su\text{-}w^a$ $w^a$ $Y^L\cdot Y^S$/y v bb/0
m-6 $XY^L\cdot Y^S$ (129-11 Parker), $y^2$ $su\text{-}w^a$ $w^a$ $Y^L\cdot Y^S$
m-7 $XY^S\cdot Y^L$, y l(1)259 w $Y^L\cdot Y^S$/y/$Dp^a(1;f)167$
m-8 $XY^S\cdot Y^L$ (110-8 Parker), $y^2$ $su\text{-}w^a$ $w^a$ $Y^S\cdot Y^L y^+$/y v bb/0
m-9 $XY^S\cdot Y^L$ (115-9 Parker), $y^2$ $su\text{-}w^a$ $w^a$ $Y^S\cdot Y^L y^+$/y v bb/0
m-10 $XY^S\cdot Y^L$ (129-16 Parker), $y^2$ $su\text{-}w^a$ $w^a$ $Y^S\cdot Y^L y^+$/y v bb/0
m-11 $Y^S X\cdot Y^L$ (FR-$1^L$,C-$2^R$), $Y^S$ y bb·$Y^L$/$y^2$ $su\text{-}w^a$ $w^a$ bb/0
m-12 $Y^S X\cdot Y^L$ (FR-$1^L$,U-$8d^R$), $Y^S$ y $w^a$ cv y $f\cdot Y^L$/$y^2$ $su\text{-}w^a$ $w^a$ bb/0
m-13 $Y^S X\cdot Y^L$, In(1)EN, $Y^S$ B f $v_L y\cdot Y^L$ $y^+$/$y^2$ $su\text{-}w^a$ $w^a$ bb/0
m-14 $Y^S X\cdot Y^L$, In(1)EN, $Y^S$ B y·$Y^L$/$y^2$ $su\text{-}w^a$ $w^a$ bb/0
m-15 $Y^S X\cdot Y^L$, In(1)EN, $Y^S$ y·$Y^L$/$y^2$ $su\text{-}w^a$ $w^a$ bb/0
m-16 $Y^S X\cdot Y^L$, In(1)EN, $Y^+$ $v^S$cv y·$Y^L$ $y^+$/$y^2$ $su\text{-}w^a$ $w^a$ bb/0
m-17 $Y^S X\cdot Y^L$, In(1)EN, $y^S$ $Y^S$ y·$Y^L$ $y^+$/$y^2$ $su\text{-}w^a$ $w^a$ bb/0
m-18 $Y^S X\cdot Y^L$, In(1)EN, $Y^S$ y·$Y^L$ $y^S$
m-19 $Y^S X\cdot Y^L$, Ins(1)EN,$dl\text{-}49^a$, $Y^S$car f v y·$Y^L$/$y^2$ $su\text{-}w^a$ $w^a$ bb/0
m-20 $Y^S X\cdot Y^L$, Ins(1)EN,dl-49, $Y^S$ f v pn y·$Y^L$
m-21 $Y^S X(Y^L\cdot)$ (FR-$1^L$,3-$18^R$), $Y^S$ y ($Y^L\cdot bb^+$)/RA,$y^2$ $su\text{-}w^a$ $w^a$ ---M-5/Y
m-22 $Y^S X(Y^L\cdot)$ (FR-$1^L$,118-$2b^R$ no. 1), $Y^S$ y ($Y^L\cdot bb^+$)/$y^2$ $su\text{-}w^a$ $w^a$ bb/0
m-23 $Y^S XY^S\cdot$ (FR-$1^L$,174-$13^R$), $Y^S$y cv v bb $Y^L\cdot$/RA,$y^2$ $su\text{-}w^a$ $w^a$ ---M-5/Y
m-24 $Y^S XY^S\cdot Y^L$ (FR-$1^L$,115-$9^R$), $Y^S$ y cv $Y^S\cdot Y^L$ $y^+$/$y^2$ $su\text{-}w^a$ $w^a$ bb/0

Altered Y Chromosomes

n-1 $B^S Y/y^+ f:=/y^2$ su-$w^a$ $w^a$
n-2 $B^S Y y^+/y$ v; bw
n-3 $B^S Y y^+/y^+/y$
n-4 $B^S Y y^{31d}/y$ w f/$Y^S$ y cv v f
n-5 bw $Y/y^+$ v; bw
n-6 bw $Y y^+/y^+ f:=/y$ v f
n-7 $y^+ Y$ (=sc$^8$·Y)/y v/$y^+$
n-8 $y^+ Y B^S$ (Y1 III)/y w/y su-$w^a$ $w^a$ bb
n-9 $y^+ Y w^+$ (11a)/y $w^a$
n-10 $y^+ Y w^+ B^S$ (Y1I)/y $w^a$
n-11 $y^+ Y^L$·(FR-2)/RA, y--In(1)$sc^8$/$Y^S X$·$Y^L$, In(1)EN, $y^+ B^m$
n-12 Ybb$^-$/In(1)$w^{m4}$
n-13 Yma-1$^+$ no. 2/$y^2$ v ma-1
n-14 $Y^L$ (Y900)/y $w^a$
n-15 $Y^L$·ac$^+$ y$^+$ (sc$^8$ EN c.o. Y B-2)/X·$Y^S$, y w/y v f
n-16 $Y^L$·bb$^+$ ac$^+$ y$^+$ (sc$^8$ EN c.o. Y T-0)/ X·$Y^S$, y w/y f:=
n-17 $Y^L$·bb$^+$ $sc^{S1}$ ac$^+$ y$^+$ ($sc^{S1}$ c.o. Y EY80)/ X·$Y^S$, y w/y v f
n-18 $Y^{LC}$/X·$Y^S$ (A-3), sc cy $v^{V1}$·$K^S$/y v f
n-19 $Y^S$·sc$^+$ ac$^+$ y$^+$ ($sc^{V1}$·$Y^S$)/X·$Y^L$, y v f(bb$^+$·)$Y^L$/y f:=
n-20 $Y^S$·$Y^S$ (Y" Stern)/y/X·$Y^L$(A-2), y w·$K^L$

Extra Y Chromosome Stocks

o-1 In(1)$w^{m4L}$ $N^{264-84R}$, y sn/FM3, $y^{31d}$ $sc^8$ dm B l/y ♀♀ x dm sn ♂♂
o-2 In(1)$w^{m4L}$ $N^{264-84R}$, y sn/FM3, $y^{31d}$ $sc^8$ dm B l ♀♀ x FM3, $y^{31d}$ $sc^8$ dm B l/y $Y^+$/$B^S Y$ ♂♂

Deficiencies and Duplications

p-1 Df(1)$ma-1^8$/In(1)d1-49, y w $lz^2$
p-2 Df(1)$N^8$/In(1)d1-49, y Hw $m^2$
p-3 Df(1)$rst^2$/FM1, $y^{31d}$ $sc^8$ $w^a$ $lz^s$ $B^{S1R}$
p-4 Df(1)sc$^{8L}$ (see Ins(1)$sc^{8L}$,d1-49,$sc^{S1R}$)
p-5 Df(1)$sc^{10-1}$/y Hw
p-6 Df(1)$w_{258-45}$ (see In(1)$w^{m4L}$,$rst^{3R}$)
p-7 Df(1)$w_{258-45}$, y/FM4, $y^{31d}$ $sc^8$ dm B
p-8 Df(1)$w_{258-45}$, y; Dp(1;3), $w^{Vco}$/y w f
p-9 Df(1)$w^{Vco}$, y $sc^5$ spl; Dp(1;3), w/y f:=
p-10 Df(3R)ry/In(3LR)$Ubx^{130}$, $Ubx^{130}$ $e^s$
p-11 Df(4)M-4/ey
p-12 Dp(1;f)3 = De1(1)3/y/$XY^L$·$Y^S$, l(1)$J1^{259}$ y w $Y^L$·$Y^S$
p-13 Dp(1;f)18 = De1(1)18/y v f/$XY^L$,$Y^S$, l(1)$J1^{259}$ y w $Y^L$·$Y^S$
p-14 Dp(1;f)24 = De1(1)24
p-15 Dp(1;f)52 = De1(1)52/y/y l(1)$J1^{259}$ w $Y^L$·$Y^S$
p-16 Dp(1;f)112 = De1(1)112/y v f/l(1)$J1^{259}$ y w $Y^L$·$Y^S$
p-17 Dp(1;f)122 = De1(1)122/y v f/l(1)$J1^{259}$ y w $Y^L$,$Y^S$
p-18 Dp(1;f)164 = De1(1)164/y v f/l(1)$J1^{259}$ y w $Y^L$·$Y^S$
p-19 Dp(1;f)1492 = De1(1)1492/$sc^{53k}$
p-20 Dp(1;f)1514 = De1(1)1514/$sc^{53k}$
p-21 Dp(1;1)$B^S$(RAG), $B^S$--In(1)$sc^8$·/Ins(1)$sc^7$, AM
p-22 Dp(1;1)$B^S$(TMG),In(1)$sc^4$·$B^S$, y $sc^4$ m f·$B^S$/Ins(1)$sc^7$, AM
p-23 Dp(1)$B^S$(TMG), In(1)$sc^{8L}$, $X^{c2R}$·$B^S$, f·$B^S$/ $X^D$/$B^S Y^L$·$Y^S$
p-24 Dp(1;2)$sc^{19i}$
p-25 Dp(1;2R)$w^{51b7}$/y w f/In(1)$w^{m4L}$, $rst^{3R}$, y w $rst^3$
p-26 Dp(1;3)$w^{49a7}$ (Spotter)
p-27 Dp(1;3)51 = T(1;3)51/y v f/$XY^L$·$Y^S$, l(1)$J1^{259}$ y w $Y^L$·$Y^S$
p-28 Dp(1;3)142 = T(1;3)142/y/$XY^L$·$Y^S$, l(1)$J1^{259}$ y w $Y^L$·$Y^S$
p-29 Dp(1;3)$sc^{J4}$/y v f/0 ?/$XY^L$·$Y^S$, y $l^{259}$ w $Y^L$·$Y^S$
p-30 Dp(1;4)$w_3^{51c20}$/y w f:=/In(1)$w^{m4L}$, $rst^{3R}$, car rst
p-31 Dp(1;4)174 = T(1;4)174/y v f/l(1)$J1^{259}$ y w $Y^L$·$Y^S$
p-32 Dp(2;f)1/sp $Pin^2$/$Px^4$
p-33 Dp(3R;f)ry$^+$/cu kar/cu kar

Triploid Stocks

q-1 y w $fa^{no}_2$/FM6 ♀♀ x FM6/$B^S Y y^+_S$ ♂♂
q-2 y sc w$^a$ ec/FM6 ♀♀ x FM6/$B^S$Yy$^+$ ♂♂
q-3 In(1)d1-49, v or f/FM6 ♀♀ x FM6 ♂♂
q-4 l(1)$J1^{259}$ y/FM4, $y^+$ w dm f ♀♀ x FM4, y w dm f ♂♂
q-5 l(1)$J1^{259}$ y w$^a$ ec/FM4, $y^+$ w dm f ♀♀ x FM4, $y^+$ w dm f ♂♂

## DUARTE, CALIFORNIA: CITY OF HOPE MEDICAL CENTER

Special Stocks

1. $ct^n$ $oc_2$ $lz^3$ v/FM6 ct v *
2. v dy $g^2$ sd $f^{36a}$
3. v dy $fw^{34e}$ $g^2$
4. Cyg ed/In(2LR) 102, $ds^w$
5. es/SM1
6. es px sp/SM1

-- (also several other stocks containing es)
7. ru h th st $^{D}$cu sr e$^{s}$ ca (rucuca)/ruTSS *
8. y v; Sp bw$^{D}$/SM5; Sb/TM3 Ser
9. y v/FM6; Pin Ubx/T(2,3)Xa
10. $CO_2$ sensitivity stocks

* see discussion in Research Notes, DIS 38 or 39.

## CZECHOSLOVAKIA

### Praha: Czechoslovak Academy of Sciences, Institute of Experimental Botany Department of Plant Physiology and Genetics

| Wild Stocks | Tester Stocks | Chromosome I | Chromosome II |
|---|---|---|---|
| Moskva | ClB/w lz | y | Cy/Bl L$^{4}$ |
| Berlin | Muller-5 | y w$^{a}$ | L$^{4}$ |
| Novosimbirsk | | Y ac sc pn sn | Cd cn vg b |
| Suchumi | | | st bw$^{r}$ |
| | | | st st$^{r}$ e$^{s}$ ro ca |

## GREECE

### Thessaloniki: University of Thessaloniki Department of General Biology

| Wild Stocks | w$_e$ | Chromosome 2 | Chromosome 3 |
|---|---|---|---|
| | w$_{bf}$ | | |
| Oregon K | w$^{n}$ | dp | jv |
| Berlin wild | fa$^{n}$ | Cy | se |
| | sn | b | st |
| Chromosome 1 | v | cn | ss |
| | f | vg | e |
| y | bs | bat | |
| pn$^{2}$ | Muller-5 | bw | Chromosome 4 |
| | | rk | |
| | | Cy L$^{4}$/Pm | ey |

Note: There are also available most of the combinations of the above mentioned stocks.

## INDIA

### Madras: Department of Animal Genetics, Veterinary College

Wild Stocks

Oregon-K
Madras

Chromosome 1

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

Chromosome 2

1 Cy Bl L$^{2}$
2 dp b cn bw
3 vg

Attached-X Chromosome

1 sc$^{S1}$ B In S w$^{a}$ sc$^{8}$ & y v f:=

Chromosome 3

1 e

Multichromosomal

1 bw st
2 y sc$^{S1}$ In49 sc$^{8}$; bw st
3 vg; e

## ITALY

### Napoli: Laboratorio Internazionale di Genetica e Biofisica

Wild Stocks

1 Canton-S
2 Lecce
3 Oregon-R
4 Roma

Chromosome 1

5 B
6 w

Chromosome 2

7 Cy/Pm

## NIGERIA

### Ibadan: Ibadan University College, Department of Zoology

Wild Stocks

1 normal (S. Africa)

Chromosome 1

2 w
3 wm

Chromosome 2

4 vg
5 cn bw
6 b cn vg

Multichromosomal

7 bw; st (2;3)

## FRANCE

### Clermont-Ferrand: M. Hovasse Laboratoire de Zoologie et Biologie generale Faculte des Sciences, Laboratoire de Zoologie

1 Oregon-R-C
2 w
3 e

## INDIA

### Calcutta: University of Calcutta, Department of Zoology

Wild Stocks

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

Chromosome I

b1 y
b2 $w_a$
b3 $w_{bo}$
b4 $w_{co}$
b5 $w_e$
b6 $w_h$
b7 $w_i$
b8 w
b9 B
b10 y $Hw^{49}$ $v^o$ $m^2$, f/$ClB^{36d}$

Chromosome II

c1 vg
c2 bw

Chromosome III

d1 $se^h_a$
d2 $ss^3$
d3 $Ly/D_s$
d4 e

Chromosome IV

e1 $ey^2_W$
e2 ci

Multichromosomal

f1 Cy/Pm, D/Sb

## New Delhi: Indian Agricultural Research Institute

### Wild Stock

1 Oregon-K

### X Chromosome

1 B
2 $sc^{S1}$ B In-S $w^{a}$ $sc^{8}$ (Muller-5)
3 ClB/sc v f car
4 y
5 y $sc^{S1}$ In-S $sc^{8}$
6 y ac sc $pn_{S1}$ w rb cm $ct^{6}$ $sn^{3}_{8}$ $ras^{4}$ v m g f car/$sc^{S1}$ B In-S $w^{a}$ $sc^{8}$

### Chromosome 2

1 vg
2 cn bw
3 b cn bw
4 dp b cn bw
5 bw
6 dp
7 Cy/Bl $L^{2}$

### Chromosome 3

1 e
2 ru h th st cu sr $e^{s}$ ca (rucuca)
3 st
4 $Ly/D^{3}$

### Chromosome 4

1 ey

### Multichromosomal

1 bw; st
2 Cy/Bl $L^{2}$; $D/LVM_{8}$
3 y $sc_{S1}$ In-49 $sc_{8}$; Cy/Bl $L^{2}$
4 y $sc_{S1}$ In-49 $sc_{8}$; cn bw
5 y $sc_{S1}$ In-49 $sc_{8}$; vg
6 $sc^{S1}$ B In-S $w^{a}$ $sc^{8}$; Cy
7 $sc^{S1}$ Y:= bw; e; ey

### Attached-X Chromosome

$sc^{S1}$ B In-S $w^{a}$ $sc^{8}$ & y v f:=

## MEXICO

## Laboratorio de Genética de la Comisión Nacional de Energía Nuclear

### Wild type

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

### Chromosome 1

b1 B
b2 w m f
b3 m
b4 w
b5 $lz^{37h}$
b6 y
b7 y $w^{a}$
b8 f
b9 v
b10 w m f/ClB
b11 $fw^{H}/y$
b12 ma-l/y f:=
b13 ec $ct^{6}$ (s) car/ClB
b14 y $Hw/\frac{Ins(1)\ sc^{S1}L,S,\ sc^{8}R}{sc^{S1}\ w^{a}\ B\ sc^{8}}$
b15 y f:= and $sc^{s}8$ f In-49 $v_{S1}w^{a}$ $sc^{8}$
b16 $sc^{S1}$ B In-S $w^{a}$ $sc^{8}$/l(1) 55aB
b17 sc cv v f
b18 $sc_{S01}$ v f B/y f:=
b19 $lz^{501}$
b20 ma-l
b21 $lz^{3}$/ y f:=
b22 ptg
b23 y v
b24 $y_{2}$w $sn^{3}$
b25 $y^{2}$ v
b26 y w
b27 y ac v

### Scute Alleles

c1 $y_{S1}sc^{S1}{}_{8}$B f In49 v & y f:=
c2 $y_{S1}sc^{S1}{}_{8}$ B f In49 v
c3 $sc^{S1}$ In49 v♂ y f:= ♀

### Combinations of Similar Scute Inversions

d1 y $sc^{4}$ B In49 $lz^{s}$ v $sc^{S1}$/sc oc ptg car
d3 m-$5_{a}$($sc^{S1}_{8}$ B In-S w sc)
d4 (y+♂) $sc^{S1}$ In49 $sn^{x2}$ $sc_{8}$ ♂ & y f:=♀
d8 $sc^{S1}$ B In-S $w^{a}$ $sc^{8}$/l
d9 $sc^{S1}$ In-S $w^{a}$ $sc^{8}$

### Altered Y

f1 ("sz+") $Y^{Lc}/X\cdot Y^{S}$
f2 $YB^{S}_{8}$ ($y^{2}$; brn)
f3 $sc^{4}_{8}\cdot Y/y\ sc^{4}\ w\ sc^{8}$ ($sc^{3}_{L}\cdot Y$ in♂ & ♀$^{VI-S}$)
f4 $y^{3}\cdot Y/sc^{VI-S}$ w-Y & y f:=
f5 $sc^{+}\cdot Y^{S}/y_{L}v\ f_{+}bb^{+}\cdot Y^{L}$
f6 Y: $bw^{+}$ ($Y^{S}$ bw·bb$^{+}_{8}$)/y v; bw
f7 $sc^{4}_{8}$ $Y/y_{S}w^{m4}$
f9 $sc^{4}_{8}\cdot Y\cdot B/y\ w^{m4}$; dp
f10 $sc^{3}\cdot Y/ac$
f11 $Y^{Lc}/y\ w\ sn^{s}$ oc· $Y^{S}$ ♂ & $Y^{Lc}$/ y v f:= ♀

f12 new"facl": $Y^{LC}$/In49 ptg oc $B^{M1}$/$sc^{S1}$ $sn^{5.1}$ w $sc^{8}$ ♀ & $Y^{LC}$/y sn oc ptg v· $Y^{S}$ ♂
f13 $sc^{v1}$ $Y^{S}$/y ac oc ptg·$Y^{L}$ ♂ & y ac oc ptg· $Y^{L}$/y $sc^{S1}$ $B^{S}$ In49 $ct^{ns}$ $v_{M}$ $sc^{8}$ ♀$^{S}$
f14 $Y^{S}$ $X_{L}$(FRI)·$K^{a}$ y $cv^{6}$ v $f^{S}$/$R^{M}$·y/$B^{S}$ $Yy^{+}$
f15 $sc^{S}$ $Y^{L}$/y ac $w^{a}$ $ct^{6}$ f $Y^{S}$/ y f:=
f16 $Y^{S}$/y v f bb $Y^{L}$/y $f^{S}$:=
f17 $scY^{L}$/ y In49 v f·$Y^{S}$;e

Chromosome 2

g1 bw
g2 l me
g3 cn
g4 dp
g5 $L^{2}$
g6 $Ml^{2}$/$ds^{33k}$ Pm
g7 l (2) me/SM1, $al^{2}$ ly $rp^{2}$
g8 l (2) me/SM1
g9 al dp b pr $Blc^{2}$ px sp/SM1, $al^{2}$ Cy $sp^{2}$
g10 Bl $L^{2}$/SM5, $al^{2}$ Cy $tl^{v}$ $sp^{2}$
g11 da/Ins(2L + 2R) Cy·Cy
g12 InCy L b pr $cn^{2}$ In Cy R

Chromosome 3

h1 se ss
h2 st
h3 e
h4 $se^{nd}$
h5 $ca^{nd}$/In (3RL) $Ubx^{130}$ m(3) l
h6 $ry^{2}$
h7 l (3) tr Ubx/TM1, me ri $sbd^{e}$
h8 Tr/me sb
h9 l (3) tr Ubx/TM1
h10 $ca^{nd}$/In (3LR) $Ubx^{130}$, M(3),$Ubx^{130}$ $e^{2}$
h11 $bx^{34e}$
h12 re h th
h13 $e^{s}$ $cn^{nd}$/TM1
h14 $e^{s}$ $ca^{nd}$/In(3LR) $ca^{v}$

Chromosome 4

i1 $ey^{2}$

Multiple Chromosomes

j1 SM1/dp b Pm; $Ubx^{130}$/ C Sb (2;3)
j2 Cy/Pm;H/Sb
j3 $sc^{8}$ Y/y sc In49$B^{m1}$; twl bw; $st^{54i}$
j4 y $sc^{S1}$ In49 $sc^{8}$; dp bw; st $p^{p}$
j5 bw st
j6 y; bw; e ci; $ey^{R}$
j7 y/y f:=; bw; st; pol (1;2;3;4) segregating for e
j8 y; bw st; pol
j9 y f:=; bw; e; ci $ey^{R}$
j10 (X, Y, 2,3,4) $Y^{S}$·In En In49y·$Y^{L}$; cn bw; e; ci ey (no free Y)
j11 Cy/Pm; H/Sb C Sr
j12 Cy/Pm $ds^{33k}$; H/Sb
j13 Cy/Pm; D/Sb

Closed X

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

Multiple Inversions

l1 Ins (2L,2R) Cy $bw^{v2}$/al dp b pr cn e px sp
l2 Ins (1) $sc^{S1}$ In49, B f v y/y f:=
l3 $sn^{3}$; cn bw; Ubx/ri
l4 y; cn bw; Ubx/ri

Triploids

t1 $y^{2}$ $sc$ $w^{a}$ ec/FM4, $y^{31d}$ $sc^{8}$ dm B

## NETHERLANDS

### Leiden: Genetisch Laboratorium der Rijksuniversiteit

Wild Stocks

1 Kolmar
2 Leiden

Chromosome 1

101 B
102 ClB/sc v f car
103 w
106 m
107 Muller-5
108 $sn^{3}$
109 $v_{2}$
110 $y^{2}$ $su\text{-}w^{a}$ & v $f^{3n}$ car:=
111 w
112 + & w:=$_{3}$
113 w cv $sn^{3}$
114 sc ct $v_{3wy}$ f car & y:=
115 f & v $f^{3n}$ car:=
117 gt v B $y^{2}$ sc gt:=
118 $y^{2}$ $su\text{-}w^{a}$ f su-f & y w f:=
119 $z_{58g}$
120 $z^{58g}$

Chromosome 2

201 al dp b pr Bl c px sp/SM-1
202 Bla/SM-5 $al^{2}$ Cy $lt^{v}$ $sp^{2}$
204 Bl L/Cy
205 $bw_{D}^{60g}$
206 $bw^{D}$
207 cl
209 cn bw
210 crc cn/Pm cn
211 crc cn/Pm
212 cn bw Kr/Cy
213 $px_{2}$sp
214 $sf^{2}$
215 vg

Chromosome 3

301 e
302 Gl/$Ubx^{130}$

303 h gs $th_{130}$
304 ltr/$Ubx^{130}$
305 Ly/$D^3$
306 ma
307 $ma-l^2$
308 $ry^2$
309 ru h th st cu sr $e^s_s$ ca
309a ru h th st cu sr $e^s$ Pr ca/Me
310 $ve_2$
311 $ve^2$
312 $ve^2$ se

Chromosome 4

401 $bt^D/ci^D$
402 $ci^W_{57g}$
403 $ci^{D-G}$
404 $ci^D/ey^D$ $spa^{pol}/spa^{cat}$
405 $spa^{pol}$

Multichromosomal

501 Cy/Pm; D/Sb
502 Cy "Oster"$/_R$Pm; $Ubx^{130}$/ Sb; ci ey
503 dor/FM-6; TM-3/Sb
504 ec br; ix/Cy
505 v; cn
506 vg; $spa^{pol}$
507 $w^a$ ♂ & y v f:=; tra/ D Ins-CXF
508 y bw st
509 y c $w^{cn}$ spl; $\frac{Cy; Ubx^{130}}{Xa}$

Triploid

FM-4 & gt:=/FM-4

## Utrecht: Genetisch Instituut Van der Rijksuniversiteit

Wild Stocks

1a Oregon K
1b Wageningen

Chromosome 1

2 $cm_{53}ct^6$ $sn^3$ & y w f:=
2a $cs^{26-48}$ & y $w_{bb}$•=
3 $car^{3N}$ $f^{3N}$ & y f:=
4 cv $f^{3N}$ & y f:=
5 $fu^2$/ClB
6 $g^2$
7 pn
8 ras dy
9 $rb_{27-4}$
10 $rb^{27-4}$ cv v $f^{3N}$
11 $sc^{S1}$ cv v f
12 $sc^{S1}$ B In S $w^a$ $sc^8$ (Muller-5)
13 "tester 1" y ac pn $w^2$ rb $wy^2$ $g^2$ & y f:=; $sc^{19i}/Cy$
14 "tester 2" y $w^a$ $cm_{19i}$ $wy^2$ $g^2$ car & y f:=; $sc^{19i}/Cy$
15 "tester 3" y $rb_{19i}$ cm ras $g^2$ & y f:=; $sc^{19i}$/Cy
16 w sn B
16a w sn
16b w cv sn
16c w
17 y $sc^{S1}$ B In(1)49 $sn^{x2}$ $sc^8$/og ptg
18 $y^{S1}$ $sc^8$ B f In(1)49 v
19 y $w_a$ m B
20 $y_{C2}w$ cv v f
20a X y B & $y_6$f:=
20b sc ec cv ct v $g^2$ $f/FM_6$
20c v dy $g^2$ sd

Chromosome 2

29 b pr vg
29a b cn vg
29b b pr cn vg
30 bw
31 Bl L/Cy
32 dp
33 dp b cn bw
33a $cn_{bw}$
34 $dp^{Th}$ Cy, In-L pr $cn^2$ In Cy R-O/In s-NSL Ins-NSR p x sp
35 $dp^{Th}$ Cy cn bw/S Sp cn bw
36 J/In(2L)t, l(2)B
36a S/Cy, EN-S
36b $S/_R ds^{33k}$ Pm

Chromosome 3

37 e
37a cu
37b gs
38 h ri
39 l tr/e In(3R)In(3L)
40 Mio/In(3R)Sb
40a ss
41 st
41a st e
41b stss
41c stsse
42 st $Sb^r$ $e^s$ ro ca
42b ro
42c ca

Chromosome 4

43 $ci^D$
44 $ci^D/spa^{Cat}$

Multichromosomal

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

Stocks selected for Abnormal abdomen

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

Deficiencies

53 $Df(1)N^8/dl-49$, y Hw $m^2$ $g^4$
54 $Df(1)N^{264-105}$(dm)/dl-49, y Hw $m^2$ $g^4$
55 $Df(1)N^{264-39}$ $w^{ch}/FM_4$, $y^{31d}$ $sc^8$ dm B

Altered Y's

21 X•Y In ENy; st(no free Y)"multi ♀"
22 l $J_1^+$•Y/l J1 $sc^{J1(+)}$ In(1)49 v ptg oc $B^{M1}/y^{S1}$ $sc^{S1}$, In car odsy f $g^2$ dy v $ras^2$ $sn^3$ $ct^6$ $cm^1$ rb ec w l pn $sc^8$("Maxy-v")
23 $sc^8$•Y/y Hw In(1)49 v ptg oc $f^{S1}$ $B^{M1}/y^{S1}$ $sc^{S1}$, In car odsy f $g^2$ dy v $ras^2$ $sn^3$ $ct^6$ cm rb eg w l pn $sc^8$
24 $sc^8$•Y/y In 49 B; $bw^D$ ♂ & y f:=; $bw^D$ "Multi ♂"
25 $Y^{1c}/X•Y^s$; bw "sterilizer +"
26 $Y^{1c}/X•Y^s$; bw "sterilizer bw"
27 $Y^{1c}/X•Y^s$; dp "sterilizer dp"
28 $Y^{1c}/X•Y^s$; cn bw; e "sterilizer cnbw; e"

GERMANY

## Tübingen: Max Planck-Institut für Biologie

### Wild Stocks

1 Berlin (Marburg)
2 Lipari (Wien)

### Chromosome 1

3 B
4 Df(1)bb In(1)bb$^{-}$, y sl$^{2}$ bb$^{-}$/FM4, y$^{31d}$ sc$^{8}$ dm B (extra Y's)
5 Df(1)bb In(1)bb$^{-}$, y sl$^{2}$ bb$^{-}$/FM4, y$^{31d}$ sc$^{8}$ dm B*/sc$^{8}$·Y (* FM4-chromosome recessive lethal)
6 Df(1)bb In(1)bb$^{-}$, y sl$^{2}$ bb$^{-}$/sc$^{8}$ bb w$^{a}$
7 sc$^{S1}$ B InS w$^{a}$ sc$^{8}$ (Muller-5)
8 sc$^{2}$
9 sc$^{4}$
10 y sc$^{8}$ w$^{a}$
11 sc$^{8}$ w$^{bb}$
12 v/Y
13 w
14 w/y
15 w$^{e}$ bb$^{1}$/w$^{e}$ bb$^{1}$/Y$^{St}$, bb & w$^{e}$ bb$^{1}$/Y (extra Y's)
16 w$^{e}$ bb$^{1}$/sc$^{8}$ bb w$^{61a}$
17 y$_{2}$ v f
18 y$^{2}$ w$^{a}$ sc ec/y
19 y w(lz)/In(1)X$^{C2}$, w$^{vc}$ f

### Attached-X

* y/w$_{2}$ $^{a}$ . . . . . (14)
* y/y$^{w}$ sc$_{2}^{ec}$ . . . . . (18)
20 y f/Y$^{S}$ & g$^{2}$ B·Y$^{L}$ Y$^{S}$
21 y$_{2}$ f/y$^{3}$·Y$^{L}$ & sc$^{v1}$ w·Y$^{S}$/y$^{3}$·Y$^{L}$
22 y$_{2}$ su-w$_{a}^{a}$ w$_{a}^{a}$ bb/O & v f B, X·Y
23 y$^{2}$ su-w$^{a}$ w$^{a}$ bb/sc$^{8}$·Y/X$^{C2}$, y v

### Attached X·Y

* g$^{2}$ B·Y$^{L}$ . . . . . (20)
* sc$^{v1}$ w·Y$^{S}$ . . . . . (21)
* v f B·Y . . . . . (22)
24 X·Y$^{L}$(A-2), y w·Y$^{L}$/y$^{S}$·Y$^{S}$

### Closed X

* In(1)X$^{C2}$, w$^{vc}$ f . . . . . (19)
* X$^{C2}$, y v . . . . . (23)

### Altered Y's

* Y$^{bb}$ . . . . . (12)
* Y$^{St}$ . . . . . (15)
* sc$^{8}$·Y . . . . . (t, 23)
* Y$^{S}$ . . . . . (20)
* Y$^{S}$·Y$^{S}$ . . . . . (24)
* y$^{3}$·Y$^{L}$ . . . . . (21)

## AUSTRALIA

## Sydney, New South Wales: University of Sydney, Faculty of Agriculture

### Wild Stock

1. Oregon-R-C

### Chromosome 1

2. w
3. B
4. w f
5. y w$_{e}$
6. y w
7. f B
8. ct v f
9. sc cv v f
10. sc cv v f B

### Chromosome 2

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

### Chromosome 3

17. e$^{s}$
18. e$_{11}$
19. st
20. e$^{4}$ wo ro

### Chromosome 4

21. ci ey$^{R}$

### Multichromosomal

22. bw; st
23. v; bw
24. dp; e
25. y w; dp

### Special Stocks

26. Pr/In(3R)1
27. w f/ClB
28. Cy/Pm
29. y/B
30. y/lz$^{57j}$
31. y; Cy/Pm, ds$^{33k}$; H/Sb

## WALTHAM, MASSACHUSETTS: BRANDEIS UNIVERSITY
### Department of Biology

D. busckii
D. funebris
D. persimilis
D. robusta
D. simulans
D. virilis

## LEXINGTON, KENTUCKY: UNIVERSITY OF KENTUCKY

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

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

D. americana americana: Independence, Ohio; Western
D. americana texana: Florida
D. ananassae: Cristobal
D. bifasciata: sex ratio; Pavia normal
D. busckii: Lankenau (Abington, Pa.)
D. equinoxialis: Puerto Rico, normal and sex ratio
D. flavomontana: Yampa River, Colo.
D. funebris: Rexburg, Idaho; Stockholm, Sweden; Upperville, Va.; white eye; Yucatan
D. gibberosa: South Mexico
D. hydei: Zurich, Switzerland, normal; Lobe (Gloor); New Haven, Conn. (originally listed as D. repleta)
D. lacicola: Fairbanks, Minn.
D. littoralis: Switzerland
D. melanica: St. Louis
D. montana: Cottonwood Canyon, Utah; Lu
D. nebulosa: Haiti, normal and sex ratio
D. nigromelanica: Cold Spring Harbor
D. novamexicana:
D. paramelanica
D. paulistorum: Belem; Bucamaranga; Cantareiras; Lancetilla; Trinidad
D. persimilis: Whitney, Calif.
D. prosaltans: Belem; Chilpancingo (stellata)
D. pseudoobscura: Pinion Standard
D. repleta: Philadelphia, Pa.
D. simulans: Lankenau
D. virilis: Japan
D. willistoni: Barbodos-3; Belem; Recife-3; Recife-6; Recife Pop. 168; ebony; pink; white eye; sex ratio
Zaprionis vittiger: South Africa

## EAST LANSING, MICHIGAN: MICHIGAN STATE UNIVERSITY
### Department of Zoology, Biology Research Center

D. virilis (California)
D. virilis:(Yale)
D. funebris: Stockholm (Yale)
D. pseudoobscura (Yale)
D. pseudoobscura (Rochester)

## ITALY

### Napoli: Laboratorio Internazionale di Genetica e Biofisica

D. busckii

## FRANCE

### Gif-sur Yvette: Laboratoire de Génétique évolutive et de Biométrie

D. ambigua
D. funebris
D. kuntzei
D. obscura
D. simulans
D. subobscura

## INDIA

### Calcutta: University of Calcutta, Department of Zoology

Drosophila ananassae

Wild Stocks

1 a6 Calcutta
2 N7 Nai - C

Chromosome I

3 y bo

Chromosome II

4 b cu
5 se cu b
6 bw vs $ss^a$
7 pr
8 cu pr

Chromosome III

9 pc
10 e

Multichromosomal

11 b ic
12 pr stw px fu
13 cu ic

## SPAIN

### Madrid: Centro de Investigaciones Biológicas, Laboratorio de Genética

D. busckii: Madrid, Santianes
D. funebris: Madrid
D. guyenoti: Santianes
D. immigrans: Madrid, La Franca, Santianes
D. melanogaster: La Franca, Madrid, Mallorca, Ribadeo, Rocafort, Ronda 10, Ronda 30, Santianes, Villagarcía
D. obscuroides: La Franca, Santianes
D. repleta: Madrid
D. subobscura: La France, Madrid, Santianes

## NETHERLANDS

### Utrecht: Genetisch Instituut der Rijksuniversiteit

51 D. simulans
52 D. busckii

## GERMANY

### Berlin-Dahlem: Institut für Genetik der Freien Universität Berlin

71 D. funebris : wild
72 D. busckii : wild
73 D. hydei : wild
74 D. simulans : v
75 D. virilis : wild

NEW MUTANTS

Report of S. K. Sarkar

bw$^{ad}$: brown Das, A. K. 63a$_7$, spontaneous from wild stock in the line of $F_2M_{24}$. Eye colour light brownish on emergence; colour varies with age from light brownish to garnet. Allelic test with bw flies shows to occupy the same genetic locus. Penetrance complete. $Rk_1$

ey$^{ad}$: eyeless Das, A. K. 63a$_7$, spontaneous from wild stock in the line of $F_2M_{24}$. Varies from no eyes to wild type. Allelic test with ey$^R$ shows to occupy the same genetic locus. Penetrance complete. $Rk_1$

ct$^{n63}$: cut-notch 63 R. K. Datta, 63B$_{11}$. One male appeared spontaneously in B stock. Wings cut to points and notched. Abdominal bands somewhat wrapped. Expression more extreme in females than in males. Penetrance complete. Fertility and viability excellent. $Rk_1$

vs$^{63}$: vesiculated 63 R. K. Datta 63C25, spontaneous from w$^1$ stock. Wings wrinkled, blistered, rough textured and no overlapping of the wings are observed. Sometimes wings are bag-like and divergent. Expression variable but the penetrance is complete. $Rk_1$

tu-h$^{63}$: tumor head 63 R. K. Datta 63C26, spontaneous from w$^1$ stock. Abnormal growths in the various regions of the head. Small protuberances are found over or near the eye and antennal region. The eyes in some cases become smaller and to some extent in the form of eyeless condition. Aggregation of small hairs are also found near the eye and antennal region.

Report of P. E. Thompson

Px$^5$: Plexate$^5$ Thompson, 1957. Spontaneous in, and inseparable from, Ins(2) Pm of al S ho/ds$^{33k}$ Pm. Produces sacs or vesicles frequently, but little irregularity of venation. Includes bs, ba, not sp. Lethal in combination with Px, Px$^2$ and Px$^4$.

Su-Cy: Suppressor of Curly Thompson, 61e. Spontaneous in, but separable from, Ins (2) Pm of SM1/ds$^{33k}$ dp b Pm; Ubx$^{130}$ e$^s$/Sb. Suppresses wing effect of Curly.

1(2)hst: lethal (2) histolytic Thompson, 59k. 2-56$^{\pm}$. X-ray induced in spermatocyte of Gla/+ male. Homozygote dies in early pupal stage. Heterozygous viability good. RK4

T(Y;2)dp: Translocation (Y;2)dumpy Thompson, 61d. X-ray induced in spermatid of SM5/+ male. Gives dumpy phenotype in heterozygotes with dp.

Report of G. H. Mickey

bw$^{61j}$: bubble wing 61j Mickey, 61j 10. 1 - ? Sex-linked recessive which arose from wild type male treated with radio frequency. Uniform expression in both males and females. Not located. RK3. Still in stock.

ct$^{62a}$: cut 62a Mickey, 62a22. 1 - 20.0. Arose from wild type male treated with radio frequency. Sex-linked recessive. Fertility and viability good. RK1. Still in stock.

ct$^{62f}$: cut 62f Mickey, 62f8. 1 - 20.0. Spontaneous in basc stock. Sex-linked recessive which is lethal in male. RK1. Still in stock.

lz$^{62k}$: lozenge 62k Mickey, 62k11. 1 - 27.7. Sex-linked recessive. Eye smaller and narrower than wild type; surface glossy from fused facets; eye color light brown, darker at rim. Females sterile. Arose from wild type male treated with X-rays. RK1. Still in stock.

$N^{621}$: Notch 621 Mickey, 6213. Sex-linked dominant, lethal in male and homozygous female. Arose from wild-type male treated with radio frequency. Not analyzed. RK3. Still in stock.

$sn^{63a}$: singed 63a Mickey, 63a17. 1 - 21.0. Arose from wild-type male treated with radio frequency. Sex-linked recessive like original singed. Females viable but sterile. Males fertile. RK1. Still in stock.

$sn^{63b}$: singed 63b Mickey, 63b19. 1 - 21.0. Arose from wild-type male treated with radio frequency. Sex-linked recessive like original singed; carried in homozygous stock. RK1. Still in stock.

$w^{62d}$: white 62d Mickey, 62d5. 1 - 1.5. Spontaneous in basc stock. Sex-linked recessive; viable and fertile in both sexes. Rk1. Still in stock.

$wh^{54a}$:whiskers 54a Mickey, 54a7. Autosomal, not located. Arose from wild-type male irradiated with neutrons from cyclotron. Many extra vibrissae, longer than normal. Rk3. Still in stock.

$y^{62b}$: yellow 62b Mickey, 62b21. 1 - 0.0. Arose from wild-type male treated with radio frequency. RK1. Still in stock.

$y^{62k}$: yellow 62k Mickey, 62k 8. 1 - 0.0. Spontaneous in basc stock. RK1. Still in stock.

In(1)$w^{m541}$: Inversion(1)white mottled 541 Mickey, 5413. Arose from wild-type male irradiated with neutrons from cyclotron. Shows mottling of eye color similar to white mottled 4. Breaks at 3C3 and 20D2. Still in stock.

In(2L)$^{53d}$: Inversion(2L)53d Mickey, 53d4. Arose from wild-type male irradiated with neutrons from cyclotron. Showed mottling or variegation in eye pigment. Breaks in 2L at 25A5 and 29F? Lost.

In(2R)$bw^{V54a}$: Inversion(2R)brown-Variegated 54a Mickey, 54a6. Arose from gamma irradiation of wild-type male. Breaks at 59D8 and 41A-B down at centromere region in heterochromatin. Still in stock.

In(2R)$bw^{V54c}$: Inversion(2R)brown-Variegated 54c Yanders, 54c5. Arose from wild-type male irradiated with neutrons from cyclotron. Breaks in heterochromatin near centromere, and at 59E1. Lost.

In(2R)$bw^{V54b}$: Inversion(2R)brown-Variegated 54b Mickey, 54b12. Arose from wild-type male irradiated with neutrons from cyclotron. Breaks in 2R at 60D10 and 41A0 in heterochromatin. Variegation of eye pigment and roughened (pebbly) surface of wing. Lost.

In(3L / 3R)$M^{54c}$: Inversion(3L / 3R) Minute 54c Mickey, 54c10. Excellent balancer for third chromosome which arose from wild-type male irradiated with neutrons in weapons test. Complex rearrangement involving at least six breaks (perhaps eight), three (or four) inversions, a transposition and a deletion or mutation. Phenotype shows Minute and also scarlet when over a chromosome carrying scarlet, which may be due either to a mutation to scarlet or, more likely, a deletion of the scarlet locus. Two (or three) inversions in 3L; a portion of 3R attached to heterochromatin of 3L near centromere with tip of 3L at opposite end. Sequence = (3L) 61A1 (end) to 61C2, (3R) 93B5 to 100B8, (3L) 80C5, centromere, (3R) 81A1 to 93B4, (3L) 80C4 to 75D7, (3L included inversion covering st) 73A10 to 75D6, (3L) 73A9 (another included inversion not delineated) to 61C3, (3R) 100B9 to 100F5 (end). Still in stock.

T(X·2)$In^{X\ \&\ 3}$: Translocation (X·2) Inversions X & 3 Mickey, 53e 11. Arose from wild-type male irradiated with neutrons from weapons test. Complex rearrangement involving reciprocal translocations between X chromosome and 2R as well as inversions in both X and 3R. Tip of X to break at 3C4 translocated to 2R at break 58F7. Tip of 2R translocated to X at break 20A2. Inversion breaks in X at 3C4 and 20A2. Inversion breaks in 3R at 89E5 and 96A13. Phenotype showed white mottled. Lost.

T(X•2•4)$w^{mf}w^{V}$: Translocation(X•2•4) white-mottled, brown-Variegated Mickey, 53f15. Arose from wild-type male irradiated with neutrons from weapons test. Complex rearrangement involving four breaks and reciprocal translocations, between X and 2, and X and 4. Sequences in the 2•X•4 chromosome = (2R) centromere to 59C4, (X) 12F3 to 3C4, (4) 101E5 to 102F6 (end). Sequences in X•2 chromosome = (X) centromere to 12F2, (2R) 59C5 to 60F5 (end). Sequences in 4•X chromosome = (4) centromere to 101E4, (X) 3C5 to 1A1 (end). The tip of X curls back to make a ring; telomeres are attracted to heterochromatin; ectopic pairing. Phenotype undoubtedly involves both white-mottled and brown-Variegated. Lost.

T(Y•2)$^{54a}$: Translocation (Y•2)54a Mickey, 54a. Pub. and fig. Mickey, 1959, Univ. Texas Pub. No. 5914:99-105. Reciprocal translocation between long arm of Y and right arm of 2. Produces mottling of eye color, more conspicuous in males than in females. Break occurred in 2R at 59C5. Still in stock.

T(2•3)$M^{V54d}$: Translocation (2•3) Mickey-Variegated 54d Mickey, 54d5. Reciprocal translocation between 2R and 3L producing variegation in eye color, much more prominent in males. Arose from wild-type male treated with gamma rays. Breaks in 2R at 43E8 and in 3L at 75C2. Could involve either cn or st loci or both. Lost.

T(3•?)$M^{V53h}$: Translocation (3•?) Mickey-Variegated 53h Mickey, 53h4. Arose from wild-type male irradiated with neutrons from weapons test. Phenotype of dominant mottling or variegation of eye pigment. Translocation of portion of 3L to another centromere forming a small ring chromosome. Probably involves four breaks: in 3L at 62D2 and 64E3. Deletion chromosome was lost, therefore stock was carried as hyperploid. Lost.

Report of F. J. Ratty and D. L. Lindsley

$vg^{U}$: Ultravestigial Ives, 55131. 2-67.0. Ives reported this dominant homozygous allele of vg as a new mutant in DIS 30. Cytological analysis indicates that the phenotype is associated with an inversion extending from 49C2 to 50C1.

Report of E. W. Hanly

$w^{mo}$: white-mottled orange I - 1.5 $\neq$ . Spontaneous male appearing in cross of attached-X females to ma-1 males. Allele of w, $w^{a}$, $w^{Bwx}$, etc., producing various eye colors when in combination with these alleles. Mutant color mottled and slightly lighter than $w^{a}$, darkening considerably with age. Mutant with ma-1 has same colored eyes as mutant without ma-1 on emergence, but becomes darker with age when ma-1 is not present. Both ommochromes and drosopterins obviously reduced in the eyes. Red pigment (apparently drosopterins) accumulates in the abdominal fat bodies of some of the males within a day or two after emergence when raised at 27°C. Penetrance of this character is about 75% in males at this temperature. Abdominal red pigments disappear during the fourth and fifth days of adult life, seemingly with the disappearance of the majority of the fat bodies. Females apparently lack this red-pigment accumulation.

Report of S. Polivanov

Lozenge$^{59}$: $lz^{59}$ is a new radiation-induced mutation in the lozenge series of pseudoalleles. It was found in the progeny of an irradiated wild-type male of D. melanogaster (3,000 roentgens). It was detected through the use of the Muller-5 chromosome. The eyes of $lz^{59}$ are reduced in size and have typical diamond or egg-shaped form, pointed at the lower end. The ommatidia are fused and the whole surface of the eye is shiny, slightly rough and almost or completely hairless. The color is light brown with a darker, slightly reddish rim. This rim is generally broader at the lower half of the eyes. However, the rim is not uniform and has no definite border. A fine diffuse meshwork of darker pigment extends from the rim toward the center of the eye, the extent of this meshwork being variable from individual to individual, making the appearance of the whole eye darker or lighter.

Phenotypically, the $lz^{59}$ closely resembles lozenge clawless ($lz^{cl}$), described by Anders (1955). In combination with vermillion (genotype $lz^{59}$vf) $lz^{59}$, as $lz^{cl}$ produces almost colorless eyes with a narrow red rim, most clearly expressed at the posterior side of the eyes.

Tarsal claws in $lz^{59}$, as in $lz^{c1}$, are so greatly reduced that they are practically absent. However, males of $lz^{59}$, unlike those of $lz^{c1}$ are completely sterile. One hundred males tested individually with wild-type virgin females produced no offspring. Although $lz^{59}$ males copulate, microscopic examination of sperm storing organs of virgin females which have been kept with $lz^{59}$ males from 45 to 70 hours did not reveal any motile sperm. The sterility of $lz^{59}$ males appears to represent another of the possible pleiotropic effects of the lozenge locus. Homozygous females have never been observed.

The allelism of $lz^{59}$ has been tested by crossing of $lz^{59}$/M-5 females to $lz^{37}$ males. Hybrid $lz^{59}/lz^{37}$ females have been obtained from this cross. They have a mutant phenotype. In their appearance they are approximately intermediate between $lz^{59}$ and $lz^{37}$. The eyes of $lz^{59}/lz^{37}$ are oval, narrower than those of $lz^{37}$, but larger than those of $lz^{59}$, and not so pointed at the lower end as in $lz^{59}$. Ommatidia are fused to a large extent, although some of them can still be recognized. The surface of the eyes is shiny but rough and covered with hairs. Often present on the surface of the eyes are dark protruding granulae similar to those found in polished (pl). The eyes are dark, carmine red, although the brightness of the eyes is variable.

Tarsal claws are present in the $lz^{59}/lz^{37}$ hybrid but are reduced more than in $lz^{37}$. The $lz^{59}/lz^{37}$ females are fertile but their fertility is strongly reduced in comparison with that of wild-type females.

Literature cited: Anders, G., 1955. Zeitschrift fuer Inductive Abstammungs und Vererbungslehre, 87:113 - 186.

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Report of G. H. Mickey

$sn^{61k}$: singed 61k G. H. Mickey 1961k. 1-21.0 Induced by cesium gammas. Very similar to original $sn^1$ by Mohr. Female entirely sterile. RK2.

$sn^{61k2}$: singed 61k2 G. H. Mickey 1961k2. 1-21.0 Induced by cesium gammas. Phenotypically like $sn^3$ by Mohr. Females entirely fertile. Rk1.

Report of M. J. Whitten

wi: witty-eye M. J. Whitten 1961g. Spontaneous in Cy j stock. 2 - 54.9±.8. Normally recessive, on occasion the heterozygote shows witty-eye phenotype but behaves as a recessive in subsequent crosses. One case of true dominance has been found but the dominant stock has since been lost. Variable penetrance and expression. Witty-eye affects facet pattern which is irregular and is generally accompanied by greater or fewer ommatidia than is present in wildtype. Extra eye pad bristles present but extremely variable in number and distribution; witty-eye is invariably confined to the lower half of the eye; fully fertile in both sexes. RK1. (see research notes).

Report of E. Markowitz

roe: roughened eye Markowitz, 61g. 3-47.6±. Spontaneous in v f B, $\overline{XY}/y^2$ su-$w^a$ $w^a$ bb stock. Eye slightly smaller than wild type and rough textured. RK1.

Report of M. B. Seiger and H. A. Bender

$lz^{63f17}$: Lozenge-63f17 A. B. Burdick 1963 Spontaneous in m - dy recombinational study. Smooth textured brownish eye (color quite variable) with strong peripheral pigmentation. The size of the eye is moderately reduced. Tarsal claws and pulvilli are strongly reduced. The spermathecae and accessory glands are missing and the reproductive capacity is very low. Allelism tests were performed by M. Borgia Klingele. Lozenge-$^{63f}$was found to be functionally allelic with $lz^{34k}$, $lz^{D}$ and $lz^{61}$ and complementary with $lz^{50e}$. The $lz^{k}/lz^{63}$ compound has variable spermathecal numbers ranging from zero to three. RK-1.

Report of K. Mayeda

corr: corrugated wing Mayeda, 61g. 2-36±. Spontaneous in local wild caught stock (Detroit). Wings are wrinkled and wavy, reduced to 3/4 size of normal and in newly emerged flies resemble puffed. Resembles mutant uex described by Maeda (DIS 36:39). At 25°C. only posterior 3rd of the wing is corrugated but at 20°C. whole wing is affected. However, even at 25°C. corrugated does not overlap wild type. RK2.

Report of M. Carfagna and B. Nicoletti

In(3R)1(3)Na Recovered as a lethal balancer for the Sb gene in an experimental population (Naples, 1960). The salivary analysis shows a large inversion in the right arm of the third chromosome, with the break points in region 86F, between bands 12 and 3 and in region 97A before the 56 doublet. Doublet 97A 12 seems to be missing. The inversion, homozygous lethal, appears to correspond to the general description of In (3R) K (Bridges and Brehme, 1944), and quite likely is the same one. As the Sb/+ flies were in competition with normal individuals from the Canton stock, it is not possible to reconstruct the origin of the inversion, selected through a balanced lethal system which allowed the Sb gene to maintain in the population a rather high frequency (14%). This inversion seems to be a good balancer for the Sb gene, since it has been maintained Sb/In(3R)1(3)Na for several years. No data on crossing over inhibition along the 3R arm are yet available.

Report of W. W. Doane

Linkage data for Amy and adp mutants of D. melanogaster Recently, Kikkawa (DIS 37:94) intimated that the mutant adipose$^{60}$ (adp$^{60}$) is located at the same locus as that designated for the Amylase mutants. I now have extensive data to support the contention that the Amy and adp loci are clearly distinguishable from one another, in fact, 6.1 cross-over units apart. These data, too involved to outline here, are being prepared for publication. They indicate the Amy and adp loci to lie at 2-77.3 and 2.83.4, respectively, on the right arm of Chromosome II. The Amy$^{ad}$ allele, recently named by Prof. Kikkawa and derived from the adp$^{60}$ stock I sent to him, appears to be distinct from any of the amylase mutants I received from him (Amy$^{s}$, Amy$^{+}$, Amy$^{wh}$). It is, however, quite separable from the adp$^{60}$ mutant which is present in that same line.

Report of G. Del Campo

dp$^{D}$: dumpy-Dominant E. B. Lewis, 62. Analysis by Del Campo. From x-rayed ca K-pn male. T(2;3). Unpublished. dp$^{D}$/+ has slight oblique wing effect and moderate vortex and comma effects on thorax. Homozygous lethal. dp$^{D}$/dp has strongly enhanced wing and thorax effects with thickened legs. dp$^{tx}$/dp$^{D}$ is lethal; dp$^{D}$/dp$^{v1}$ and dp$^{D}$/dp$^{v2}$ have strongly enhanced thorax effects. dp$^{D}$/dp$^{o}$ has strongly truncated wings with possible enhancement of thoracic effects. Salivary gland chromosome analysis by Lewis and Del Campo shows reciprocal translocation with breaks in 25A and 95 B-D.

Report of S. Mora

Tg: Tegula E. B. Lewis, 62. Analysis by Mora. 2- 0.0 - 4.0±. In(2L). From x-rayed ca K-pn male. Unpublished. Wings extended uniformly at 90° from body axis, often drooping. The tegula (small plate at the base of the wing) is uniformly reduplicated and the adjoining anterior supra-alar bristle is usually twinned as well. Homozygous lethal. Salivary gland chromosome analysis by Lewis and Mora shows short inversion from 21C to 22F. RK3A.

Report of W. Scharloo

ci$^{D}$-G cubitus interruptus dominant of Gloor. 4- Found as a recombination product of 2 4th chromosomes, with ci$^{D}$ and spa $^{pol}$ respectively, by H. J. Gloor. The ci$^{D}$ in the new ci$^{D-G}$, spa $^{pol}$ chromosome differs from the original ci$^{D}$ in the following points:

1. Expression always less extreme; tested in a variety of backgrounds.
2. The wings are not spread and no black clots were observed in the axillary spiracles.
3. The temperature sensitive period falls largely before pupation instead of after pupation as in ci$^{D}$.
4. Expression has an approximate linear relation with temperature and overlaps easily with wildtype. This in contrast with ci$^{D}$ where expression shows a marked facilitation in response when about 70% of the 4th vein is present, and change is very difficult when wildtype is approximated.

Report of D. Childress

wm: watermelon E. Ehrlich, 1962. 1-6.7 X-ray induced. Eye watermelon color; darkens considerably with age. Viability good.

Gill, Kulbir S. A mutation causing abnormal mating behavior.

This mutation, a third chromosomal recessive, was X-ray induced and originally obtained as a male-sterile mutation. Female flies homozygous for this mutation are fertile and show no atypical behavior. Homozygous males, on the other hand, are completely sterile and exhibit abnormal mating behavior. Male flies actively court each other. A courted male starts running away from a courting one, only to be pursued actively by the latter; a third male then may start courting the second, thus forming a line of three. By a similar process, this line can be extended to include additional males. Any time during this process, if the first male comes near the last male, the former starts courting and being courted. In spite of this active courtship, no matings between males were ever observed. This homosexual behavior is observed even in the presence of female flies. Courtship between the mutant males and normal females extends only for a few seconds and in a two hour period mating between males and females was never observed. Female flies generally fly away from the courting males. A closer observation of this courting pattern may explain the failure of mating between these males and females.

Normal female flies kept with mutant males from one to six days did not show any sperm in the spermathecae and ventral receptacles. It is thus possible that the sterility may be only behavioral. Transplantation of mutant testes into normal larval hosts and vice versa is expected to decide this point. The name "fruity" has been tentatively suggested for this mutant.

Yanders, Armon F. The rate of *D. melanogaster* sperm migration in inter- and intra-strain matings.

Although the details of insemination and fertilization in Drosophila are not yet clear, it is evident that every sperm which has succeeded in fertilizing an egg has overcome a series of obstacles. One of the first, and possibly the most demanding, occurs after the male has deposited the spermatophore in the genital chamber of the female. Each sperm which ultimately reaches the ventral receptacle or one of the paired spermathecae (it seems unlikely that fertilization is accomplished by any sperm not first resident in one of these organs) must become free of the spermatophore and migrate the necessary distance. The spermatophore is a viscous, tangled mass, and my experiences with Italian spaghetti lead me to believe that the separation of individual sperm is a rather severe test in itself; but it must be accomplished with some dispatch, since the first egg which is laid after copulation will effectively sweep out those sperm which still remain in the genital chamber.

Dissection of females soon after copulation reveals that the migration is indeed rather rapid. The ventral receptacle reaches its maximum fullness within an hour, the first sperm appearing in the organ only a few minutes after ejaculation. However, the relative speed of filling seems to differ in various inter- and intra-strain crosses, as described below.

Virgin males and females 3 to 4 days of age were mated in pairs in small glass tubes and observed for 3 to 4 hours. The time of mating was recorded for each successful pair. Twenty minutes after the onset of copulation, the female was removed, dissected immediately in saline, and the excised ventral receptacle scored at 250x for presence of sperm. The scores are estimates of the relative fullness of the ventral receptacle: 0 = no sperm, 1 = 1 sperm to 1/4 full, 2 = 1/4 to 1/2 full, 3 = 1/2 to 3/4 full, and 4 = 3/4 to complete fullness. Scores were recorded in the "0" category only if a spermatophore had actually been deposited; mating without ejaculation occurred only in about 1% of the cases observed.

Flies from four geographical strains were used: Oregon-R (OR), Canton-S (CS), Crimea (C) and Swedish-B (SB). A minimum of 36 pairs was sent up for each of the 16 possible male-female combinations. Although a definite reluctance to mate was noted in some combinations, 264 successful matings were obtained and scored. All females to be scored were assigned code numbers to reduce the possibility of subjective bias.

To enable comparisons between combinations to be made more easily, the raw scores have been converted to a value called the Insemination Index (I.I.). The I.I. is an estimate of the relative success of insemination, and is the sum of the raw scores for each group divided by the theoretical perfect total (four times the number of flies scored). The I.I. values for each of the combinations tested are as follows:

| | Strain of ♀ | | | |
|---|---|---|---|---|
| Strain of ♂ | OR | CS | C | SB |
| OR | .27 | .39 | .50 | .48 |
| CS | .18 | .17 | .30 | .35 |
| C | .44 | .56 | .17 | .50 |
| SB | .35 | .40 | .43 | .28 |

The average duration of copulation was similar in all groups, so it is assumed that the average time of ejaculation was also similar. If this is true, the I.I. values provide an estimate of the rate of filling of the ventral receptacle, and indicate that sperm migration is measurably slower in certain combinations of strains. This could be attributed to an inhibitory action of the female genital tract on the motility of certain classes of sperm, a type of "insemination reaction" not heretofore noted in melanogaster. This does not seem to be the typical insemination reaction as described by Wheeler (U. of Texas Publ. 4720:78, 1947) since the strongest reaction would not be expected to occur in the intra-strain matings. In the present experiments, with one exception, the lowest I.I. value for each strain does occur in the intra-strain matings. The single exception (CS♂ x OR♀) is based on only eleven scores, however, and the data from other experiments indicate that this I.I. value may be abnormally low.

One would hesitate to conclude, on the basis of these results, that melanogaster commonly exhibits a weak "self-sterility" based on an inhibition of sperm motility. But, considering the limitation of the technique, the data suggest that such a mechanism may exist, and the possibility is interesting enough to warrant further study.

Scharloo, W. Long term selection on the expression of $ci^D$.

Two way selection on the expression of cubitus interruptus dominant ($ci^D$) from 3 unrelated base populations changed expression considerably until after some 20 or 30 generations plateaus were reached at rather similar expression values of about 80% in the high lines and 15-20% in the low lines (Scharloo, 1962). Expression of $ci^D$ was measured as the percentage ratio of the length of the fourth vein present and the length of the 3rd vein, both measured from the posterior crossvein.

Immediately after stopping selection in these lines crosses between the 3 high lines (selected for long 4th vein) and between the 3 low lines (selected for large 4th vein gap) were performed and carried through to the $F_3$'s which were used as base populations for new lines selected for extreme expressions during 10 or 15 more generations. Each line consisted of two cultures A and B. From each culture 20 individuals of each sex were measured. The four most extreme individuals from each sample were used as parents for the next generation, ♀♀ from culture A were crossed with ♂♂ from culture B and vice versa.

In the crosses the mean value approximated the midparent values. So there is no indication for either heterosis in the $F_1$ or "hybrid breakdown" in $F_2$ or $F_3$. This agrees with earlier results in crosses of long inbred lines.

In all lines progress was obtained. In the 3 high lines after some generations individuals with a full fouth vein were obtained. These were predominant in two of the lines when selection was suspended. At that time in the two lines mentioned last, a phenotype was fixed similar to one occurring regularly in one of the lines involved in the origin of the new lines.

In the other high line a similar pattern was found in low frequency. This phenotype showed extra venation in a rather constant pattern. In one of the lines it could be shown that this extra venation pattern is dependent on both 2nd chromosomes of this line.

Substitution of wild type 4th chromosomes for the $ci^D/spa^{Cat}$ system produced flies with extra venation in all 3 high lines. This extra venation is therefore independent of the presence of $ci^D$.

In the 3 low lines also progress was obtained down to levels of expression ratio of 10-15%.

In one of the lines an appreciable shortening of the 5th vein occurred and a break between the posterior crossvein and the 4th vein was a regular feature.

In the other lines the 5th vein was affected to a lesser extent what points to a certain specificity of the modifiers accumulated by selection on the 4th vein.

Replacement of the $ci^D/spa^{Cat}$ chromosomes by normal 4th chromosomes did not show an effect on the 4th vein of the modifiers without the presence of the mutant. However, in the low line mentioned first, instead of "assimilation" of the mutant character selected for, the terminal break of the 5th vein appeared to be independent of the presence of the mutant.

This low line with substituted 4th chromosomes showed also a high frequency of crossvein defects. Both the substitution of one 2nd and one 3rd chromosome by respectively chromosomes marked with Pm and Sb causes smaller 5th vein gaps with incomplete penetrance, which vanish completely when both marked chromosomes are introduced together. This points to multigenic control of the 5th vein gap independent of $ci^D$.

Ehrman, Lee. Sexual isolation between the Mesitas and Santa Marta strains of D. paulistorum.

Carmody, Diaz Collazo, Dobzhansky, Ehrman, Jaffrey, Kimball, Obrebski, Silagi, Tidwell, and Ullrich (1962, American Midland Naturalist) reported results of the "male choice" experimental technique used in testing inter- and intraracial mating preferences of the six D. paulistorum races; the data involved the dissection of some 16,335 ♀♀. Since then a newly-collected Transitional race strain from Colombia has been found to produce fertile female and some sterile male hybrids when crossed to another Transitional race strain - Santa Marta. A genetic analysis suggested (Ehrman, PNAS, 1963) that the sterility in this case is caused, chiefly if not exclusively, by interaction of the Y chromosome of one strain with the cytoplasm of the other strain. If the Y and the cytoplasm come from the same strain, the male is fertile, while if their sources are different, the male is usually sterile.

The same experimental and statistical techniques employed by Carmody et al. were applied to these two interesting strains:

| ♂♂ and ♀♀ | ♀♀ | Homogamic + | Homogamic - | Heterogamic + | Heterogamic - | Isolation Index | Chi Square |
|---|---|---|---|---|---|---|---|
| Santa Marta | Mesitas | 26 | 24 | 1 | 49 | +0.93 | 29.2 |
| Mesitas | Santa Marta | 27 | 23 | 14 | 36 | +0.32 | 6.0 |

(An isolation index of +1.00 indicates that only homogamic matings have taken place, an index of 0 that the matings take place at random; any chi-square > 3.84 is significant.)

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Myszewski, Michael E. and A. F. Yanders. The effects of storage upon the differential survival among sperm.

By observing the relative occurrence of progeny of different genotypes in heterozygous stocks of Drosophila melanogaster, it was hoped to determine if selection in the form of differential survival could be detected. Rather than study this subject on a population basis, the phenomenon of differential survival was sought in the mature gamete. One may postulate the existence of a class of genes whose actions would result in the non-random loss of those sperm carrying the gene, or the inability of these sperm to successfully accomplish fertilization.

To test this notion, five stocks were made up, each of which was heterozygous for two second chromosome lethals. In each case, a curly (Cy) -bearing chromosome was balanced with one of five X-ray induced second chromosome recessive lethals which had been detected by standard tests and were maintained in balanced lethal stocks. The two lethal chromosomes in each heterozygous stock (Cy and lethal) should segregate such that any given offspring would normally be heterozygous for one of the lethals. A 1:1 ratio between the two types of heterozygotes should then result from normal segregation and fertilization.

Males one to seven days of age were mass mated to virgin Oregon-R females which had been stored at 10°C. for one week prior to mating. The males were then discarded and the females randomly divided into two groups. The first of these groups was placed in individual vials and allowed to lay eggs immediately. The second group

of females was returned to cold storage at 10°C. for 15 days. The cold storage postponed normal development of the ovaries of the female and reduced sperm loss associated with egg laying. After the storage period, the stored females were placed in individual vials and allowed to lay eggs. Both the stored and non-stored females were transferred to fresh vials after 6 days and were discarded after 12 days. All stocks were maintained at 22°C. when they were not being stored.

Of the five lethals tested, only one showed a deviation from expected values:

| | Curly | lethal | |
|---|---|---|---|
| Non-stored | 4,822 | 4,136 | 8,958 |
| Stored | 4,500 | 4,401 | 8,901 |
| Total | 9,322 | 8,537 | 17,859 |

When the data for this lethal were submitted to a 2x2 contingency $\chi^2$ test, a Chi-square value with an associated probability of less than .01 was obtained. It may be seen in the non-stored group that more individuals of the Curly genotype are recovered than those of the lethal genotype. Upon storage this variation is reduced to the point where the difference between the two lethals is not significant.

The deviation might be explained by assuming an initial advantage of the Curly-bearing sperm over the lethal-bearing sperm which disappears or is reversed after cold storage. It may be premature at this time to suggest a mechanism, but a differential survival of sperm could account for the deviation.

Trosko, J. E., and A. F. Yanders. Cold storage effect on irradiated Drosophila sperm.

With reference to possible recovery in irradiated Drosophila sperm, Novitski (Progress Report, University of Rochester, 1947), Oster, and Von Borstel (noted in discussion p. 207, J. of Cell and Comp. Physiol., suppl., 1961) reported that they found no appreciable change in the mutation frequency after storage of the inseminated females. On the other hand, Herskowitz (Rec. Genet. Soc. Amer., 1957) found an increase in translocation after 18 days of storage, and Novitski (DIS 35, 1961) found an increase of lethals after a post treatment cold shock. It has been noticed in our laboratory that females inseminated by either irradiated or unirradiated males and stored for two weeks at 10°C. had fewer offspring when returned to normal temperature. The reduction in number of offspring was approximately 50% for the unirradiated sets and 70% for the irradiated sets. However, a number of females were dissected immediately upon removal from the cold room at the end of the two weeks and their ventral receptacles were relatively full of sperm, suggesting that the stored females lose sperm after they are returned to normal temperatures and not during the cold storage period.

Experiments were designed to test whether this reduction in number of offspring was related to the induced mutation frequency. Three-day-old Oregon-R males were treated and mass mated to three-day-old Muller-5 females for 24 hours. The females were separated and randomly divided into two groups; the first group was allowed to lay eggs immediately, and the second group was stored at 10°C. (or, in one case, 15°C.) for two weeks, and then returned to the normal temperature for laying. The frequencies of lethal X chromosomes in the progeny of these groups are as follows:

| | Non-stored | | Stored | | | |
|---|---|---|---|---|---|---|
| Exp. | lethals/normal | % lethal | lethals/normal | %lethal | $\chi^2$ | p |
| A[1] | 297/2851 | 9.43 | 44/346 | 11.28 | 1.35 | .25>P>.10 |
| B | 91/1008 | 8.28 | 37/284 | 11.52 | 2.76 | .10>P>.05 |
| C | 94/1036 | 8.32 | 64/519 | 10.98 | 2.94 | .10>P>.05 |
| D[2] | 252/2722 | 8.84 | 148/1276 | 9.19 | .15 | .75>P>.50 |
| E | 225/2320 | 8.47 | 163/1611 | 10.39 | 4.07 | .05>P>.025 |

1 Pooled results of three runs
2 Storage at 15°C. rather than 10°C.

With the use of Sir Ronald Fisher's method of combining separate probabilities, a $\chi^2$ value of 19.37, with 10 degrees of freedom ($.05>P>.025$), is obtained. These results suggest that cold storage of the inseminated females increases the frequency of recoverable mutations. If this increase is real, two alternative hypotheses are suggested: first, that cold temperatures following radiation actually enhance the radiation damage during storage by suppression of a normal repair system or by shunting "non-lethal", but damaged, sperm into the lethal class; or second, that a type of non-random sperm loss occurs during or soon after storage, so that the increased frequency of recoverable mutations reflects a differential survival or fertilization rate of affected versus non-affected sperm, rather than a true increase in radiation damage. Experiments which are now in progress should enable us to test which, if either, of these hypotheses is supported.

Hillman, Ralph. Competition and expressivity in Notch-deformed. The variability in expression of the phenotypic abnormality Notch-deformed has been described previously (Hillman, 1961, Genetics 46:1395; 1962, Genetics 47:11). The temperature effect which was reported as the main determinant of the expressivity of the altered genotype was admitted to be only one of several environmental variables which were affecting the degree of abnormality of the head capsule. One of the variables suspected of being uncontrolled in the experiments was a competitive effect of developing larvae on the development of the deformed head capsule.

In order to test this assumption the $Df(1)N^{8}/In(1)dl^{49}$, y Hw $m^2$ $g^4$ stock which was reported in the first of the above references was utilized. At the time of this current work, this $N^{8}$-deformed stock had been selected for fifty-six generations and was consistently showing a low frequency of head capsule abnormalities. Vials containing 25 ml of standard cornmeal-molasses medium were prepared and seeded with live yeast. From one to five virgin females, together with twice their number of males, were placed in each vial. The experiment was carried out at 18±1°C. After two weeks parents were removed from the vials. Progeny were observed twenty days after egg laying had begun. The flies were collected and examined at two to three day intervals until no more adults were emerging. The results, in terms of eye abnormalities only, are shown in the Table.

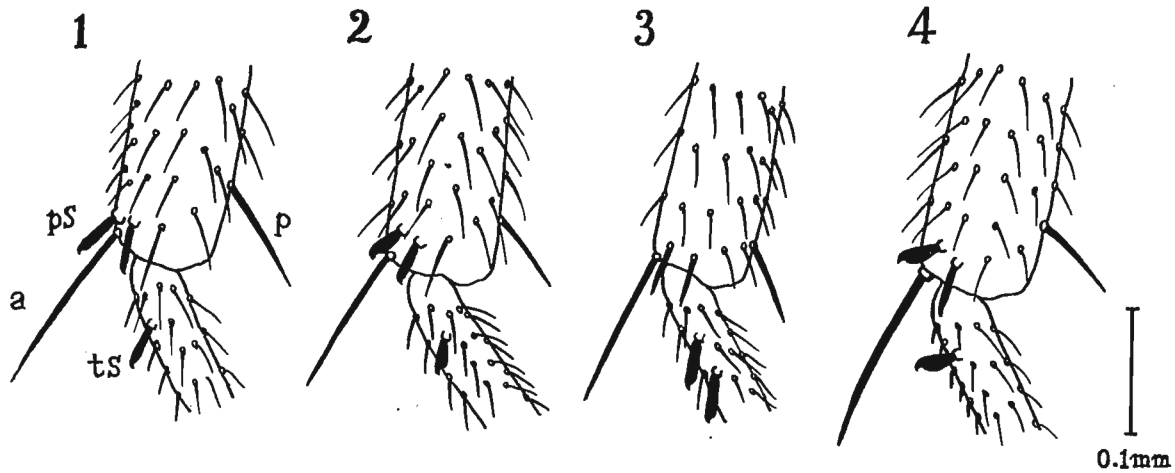
| Females/vial | Total adults | Total Notch | Eyes scored | Eyes abnormal | Freq. abnormality |
|---|---|---|---|---|---|
| 1 | 120 | 59 | 118 | 37 | 0.314 |
| 2 | 175 | 74 | 148 | 31 | 0.208 |
| 3 | 226 | 112 | 224 | 31 | 0.139 |
| 4 | 202 | 82 | 164 | 22 | 0.134 |
| 5 | 234 | 110 | 220 | 21 | 0.095 |

It is clear that an increase in the number of females per vial, and consequently in the number of developing organisms, results in a decrease in the expressivity of Notch-deformed. In these preliminary results it is impossible to determine if the decrease in the frequency of eye abnormalities is due to 1) an increased death rate of abnormal flies under crowded conditions, 2) an actual change in the physiology of the developing organism which reduces the phenotypic expression, or, 3) a change in the composition of the medium which would secondarily affect the physiological conditions of development. The data show a high mortality under crowded culture conditions. This is supported by the fact that the food in crowded cultures had been worked into more deeply than the food in vials containing only one or two females. However, the mortality involves all individuals in the culture since the frequency of Notch females to total adult flies appears the same in both crowded and uncrowded cultures. Since only Notch females show the abnormality, any explanation based on mortality must take into account the non-Notch flies. Investigation of these alternative explanations is now in progress.

Okada, T. Hooked scaly bristles found on the male mid legs in some Drosophilid flies.

A special kind of hooked scaly bristles, hitherto seemingly overlooked, was found on the male mid legs of some drosophilid flies. These scales are located in one or sometimes two pairs at the lateral sides of apical bristle of tibia and in one or rarely two in number near the proximal end of metatarsus along its mid-dorsal line. The former will be named parapical scales (ps) and the latter tarsal scale (ts). The taxa in which these scales have been found are the suzukii-⌈suzukii (fig. 1), pulchrella⌉, fiscusphila-⌈ficusphila (fig. 3)⌉, and takahashii-⌈takahashii, nepalensis (fig. 2), lutea⌉ subgroups of the melanogaster species group and also in Tanygastrella gracilis Duda (fig. 4). The scales in suzukii and ficusphila subgroups are slender, while they are thicker and shorter in takahashii subgroup and Tanygastrella gracilis. These scales are seemingly replaced by the normal spiny bristles in the tibia of ficusphila and in both tibia and metatarsus of the females of those species mentioned above as well as of the both sexes of the other species of Drosophilidae examined so far. The author's previous assumption that the takahashii subgroup is closely related to the suzukii subgroup and that the ficusphila subgroup is an offshoot of the suzukii-takahashii line may be agreed also from the present aspect that they are unique among the subgenus Sophophora in having the scales. Furthermore, hitherto not fully clarified systematic position of Tanygastrella becomes somewhat definite. That Duda (1926. Suppl. Ent., 14:53) placed it under the genus Drosophila at most as a subgenus is valid, and that its closest relative is the takahashii subgroup is highly plausible, as these two taxa share in common the same type of the scales. It is, however, safe to wait to establish generic synonymy, as stated by Wheeler (1959. Univ. Texas Publ., 5914:185), until sufficient knowledge about T. hypopygialis Duda can be obtained. Finally, D. unipectinata Duda should better be excluded from the suzukii subgroup in view of the fact that no scales can be found in this species.

Figure Lateral view of male mid legs (apex of tibia and proximal part of metatarsus) to show disposition of scaly bristles in 1. Drosophila suzukii (Matsumura), 2. D. nepalensis Okada, 3. D. ficusphila Kikkawa and Peng, and 4. Tanygastrella gracilis Duda. a, apicals; p, preapicals; ps, parapicas scale; ts, tarsal scale.




Okada, T. Ethological significance of the thorn-like warts on wings in the steganine flies.

The flies belonging to the subfamily Steganinae (Stegana Meigen, Amiota Loew, Leucophenga Mik etc.) of the family Drosophilidae have been known usually to have special kind of sensilla, named "thorn-like bristles" (Sturtevant, 1927. Phil. Journ. Sci., 32:361) or "thorn-like warts" (Wheeler, 1952. Univ. Texas Publ., 5204:165), on the underside of the third

costal section of wings. This kind of sensilla seems to be a character adapted to their special ethological manners of quickly flying in vertical circles and of starting to fly in caudodorsal direction, in contrast to the slow and linear flying and starting to fly in cephalodorsal direction which are characteristic to most of the general belonging to the subfamily Drosophilinae. Concentration of sensory organs to the body apices toward the direction of locomotion is a general tendency found in the animal kingdom and is thought to have been achieved by selection and adaptation in the course of anagenetic evolution. This certainly holds good also here, as the localization of the warts near the wing tips of the flies corresponds to the direction of their starting to fly, and as their special manner of quick flying may also be favored by this localized distribution of the warts.

Okada, T. 'Law of Unspecialized' applied to the family Drosophilidae. The subgenus Paradrosophila Duda (or Pholadoris Sturtevant) has been thought to be one of the most unspecialized and primitive taxon among the genus Drosophila, characteristic in having, for example, low phallosomal index, simply elliptical or weakly coiled testis and short and only once or twice folded ventral receptacle. Also the low grade of accumulation of pteridine pigments in testis in this subgenus is proved by Throckmorton (1962. Univ. Texas Publ., 6205:459) to be a biochemically primitive feature. He (ibid.:319) believes that "the stem population of this subgenus originated at an earlier time than did those for the other subgenera". Moreover, the presence of well developed first abdominal sternites in some species of this subgenus, recognized by Wheeler (1960. Annal. Ent. Soc. Amer., 53:133), and a subgeneric character of well developed prescutellars may be the features bridging Steganinae and Drosophilinae or, in other words, common ancestral between the two subfamilies. Furthermore, the species of this subgenus have generally small bodies. Possession of these simple and ancestral features as well as small bodies suggests Cope's Law of Unspecialized to be applicable here to the effects that 1), the present status of cladogenetic splitting of the family Drosophilidae had originated from a stock of small ancestral form resembling Paradrosophila and that 2), the future evolutionary potentiality of the family is best stored in this subgenus.

Smith, Patricia A. and R. C. King. Three new tumorous fused mutants. Three new fused mutants were recovered by Myrtle J. Fahmy among the progeny of males irradiated with $Co^{60}$ gamma rays. X1781 (here designated $fu^{62f-1}$) and X1803 ($fu^{62f-2}$) arose in a sperm sample delivered 0-3 days after treatment and X1845 ($fu^{62f-3}$) 3-6 days after treatment. All alleles resemble fu with regard to its effects in both sexes upon wing venation and on ocellar and ocellar bristle number. The wings of males and females are also held outstretched as in fu. Ovarian tumors occur in the ovaries of homozygous females, and the tumor incidence increases with age as is demonstrated in the following Table:

Tumor incidence as a function of age of female homozygote

| | 2.5 days | N | 10 days | N |
|---|---|---|---|---|
| $fu^{62f-1}$ | 1.3% | (77) | 23.9% | (134) |
| $fu^{62f-2}$ | 8.1% | (185) | 41.2% | (199) |
| $fu^{62f-3}$ | 8.5% | (246) | 58.7% | (259) |

N equals the number of ovarian egg chambers observed.

Crosses were made to produce females containing different pairs of fused alleles, and their ovaries were examined eight days after eclosion. All hybrids showed both the fused wing phenotype and ovarian tumors. The females studied were of the following 19 genotypes: $fu/fu^{59}$, $fu/fu^{ff}$, $fu^{59}/fu^{ff}$, $fu/fu^{57a}$, $fu^{59}/fu^{57a}$, $fu/fu^{62f-3}$, $fu^{59}/fu^{62f-3}$, $fu^{ff}/fu^{62f-3}$, $fu^{57a}/fu^{62f-3}$, $fu/fu^{62f-2}$, $fu^{59}/fu^{62f-2}$, $fu^{62f-3}/fu^{62f-2}$, $fu/fu^{62f-1}$, $fu^{59}/fu^{62f-1}$, $fu^{ff}/fu^{62f-1}$, $fu^{57a}/fu^{62f-1}$, $fu^{62f-3}/fu^{62f-1}$, $fu^{ff}/fu^{62f-2}$, and $fu^{62f-2}/fu^{62f-1}$.

Ronen, A. The effect of heterologous inversions on spontaneous and induced somatic recombination.

In Drosophila melanogaster, as well as in other species of the same genus, heterozygosity for inversions in a given chromosome may result in an increase in crossing over values in other, non-homologous, chromosome.

One of the current theories which accounts for this phenomenon (Oksala, 1958. Cold. Spr. Harb. Symp. Quant. Biol., 23:197-210) is based on the assumption that the frequency of recombination in the proximal region of a chromosome is positively correlated with synapsis in that region. Chromosomes in which this region is synapsed will have it recombined more often than other chromosomes, in which it is less intimately synapsed or where it shows asynapsis. It is postulated in Oksala's theory that while in the structurally normal nucleus there is a fair chance for the proximal part of each chromosome to pair with heterologous chromosomes (illegitimate pairing), the presence of an inversion in a given bivalent increases the chance that other bivalents will synapse legitimately.

On the other hand, Schultz and Redfield (Schultz and Redfield, 1951, Cold Spr. Harb. Symp. Quant. Biol., 16:175-197) and Schultz (see discussion in Oksala, 1958) maintain that the opposite is true: the presence of inversions with their pairing difficulties may create a situation where legitimate pairing is interrupted, and this may lead to an increase in crossing over in the adjacent, paired regions. It is thus the chromosome that is allowed to pair to the full extent, which will show a minimum amount of crossing over.

In the course of an analysis of this point, a study was made of the influence of autosomal inversions on spontaneous and induced somatic recombination in the X-chromosome.

Females carrying a y $w^a$ spl X-chromosome with normal second- and third-chromosomes of an isogenic, Berlin-A stock, were mated to sn males carrying the inversion Curly and Dichaete. In the males, the normal autosomes were also of the Berlin-A isogenic stock.

Their daughters were collected as third-instar larvae, when climbing on the walls of the culture bottles to pupate, and were irradiated with 1170 r of X-rays. They were then placed in fresh culture bottles and allowed to pupate. The unirradiated sisters of these females served as controls.

The adult flies were examined for the appearance of yellow and singed single spots, and yellow-singed twin spots, due to somatic crossing over between the loci y and sn, and/or between sn and the centromere.

Among the unirradiated flies, the recombination values were about 50% higher in the presence of the two inversions than in their absence (Table 1). This effect was less marked when, in a preliminary experiment, either one of the inversions was singly introduced into the females.

In structurally normal flies, irradiation increased the mitotic recombination rate, as was expected. However, no higher total recombination values were found in the irradiated females which carried the inversions, as compared with their normal sisters (Table 1). The net induced recombination rate is, therefore, lower in the presence of the heterologous inversions than in normal cells.

The fact that the relative frequency of the various single and twin spots remained unchanged in the irradiated flies (as compared with the controls) is, to some extent, an indication that induced mutation had no detectable role in increasing the frequency of spots.

No evidence was found for the occurrence of somatic crossing over, either spontaneous or induced, distal to the sn locus, in any one of the four groups of flies. It may be assumed that most of the recombination events occured near the centromere, in or near the proximal heterochromatin (Kaplan, 1953. Genetics, 38:630-651; Brosseau, 1957. J. Expl. Zool., 136:567-593.

X-rays were relatively less efficient in producing somatic recombination in this region in the presence of Cy and D than in their absence (1.24 induced spots per fly in normal flies, as compared with 0.87 induced spots per fly in the presence of both inversions). This could be explained on the assumption that in the presence of autosomal inversions, pairing of the proximal ends of the two X-chromosomes is less intimate than usual.

This effect of the heterologous chromosomes may be due to their own pairing difficulties, increasing their chance to engage in pairing with non-homologous chromosomes and reducing the potential for structurally homozygous chromosomes to pair.

If this is true, heterologous inversions affect spontaneous crossing over in a given bivalent by producing pairing difficulties in certain sections of this bivalent. Despite its dependence on synaptic contact, crossing over in a given pair of homologues may thus be increased by partial asynapsis. X-ray induced crossing over, depending on a different mechanism, will not be increased by partial asynapsis.

The data suggest that, in D. melanogaster, heterozygosity for inversions interferes with the legitimate pairing in the proximal, heterochromatic, regions of heterologous chromosomes. In order to account for the subsequent increase in spontaneous crossing over in these chromosomes, it has to be assumed that pairing difficulties in parts of a given bivalent can cause a recombination increase in neighbouring regions. Such a mechanism could be accounted for by Darlington's (1935) "torsion" or White's (1954) "frontier" theories.

The data also support the view that spontaneous and induced crossing over are due to different causes, since they can be shown to differ in their response to the presence of heterologous inversions.

Table 1

The frequency of spontaneous and induced spots in y $w^a$ spl/sn; +/+; +/+ and y $w^a$ spl/sn; Cy/+; D/+ females (all types of spots combined).

| Treatment | Genotype (autosomes) | | | |
|---|---|---|---|---|
| | +/+ ; +/+ | | Cy/+ ; D/+ | |
| | No. of flies | mean spot frequency | No. of flies | mean spot frequency |
| unirradiated | 100 | 0.66±0.07 | 94 | 0.98±0.11 |
| irradiated | 147 | 1.90±0.11 | 98 | 1.85±0.12 |

Begg, M., and W. J. Cruickshank. Partial analysis of Drosophila larval haemolymph.

Partial analysis of haemolymph of last instar larvae of Canton-S Drosophila melanogaster gave the following results: Osmotic pressure equivalent to 1.05% NaCl; pH 6.6 - 6.7; $Na^+$ 56.5 mM/l; $K^+$ 40.2 mM/l; $Ca^{2+}$ 8 mM/L; $Mg^{2+}$ 20.8 mM/l; $Cl^-$ 42.2 mM/l; $PO_4^{3-}$ 2.8 mM/l; amino acids (approx.) 137 mM/l. Na:K ratio, 1.4. Ca:Mg ratio, 0.38.

Browning, Luolin S. and Edgar Altenburg. Slight or doubtful mutagenic effects of some biologically highly reactive compounds when applied to the polar cap cells of Drosophila.

Because of the importance attributed by some workers to free-radicals in the induction of mutations, a study was made of the mutagenicity of two free-radical forming compounds (benzaldehyde and ethyl-diazoacetate) when applied to the polar cap cells of Drosophila. A third compound (azaserine) was tested because it had been shown (by Buchanan) to interfere with the synthesis of the purine ring of inosinic acid and in addition was a highly reactive antibiotic. The chemical action of two of the compounds tested (ethyl-diazoacetate and azaserine) is catalyzed by ultraviolet. The mutagenic effect of a combined treatment of each of these two and a very low dose of ultraviolet (barely mutagenic) was therefore tested in addition to each compound by itself, to see whether the combination acted synergistically. Also, feeding azaserine to the developing flies was tested. Lethal mutations in the second pair of autosomes were detected by means of Muller's sifter technique.

As shown in the table below, the mutagenic effect of the three chemicals is not marked, even in the case of the combined chemical and ultraviolet tests, although azaserine gives a low but significant rate either when applied to the polar caps (5-10% aqueous solution) or when fed (85 mg. in 30 ml. of food).

Possibly all of these compounds are somewhat mutagenic but factors such as penetration of the agents through the vitelline membrane to the pole cells, the short exposure times caused by the limited duration of the polar cap stage of development, possible destruction of the agent by cellular enzymes, low dosage of the agents necessitated by their toxicity, and so forth may be at work in reducing the possible per cent of induced mutations.

Table

| Agent* | No. males tested | No. Chromosomes tested | No. lethals | Recovered mutation rate (%) | Induced mutation rate (%) |
|---|---|---|---|---|---|
| Benzaldehyde | 16 | 351 | 6 | 1.7±0.8 | 1.0±0.8 |
| Ethyl-diazoacetate | 39 | 530 | 5 | 0.9±0.4 | 0.2±0.4 |
| Azaserine | 30 | 721 | 15 | 2.1±0.6 | 1.4±0.6 |
| Ethyl-diazo. + u.v. | 30 | 488 | 11 | 2.3±0.7 | 1.6±0.7** |
| Azaserine + u.v. | 36 | 887 | 17 | 1.9±0.5 | 1.2=0.5** |
| Azaserine (fed) | 40 | 785 | 15 | 1.9±0.6 | 1.2=0.6 |
| Control *** | | | | 0.7=0.1 | |

* Administered to polar caps except as otherwise indicated.
** Not significantly different from the u.v. only rate.
*** Control rate based on combined results (totalling over 6,500 tested chromosomes) in this and other experiments.

(This investigation was supported by the American Cancer Society, Grant No. e-G-9B, and later by U.S. Public Health Service Research Grant No. C-5961.)

Brosseau, George and J. E. Divelbiss. An attempt to find gene conversion in the absence of crossing over.

An unequivocal answer to the question of whether gene conversion requires crossing over would be obtained if gene conversion could be found when crossing over is absent. Drosophila males provide a potentially favorable test situation although the failure to find conversion would not provide a definitive answer. The brown locus was selected for the test because it is a compound locus which has yielded both recombination and conversion in heterozygous females. The recombination between the two known subunits and gene conversion show about the same frequency, about 1/100,000. The alleles used were $bw^{75}$ and $bw^{59}$ because they occupy recombinable sites, both give conversion and they have a phenotypically distinguishable double mutant. px $bw^{75}$ sp/+ $bw^{59}$ + ♂♂ were crossed to px bw sp ♀♀. No heterologous inversions were used. A few heterozygous ♀♀ were also tested as controls. Since an exact count was not desired, only every 10th bottle was counted and the total number of flies examined estimated from these counts. An estimated 1,250,000 progeny from heterozygous males were examined and no conversions found. 280,000 progeny from the control females yielded no conversions and 1 cross over. Thus the results were rather unsatisfying. The interpretation is uncertain, for although no conversion was found in male Drosophila where crossing over is absent, it is also possible that some other condition necessary for gene conversion is also absent in the male, e.g. the proper synaptic association. It does seem clear, however, that if conversion does occur in Drosophila males, the frequency of this event is much lower than it is in females. A much larger scale experiment would be necessary to demonstrate it.

Keller, E. C., Jr. and Edward Glassman. Investigation of a strain of Drosophila melanogaster possessing low xanthine dehydrogenase activity.

From a survey of a series of wild type strains of D.m., several lines were obtained which possessed low xanthine dehydrogenase activity. One of these strains was chosen and critically examined to determine if the cause of this low activity had a discernable genetic basis. It was found that the low activity (about 25% of that in wild type Oregon-R) was due to a single recessive gene located on the left arm of the third chromosome probably between 15 and 30. The linkage data for this gene (lxd, low xanthine dehydrogenase) was obtained with a series of dominant markers. Further linkage studies are being carried out with recessive markers in this region of the third chromosome.

Bennett, J. and James A. Bort. Behavioral differences in selected Drosophila lines.

Eight different strains of Drosophila melanogaster from this and other laboratories were examined to test the hypothesis that resistant lines might actually be hypersensitive to DDT and move off of DDT contaminated surfaces thus avoiding a lethal dose. All flies were raised on an agar, yeast, sugar, propionic acid medium (Mittler and Bennett, DIS 36: 131-2) and at a temperature of 25° C.

In the first experiment test vials (lined with a cylinder of DDT impregnated paper, but without paper on the bottom) were placed on a glass shelf, and the positions of the flies recorded, using a mirror, during the first and last five hours of the eighteen hour test periods. The various lines were then scored according to the amount of time the flies spent on the DDT papers, versus the time spent on top or bottom of the vials which were DDT free. Three different concentrations of DDT were used, the rank orders changed only slightly between concentrations, but the spread of values was small at the low concentration and large at the highest concentrations (6% and 45% respectively at 1 and 625 ugm. DDT/$cm^2$).

Table 1 gives the results of the test vial experiment. The stocks are listed in order of DDT resistance (determined at the same time). An apparent weak relation appears between $LD_{50}$ and amount of time spent on DDT. On consideration we feel that part, or all, or this effect may be an artifact of scoring. A fly that had absorbed enough DDT to fall from the sides of the vial, but which could stillwalk on the horizontal bottom was counted as a live normal fly. However for any given exposure time there should be more such flies in a sensitive than in a resistant line, thus giving false indication of a lower preference for the DDT paper.

The second experiment involved use of a maze in which the flies were presented with the choice between entering a DDT lined tunnel and an uncontaminated tunnel, at successively higher dosages of DDT (1, 25, and 625 ugms DDT/$cm^2$). Figure 1 presents the data for the maze experiment, and Figure 2 is a representation of the maze.

The upper four lines in Figure 1 have been selected for DDT resistance. The $LL_2T$ line was slected for DDT sensitivity. The other lines represent a spectrum of laboratory standard lines. Figure 1 reveals a curious relation between the shape of the Dosage-Response Curve for each line and the percentage entry into the DDT lined tunnels. The three most resistant lines have D-R curves that are concave upward and have a relatively high frequency of entry into the DDT tunnels. The weakest of the resistant lines has an essentially linear D-R curve and an intermediate rate of entry into the DDT tunnels. The selected sensitive line $LL_2T$, however, has a D-R curve that is concave downwards and has a relatively low frequency of entry into the DDT tunnels. The standard laboratory lines however fall into both patterns. Oregon-R and M-5;Cy/Pm;Sb/Ubx follow the pattern of $LL_2T$. But cn bw; e follows a pattern typical of the resistant lines in that it has a linear D-R curve and a high rate of entries into the DDT tunnels. This could be interpreted as an indication that the cn bw; e background is of the resistant type, but that the physiological result is depressed by the homozygosity for the three recessives.

We conclude that these results offer no support to the idea that the DDT resistance in the selected lines is based on a behavioral factor of avoidance of DDT. It is apparent that behavioral differences exist among the lines tested in regard to choice of DDT lined or free tunnels in a maze, but these do not seem to be related to absolute degree of resistance. Similarly it appears that differences noted between lines selected to be DDT sensitive or resistant were also found in the unselected laboratory standards. Finally the different lines demonstrated great differences in the proportion of flies traveling through the maze in the allowed time, though this value did not show any direct relation with DDT resistance.

Table 1

Time on DDT Paper in Test Vial and Resistance

| Stock | $LD_{50}$ ($Log_5$Unit Dose DDT) with 95% Confidence limits | | | Time on 625 ugm DDT/$cm^2$ paper in test vial | |
|---|---|---|---|---|---|
| | $LD_{50}$ | Limit | N(Flies) | % | N(observations) |
| $HL_2TBM$ | 4.31 | ±1.45 | 280 | 59 | 591 |
| ORS 1001 | 4.16 | ±1.83 | 280 | 69 | 648 |

Table 1 (Cont.)

| | $LD_{50}$ | Limit | N(Flies) | % | N(observations) |
|---|---|---|---|---|---|
| Brown eye-R | 3.86 | ±1.60 | 287 | 72 | 589 |
| $HL_1QBM$ | 3.17 | ±1.83 | 288 | 44 | 585 |
| $LL_2T$ | 2.48 | ±1.26 | 285 | 38 | 531 |
| Oregon-R | 2.20 | ±0.93 | 266 | 27 | 462 |
| M-5; Cy/Pm; $Sb/Ubx^{130}$ | 1.89 | ±0.96 | 267 | 29 | 486 |
| cn bw; e | 1.86 | ±1.65 | 266 | 35 | 454 |

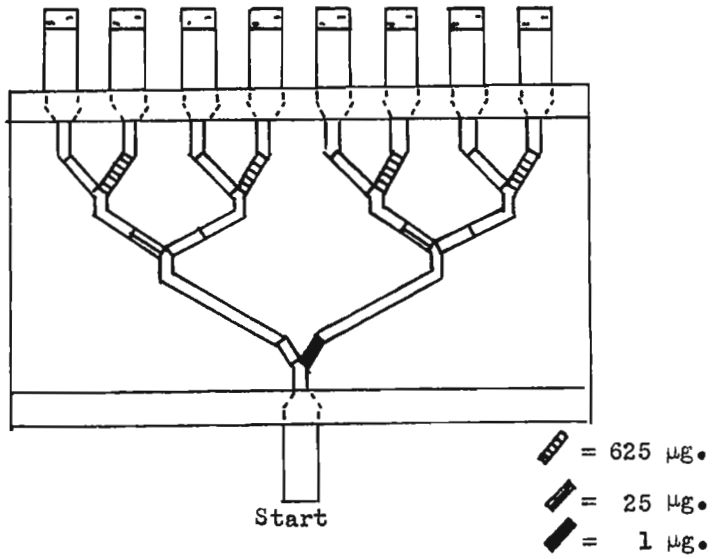
Figure 1

DDT Dose-Response Relation and DDT Maze Behavior

| Stock - in order of resistance. | Dose - Response Relation (DDT dose in µgms./$cm^2$) Dose 0 % Kill 100 | N | Entry into DDT lined tunnel in maze. % | N | Flies completing trip through maze. % |
|---|---|---|---|---|---|
| | 1 | 95 | | | |
| $HL_2TBM$ | 25 | 94 | 61.4 | 163 | 43.5 |
| | 625 | 91 | | | |
| | 1 | 94 | | | |
| ORS 1001 | 25 | 92 | 59.3 | 140 | 26.9 |
| | 625 | 94 | | | |
| | 1 | 95 | | | |
| Brown eye-R | 25 | 96 | 55.0 | 129 | 26.3 |
| | 625 | 96 | | | |
| | 1 | 93 | | | |
| $HL_1QBM$ | 25 | 96 | 49.5 | 160 | 35.6 |
| | 625 | 99 | | | |
| | 1 | 95 | | | |
| $LL_2T$ | 25 | 96 | 43.4 | 180 | 38.4 |
| | 625 | 94 | | | |
| | 1 | 89 | | | |
| Oregon-R | 25 | 87 | 50.8 | 124 | 43.2 |
| | 625 | 90 | | | |
| | 1 | 89 | | | |
| M-5; Cy/Pm; $Sb/Ubx^{130}$ | 25 | 89 | 47.0 | 49 | 13.0 |
| | 625 | 89 | | | |
| | 1 | 88 | | | |
| cn bw; e | 25 | 88 | 66.6 | 138 | 42.3 |
| | 625 | 90 | | | |

Figure 2

A diagram of one of the mazes used in the behavioral analysis.




Maze cast from plaster of Paris. Tunnels lined at choice points with filter paper impregnated with indicated amount of DDT, or with clean filter paper as control. Receiving vials contain food, starting vial dry.

Aronson, Margaret. Further description of the third chromosome of $T_2(1;3)^K$.

The translocation $T_2(1;3)^K$ was found and described by Krivshenko (DIS 30:76) who has since obtained a strongly viable and fertile stock homozygous for this translocation. He found that the third chromosome was made up of a rod and a J-shaped element in ganglia cells, but that it appeared to be normal in the salivaries. He also showed that the third chromosome bears a convenient marker, the normal allele of yellow ($y^+$).

Because the salivary chromosome shows no aberration, the break must have been in the region of the chromocenter. From crossover data on this chromosome the point of genetic breakage was found to be to the right of the most proximal 3L marker available - radius incompletus, (47.0), and to the left of pink-peach, (48.0), on 3R.

To determine the arm in which the third chromosome break occurred, $T_2(1;3)^K$ was crossed separately with five different T(3;4) translocations in which the location of breaks was known (Pasadena stocks; T(3;4)A12, T(3;4)A28, T(3;4)c and T(3;4)e; Bloomington stock; T(3L·4L;4R·3R)(Dubinin2). The pairing pattern of the heterozygous third chromosomal elements was observed in ovarian tissue where somatic pairing is strong. Thus, when the T(3;4) chromosome was broken in 3R, the two J-shaped elements (3L) and the two rods (3R) paired together. When the T(3;4) chromosome was broken in the left arm however, each J-shaped element paired with a rod.

To determine which of the fragments of $T_2(1;3)^K$ carried the normal allele of yellow, two third chromosomes having "separated arms", one arm of which originated from $T_2(1;3)^K$ and the other from Dubinin's translocation T93L·4L;4R·3R) were synthesized. Two crosses were made to $y^2/w^a$;DCxF/P(Dfd) females of males carrying only one of the fragments of the $T_2(1;3)^K$ chromosome. When the 3L fragment of $T_2(1;3)^K$ was present, one-half of the male progeny were non-yellow, but all males were yellow when the 3R fragment was present. The $y^+$ marker is therefore carried by the 3L fragment, which is the J-shaped element of $T_2(1;3)^K$.

Thus, in all cases the cytological and genetical evidence were consistent and showed that the $T_2(1;3)^K$ chromosome had been broken in the centromeric heterochromatin of the right arm, that the acentric part of 3R distal to the break was trans-

located onto the centromeric part of X, and that the proximal centromeric part of 3R was capped by the telomeric part of X which carries the $y^+$.

The amount of X chromosomal material in each fragment of the $T_2(1;3)^K$ chromosome could be estimated as follows. Because the $T_2(1;3)^K$ translocation was recovered from an X-rayed scute-8 X chromosome, the centromeric part of X (onto which the 3R distal to the break is translocated) consists of XR, centromere, and probably a small proximal part of X heterochromatin. Since the translocation does not carry the scute allele, the proximal break of X could not have been to the left of the scute-8 break at 20D1, and may have been anywhere from 20D1 to the centromere. The telomeric X chromosomal material which capped the centromeric part of 3R consists of the distal, euchromatic tip of the X bearing $y^+$, and probably includes X heterochromatin extending from the proximal $sc^8$ break at 20D1 toward the nucleolus organizer but not including it. Since the translocation does not have the bobbed locus, and if the tentative assignment of bb at 20C2 by Cooper is considered, the limits between which the break must have occurred are 20D1 to 20C2, or somewhere in the proximal third of hB according to the terminology of Cooper for the X heterochromatin (Chromosoma, 10:535-568, 1959).

In prophase neuroblast nuclei, the distance from the centromere to the tip of translocated material at the end of the J-hook is about one-sixth the length of the complete left arm. Of this, probably not more than one-third is X chromosomal material and two-thirds is that part of the third chromosome between the break in 3R and the third centromere. This two-thirds represents basal heterochromatin which must be within the chromocentral region of the salivary chromosome.

Redfield, H., and J. Schultz. Recombination for the X of structurally standard triploids of D. melanogaster.

Inquiries have been received concerning results derived from the cross of $y^2$ cv v f car bb/+/+ triploid females by $y^2$ cv v f B car bb males, and reported in part in Table 9 of the 1951 paper of Schultz and Redfield (CSHSQB, 16). In this table results were not given for zygotes receiving 2X chromosomes from the mother. We therefore give here additional data and correct an error in tabulation. It must be realized that some difficulties are encountered among the chromosome types listed below with regard to classification of mutants, differential viability, etc., which make comparisons involving several of these types rather tentative. The data for non-Bar female exceptions, from 2X1A eggs, may be listed first. As is seen, N is a respectable number; furthermore presumably special viability and classification complications are absent here. Bobbed is not represented since it is not detectable in the presence of the Y. For each pair of apparent contrary classes the class with $y^2$ is given first.

2XY2A Zygotes

| Classes | | | | | | % homozygosis | |
|---|---|---|---|---|---|---|---|
| 0 = | 1 + | 3,477 | 1,3 = | 0 + | 10 | $y^2$ = | 2.7 |
| 1 = | 65 + | 0 | 2,3 = | 0 + | 7 | cv = | 1.5 |
| 2 = | 18 + | 1 | 2,4 = | 0 + | 2 | v = | 0.9 |
| 3 = | 11 + | 3 | 3,4 = | 0 + | 1 | f = | 0.3 |
| 4 = | 2 + | 0 | | | | car = | 0.1 |
| 1,2= | 0 + | 11 | | N = 3,609 | | | |

The percentage of homozygosis, calculated directly, provides a measure of sorts of the distance of each gene from the spindle fibre attachment. It may be seen that within their own range a higher value for the distal end of the chromosome ($y^2$ to cv) is obtained relative to the semi-distal end (f to car), 2.7 minus 1.5 being some 6 times as great as 0.3 minus 0.1, as is within their range true for the 1X1A gametes of Table 9 (given by regular males and females - the recombination value for $y^2$ to cv being about 3 times the value for f to car); but on the other hand the factor is very near that found for 1X2A gametes of Table 9 (the Bar intersexes, see also modified values given below, for $y^2$ to cv recombination is about 5 times that for f to car). It may be noted that 24 superfemales (3X2A) were additionally obtained from similar 2X1A eggs fertilized by XA sperm instead of by XY sperm; these were all wild type in appearance.

Eggs of the composition 2X2A produced either Bar triploids (3X3A, N = 551) or non-Bar intersexes (2XY3A, N = 549). Among the $F_1$ triploids the apparent classes were as follows.

3X3A Zygotes

| Classes | | % homozygosis |
|---|---|---|
| 0 = 0 + 510 | 2,3 = 0 + 4 | $y^2$ = 4.7 |
| 1 = 19 + 0 | 2,4 = 0 + 1 | cv = 2.7 |
| 2 + 5 + 0 | 2,5 = 0 + 1 | v = 1.8 |
| 3 = 2 + 0 | 3,5 = 0 + 1 | f = 0.7 |
| 1,2 = 0 + 6 | | car = 0.4 |
| 1,3 = 0 + 1 | N = 551 | bb = 0.0 |
| 1,4 = 0 + 1 | | |

Here, as above for the 2XY2A zygotes, the ratio between $y^2$ to cv recombination which is proportional to 4.7 minus 2.7, and f to car recombination proportional to 0.7 minus 0.4, is high (i.e. about 7). The total number of triploids upon which the homozygosis values are based here is, of course, much smaller than the total for the exceptional females. However if one uses the data as they stand each value for homozygosis here is higher than the corresponding value given by the exceptional females. Since both types receive two X chromosomes from the triploid mother, the indication then is that the observed differences depend upon the accompanying disjunction of the autosomes. That is, on comparing these two types derived from 2X eggs, it is found that sister representatives of each of the mutant genes of the marked X appear in larger proportion in those eggs receiving the double set of autosomes, than is true for the eggs receiving a single set of autosomes.

Corresponding data for non-Bar intersexes, also from 2X2A eggs but fertilized by YA sperm, give the following apparent classes and homozygosis values.

2XY3A Zygotes

| Classes | | % homozygosis |
|---|---|---|
| 0 = 0 + 518 | 1,2 = 0 + 2 | $y^2$ = 4.7 |
| 1 = 18 + 0 | 2,3 = 0 + 3 | cv = 1.8 |
| 2 = 6 + 0 | | v = 1.0 |
| 3 = 2 + 0 | N = 549 | f = 0.0 |
| | | car = 0.0 |

It is of interest that the value here for $y^2$ is, as expected, identical with that derived from triploid zygotes. Other homozygosis values for these two types differ, but they depend upon smaller numbers.

Recombination values, as opposed to the homozygosis values necessarily used in dealing with offspring receiving two X's from the mother, are given in Table 9 of the 1951 paper for 1X1A eggs (856 regular Bar females and 749 regular non-Bar males) and for the 1X2A eggs producing Bar intersexes. Two points which perhaps have not been made clear should be explained about values shown in this table. In the first place non-bobbed classes only were used for the 2X3A intersexes since bobbed is usually lethal in such intersexes. (Bobbed classes gave a total of 14 individuals here.) Secondly both columns of recombination percentages of Table 9 involve a correction factor of 3/2 (following the earlier usage of Redfield, Genetics 15, 1930, for triploids in which one only of three homologues is marked); this is to be regarded as a convenient constant comparison with values from diploids, or with values from triploids in which recombination is almost exclusively between two structurally standard chromosomes of a homologous set in which the third chromosome is inverted (as in the ClB triploids of Table 10, where the values obtained seem to justify this usage). Obviously the correction factor should be taken into account when comparing these corrected values with the simple homozygosis values as given directly by the data for the 2X eggs.

There is an unfortunate error of tabulation in the column for 1X2A eggs in Table 9 of the 1951 paper which we have just now discovered and which we should like to correct here. This column should read:

(% recombination in) 1X2A eggs (taken from 2X3A zygotes)

| | | | |
|---|---|---|---|
| $y^2$ - cv | 15.4 | f - car | 3.6 |
| cv - v | 9.9 | car - bb | 3.3 |
| v - f | 7.7 | Total | 39.9 |

N = 1707

In addition to these Bar intersexes, 16 non-Bar supermales (XY3A) were also obtained from

such 1X2A eggs - 15 were wild type and one was $y^2$; they were not included since adverse viability effects are pronounced among supermales. The values given above differ somewhat from the values published in Table 9; but the only necessary change in the text would be to read "three-fourths" instead of the word "half" in the statement (p.190) "... although even at the distal end of the chromosome the value in the asymmetrical disjunctions is half what it is in the symmetrical ones".

Recombination values for the diploid controls, i.e. from $y^2$ cv v f car bb/+ mothers by $y^2$ cv v f B car bb males should be mentioned. Included here were 2,287 Bar $F_1$ females and 2,266 non-Bar $F_1$ males; these gave by customary computation the following uncorrected values. The bobbed to carnation recombination was, of course, derived from the females alone.

| | % recombination from diploid control | | |
|---|---|---|---|
| $y^2$ - cv | 11.2 | car - bb | 2.8 |
| cv - v | 21.6 | | |
| v - f | 20.2 | Total | 62.4 |
| f - car | 6.6 | N = | 4,553 |

General implications of the data presented in this note cannot be dealt with at the moment.

Barigozzi, C., A. M. Kravina, and M. Sari Gorla. Melanotic tumors, a character transmitted through both nucleus and cytoplasm in Drosophila.

Melanotic tumors are very common as a transmitted trait in Drosophila. Their transmission is controlled by factors localizable in most cases in the 2nd chromosome; they behave as recessive, and are generally referred to as tu.

During the past year two tumorous stocks have been found whose chromosomal factors have been localized on distinct sections of the 2nd chromosome, one of them (tu $B_3$) is located near the right end of the chromosome, while the other (tu $A_2$) proved to be localized near the left end of it. At the same time, using the method of chromosome replacing, a weak effect of the cytoplasm has been demonstrated, which seemed to be independent from the chromosomes.

Since several abnormalities in the transmission of the melanotic tumors made likely an influence of the internal environment upon the chromosomes, experiments have been made in order to test the existence of such an influence.

The first attempt has been brought about grafting ovaries of a tumorless marked stock into tumorous larvae. The results (C. Barigozzi, C. Halfer and G. Sgorbati, Heredity 17, 571, 1962) prove that the oocytes matured in the grafted ovary are capable of transmitting to the offspring (and persistently, during subsequent generations) the tendency to produce a proportion of tumors, whose anatomical site corresponds to that of the recipient stock (abdominal or thoracic tumors).

The transmission takes place through both gametes, thus either the transmissible principle (or unit) has penetrated the nucleus or is located in the small amount of cytoplasm present also in the sperm.

The transmissible unit may be conceived as a self-perpetuating particle, which might have hypothetically the characteristics of an episome.

Further evidence of the existence of an extrachromosomal element, acting in addition to the factor located in the 2nd chromosome, has been gathered using the following techniques.

Larvae of a tumorless highly imbred stock have been injected with a cell-free extract of tumorous adult flies, according to the technique used by L'Héritier. No effect was found in the treated individuals, nor in the offspring, until the 2nd chromosome had been made isogenic. The manifestation was identical to the phenotype of the stock used for the cell-free extract. The penetrance of the character was very low, being of about 0.2%, but reached the same frequency independently from the treated sex. This fact confirms that the transmissible unit is carried by both gametes, thus, unlike all cases of extrachromosomal inheritance, not bound to the ooplasm. In other words, this case of extrachromosomal transmission is not matroclinous. Obviously, controls have been made injecting physiological solution into tumorless larvae, but in their offspring, made isogenic for the 2nd chromosome, not one single tumorous phenotype of the donor type was found (Barigozzi, in press; Barigozzi a. Kravina 1963).

The most recent development of the investigation involved the stock Freckled

(Frd, obtained after X treatment), which has the following characterisitics: phenotype: tumors scattered in the whole body including the head (never found in any other tumorous stock); penetrance 100%; genotype: a dominant factor is localized at the right extremity of the 2nd chromosome (about 5 unit right of plexus), lethal in double dosis. Thus, the stock is kept in obligatory heterozygosity balanced over Cy.

In spite of its typical mendelian behaviour, Frd shows the same property of the other tumorous stocks. Treating the same larvae of the tumorless stock used previously with cell-free extract of Freckled flies, a clear tumorous manifestation in the head appeared already in the first generation after treatment. As compared with the original Frd, the expressivity of the induced character is reduced, and its frequency of about 2-3% (higher than with tu $A_2$ treatment). This reduced from of Frd was transmitted again through both sexes.

These data confirm the previous ones, and reinforce the evidence that tumorous stocks are characterized by a stronger chromosomal mechanism of transmission and a weaker extrachromosomal one, which is strictly bound to that living substance, which is common to both gametes.

The genetical analysis of stock Frd/Cy brought to the observation of an unexpected fact.

The cross: ♂Frd/Cy x ♀ +/+ gave rise to a proportion of 27.7% of apparent recombinants +/+ and Cy Frd/+ (further analysis of the individuals carrying as well Cy as Frd proved that the genotype was actually as indicated). If one bears in mind that crossing over is practically absent in Drosophila males, and that Cy eliminates all single crossovers, it becomes difficult to interpret the "recombinants" in terms of crossovers.

The same experiment has been repeated, and again Cy Frd/+ individuals have been found, but at a much lower rate : from ♀ Frd/Cy x ♂ +/+ the following segregation has been obtained: Frd/+ 1009; Cy/+ 929; Cy Frd/+ 5; +/+ 3. In this case, more favourable to crossing over owing to the feminine sex of the heterozygotes, only 0.5% of "recombinants" has been found. This again contradicts the interpretation in terms of crossing over. Until now, the phenomenon of transfer from an homologous to the other has been found in the following conditions:

Frd → Cy (in two different experiments, see above)
Frd → Gla
Cy Frd → +

In all cases, after the transfer, for at least one generation, the Frd factor segregates quite normally, as in case of an ordinary gene.

The phenomenon of transfer is, thus, discontinuous in a sequence of generations where the rule is mendelian segregation. It is worth noting that transferred Frd keeps its original expressivity and lethality in double dose.

Summarizing, Frd shows at the same time, 1) a factor localized at the right end of the 2nd chromosome, 2) an instability of the same factor, inasmuch it tends to jump from one chromosome to the homologous, with changing frequencies and apparently not through ordinary crossing over, 3) a self-perpetuating extrachromosomal component, transmissible through both gametes, capable of inducing a weaker manifestation.

The complex of these properties of Freckled shows a trait in common with the other tumorous stocks tu $A_2$, and tu $B_3$, i.e. a double mechanism of transmission. Thus Freckled also justifies the hypothesis according to which the two modes of transmission are interconnected by an episome-like structure endowed with a chromosomal location with stronger effect and with a cytoplasmic location with weaker effect. The crucial proof of this hypothesis should consist in the demonstration that the induced character (from injected larvae) is localizable on the chromosome. This evidence is still lacking.

Nonetheless, Frd, of its own, shows a transfer phenomenon which recalls the behaviour of the controlling elements in maize. The transfer, especially for its high frequency (in one experiment it reached 100%), shows also a similarity with paramutation, equally found in maize.

The similarities between the controlling elements of maize and Frd reinforces provisionally the hypothesis that an episome is at work in the transmission of melanotic tumors of Drosophila.

Pentzos-Daponte, Athena. Structural polymorphism in a natural population of Drosophila subobscura from Thessaloniki (Greece).

A population of Drosophila subobscura from Thessaloniki was cytologically analysed. About one hundred wild males were mated to standard females in pairs and eight larvaes from each pair examined. The most common structural types of the wild males were:

| | |
|---|---|
| A Chromosome | A st, A 1, A 2; |
| I Chromosome | I 1, I st; |
| E Chromosome | E 1+2+9, E 8, E st; |
| U Chromosome | U 1+2, U 1+2+6, U st; |
| O Chromosome | O 3+4, O st, O 3+4+1, O 3+4+22; |

The degree of structural heterozygousity and the inversion frequency are the highest ever found for natural populations of Drosophila subobscura. The index of free recombination is also very small. The values of Kunze-Mühl and Sperlich (1962) were used for the calculations. For the I - Chromosome only two structural types (I 1 and I st) were found. The frequency of I 1/I st - heterozygous individuals in nature is about 83%, much higher than the 50% which one could expect as a maximum by the rule of Hardy-Weinberg.

(This work is being continued at the Institut für Allgemeine Biologie, University of Vienna, Austria.)

Pollock, Robert. Further characterization of the Dinty translocation.

The original description of Dinty, recovered from an X-ray experiment, was reported by Braver (DIS 29:70). The following is a more detailed characterization. Dinty is associated with a double translocation, a T(1;3) and a T(2;3), with breakpoints respectively at 3C in the X and at 63A in 3L, and at 39D in 2L, and 73A in 3L. In the original report only the T(1;3) was noted. A check by D. Lindsley (1961) revealed the T(2;3) which probably was present in the original stock of Dinty.

Dinty is viable and fertile in the male and heterozygous female, and is homozygous lethal. It is associated with the following phenotypic characters: (a) the central portion of wing vein L II is interrupted for 20-30% of its total length in the females and for 35-41% in the males; (b) the posterior supra alar bristles are present in 1-5% of the females and 0.5-3% of the males; (c) the anterior post alar bristles are absent in 6-11% of the females and 2-6% of the males; (d) the wings are divergent in a small percentage of the flies. The values for characters (a), (b), and (c) of the females are significantly different from those of the males. No attempt was made to measure character (d).

Van Valen, L. Possible dual genetic control of chromosomal bands in D. virilis.

Dr. Novitski has informed me of a paper by S. Fujii (Cytologia 10(1940):294-301) that deals with a remarkable case of abnormal salivary chromosome banding. Most or all the bands in a rare pattern of the dot chromosome of D. virilis are distinguishable from their presumed homologs in the common pattern. The heterokaryotype has each chromosome of the pair distinct. Progeny from a rare crossover individual between marker genes have the proximal half of the chromosome with the common pattern and the distal half with the rare pattern; a functional difference between the two patterns is suggested by poor viability and fertility of only these crossover progeny.

The least unlikely hypothesis to explain this situation (well documented by photographs) seems to be the following. The rare pattern was formed originally by a single mutation later lost or inactivated in some way (perhaps from being on another chromosome), or just possibly by an environmental shock. Each band replicated itself thereafter. In other words, at least two genes (the original mutant and one on or constituting the band itself) are probably involved in the expression of most or all the bands of the dot chromosome, although the commoner allele of one of them permits the occurrence of both patterns.

Further investigation by a cytogeneticist is clearly needed for an understanding of this case.

Frye, Sara H. Methods used in an investigation of the X-ray dose-frequency relation of proved and presumptive minute structural changes in the yellow region of Drosophila melanogaster.

Males having the dominant marker Bar eye located in an inverted X-chromosome (scute-8) were reared in 36 half-pint culture bottles of the same age and containing standard corn meal molasses medium. Twenty-four hours prior to irradiation in the late afternoon all culture bottles of the male stock were carefully re-cleared (all stock parents having been removed between the fifth and seventh day from the time the stock was made). At approximately 23 hours after the second clearing the males were collected from all the male-stock bottles and mixed together. At the time of irradiation the scute-8 Bar males (designated $sc^8$ B) were less than 24 hours old and these were irradiated at four different X-ray doses - 500r, 1000r, 3000r, or 4000r. After irradiation the treated males were then mated immediately to virgin females containing the marker yellow. These yellow females (also containing the markers, white eye and forked bristles) were reared in 72 half-pint culture bottles, and were collected as virgins and aged so that at the time of either the first or second post-treatment matings with either irradiated or control $sc^8$ B males their age ranged from 48 to 72 hours. The number of female and male parents introduced in a ratio of 1:1 per bottle were respectively: 4000r - 20 prs., 3000r - 14 prs., 1000r - 8 prs., 500r - 6 prs., and controls - 2, 3, 4, 5, or 6 prs. (but usually 4 prs.).

Experimental Series I (simultaneous controls, 1000r or 4000r) is referred to hereafter as ES-I (see Figure 1 for brood pattern and genetic scheme). ES-I, consisting of 26 irradiation subseries and simultaneous controls, was conducted at Indiana University from October, 1955, through June, 1957. The relatively small numbers among the different ES-I subseries allowed considerable variation in the mutation frequency (see Table 1). A heterogeneity test conducted on these data showed that the variation was not greater than that to be expected on random sampling. Independent controls were carried out by the author during the summer of 1957 - these data are presented here:

| Subseries | $F_1$ B ♀♀'s | y ♀♀'s Abs. No. | % | Subseries | $F_1$ B ♀♀'s | y ♀♀'s Abs. No. | % |
|---|---|---|---|---|---|---|---|
| Ind. I a | 7,921 | - | - | Ind. I h | 5,070 | - | - |
| Ind. I b | 6,233 | - | - | Ind. I i | 7,596 | - | - |
| Ind. I c | 8,194 | - | - | Ind. I j | 9,704 | - | - |
| Ind. I d | 4,685 | - | - | Ind. I k | 7,319 | - | - |
| Ind. I e | 7,058 | - | - | Ind. I l | 10,583 | - | - |
| Ind. I f | 8,608 | - | - | Ind. I m | 9,350 | - | - |
| Ind. I g | 7,966 | - | - | | | | |
| | | | | I a - I m | 100,287 | | |

Again in the summer of 1958, the author carried out independent controls because of doubts that were raised as to the validity of her previously reported control frequency of yellow mutations in the scute-8 Bar (Bloomington $) stock. Consequently, another group (working under the direction of Dr. I. I. Oster in the Bloomington laboratory) carried out control tests with the same stock and under the same experimental conditions as those that the author conducted hers. There was no discrepancy in the results of these two simultaneous and independent projects (see Bart, C., A retest of the frequency of spontaneous loss of the yellow$^+$ region of the scute-8 chromosome, DIS 32). The author's 1958 control data are given below:

| Subseries | $F_1$ B ♀♀'s | y ♀♀'s Abs. No. | % | Subseries | $F_1$ B ♀♀'s | y ♀♀'s Abs. No. | % |
|---|---|---|---|---|---|---|---|
| Ind. I n | 9,440 | - | - | Ind. I q | 14,792 | - | - |
| Ind. I o | 12,350 | - | - | Ind. I r | 13,749 | - | - |
| Ind. I p | 10,699 | 1 | - | | | | |
| | | | | In - I r | 61,030 | 1 | |

ES-I progeny originated from y w In49 f eggs fertilized by $sc^8$ B sperm of one age from time of treatment, i.e., 2-4 days represented by Male-brood II and transfers.

$P_1$ --(-----B-----)-- $sc^8$ ♂ X $\frac{y\ w\ In49\ f}{y\ w\ In49\ f}$ ♀

(0-24 hrs. old) (48-72 hrs. old)

age of sperm from time of treatment

Male-Brood I ($PTM_1$) X Group I ♀♀'s (progeny scored and transferred in ES-II only)

48 hours

Male-Brood II ($PTM_2$) X Group II ♀♀'s (progeny scored in ES-I and ES-II) — TRANSFER Ia

48 hours — TRANSFER IIa (♀♀'s only) — 48 hours TRANSFER Ib

48 hours — TRANSFER IIb — 48 hours TRANSFER Ic

48 hours — TRANSFER IIc — 48 hours ♀♀'s removed

48 hours ♀♀'s removed

$F_1$ REGULAR GENOTYPES AND PHENOTYPES:

1.) --(-----B-----)-- $sc^8$ ↙
$\frac{y\ w\ In49\ f}{\quad}$ (B ♀)

2.) $\frac{y\ w\ In49\ f}{- - - - -}$ ↙ (y w f ♂)

EXCEPTIONAL ♀♀'s SCORED: yellow, white, white-mottled, forked, non-Bar

Figure 1. Brood pattern and genetic scheme for Es-I (0 r, 1000 r, 4000 r) and ES-II (0 r, 500 r, 3000 r). The $sc^8$ B males of ES-I and ES-II were mated immediately after irradiation and remained with the y w In49 f virgin females for 48 hours after exposure. These matings were the first post-treatment matings ($PTM_1$) and were designated as Male-Brood I. The second post-treatment matings ($PTM_2$) in all cases were between ES-I and ES-II males that had engaged in the $PTM_1$ matings 48 hours previously and fresh virgin females. The second post-treatment matings were designated as Male-Brood II. After 48 hours, the inseminated females (the males having been discarded) of Male-Brood I of ES-II and of brood II of ES-I and ES-II were transferred three more times at 48-hour intervals. The irradiated X and Y chromosomes are here indicated by arrows pointing to them.

| | CONTROLS | | | 1000 r | | | 4000 r | | |
|---|---|---|---|---|---|---|---|---|---|
| | | ♂ ♀♀'s | | | ♂ ♀♀'s | | | ♂ ♀♀'s | |
| Subseries | $F_1$ B ♀♀'s | Abs. No. | % | $F_1$ B ♀♀'s | Abs. No. | % | $F_1$ B ♀♀'s | Abs. No. | % |
| ES I a | 4,737 | - | - | 6,915 | 6 | .08 | 1,702 | 4 | .23 |
| ES I b | 7,318 | - | - | 10,883 | 10 | .09 | 2,020 | 2 | .10 |
| ES I c | 7,908 | - | - | 9,465 | 2 | .02 | 2,725 | 3 | .11 |
| ES I d | 7,094 | - | - | 6,399 | 8 | .12 | 2,026 | 4 | .19 |
| ES I e | 9,273 | - | - | - | - | - | 2,137 | 6 | .28 |
| ES I f | 9,829 | - | - | - | - | - | 2,073 | 9 | .43 |
| ES I g | 5,608 | - | - | 3,931 | 2 | .05 | 725 | 2 | .27 |
| ES I h | 6,042 | - | - | 7,084 | 5 | .07 | 1,339 | 6 | .44 |
| ES I i | 5,287 | 1 | | 7,234 | 11 | .15 | 1,157 | 12 | 1.03 |
| ES I j | 6,444 | - | - | 7,452 | 10 | .13 | 926 | 6 | .64 |
| ES I k | 6,403 | - | - | 8,759 | 4 | .04 | 2,239 | 7 | .31 |
| ES I l | 5,343 | 2 | | 6,225 | 5 | .08 | 1,280 | 2 | .15 |
| ES I m | 5,125 | - | - | 7,607 | 7 | .09 | 1,519 | 2 | .13 |
| ES I n | 6,123 | - | - | 6,664 | 5 | .07 | 1,376 | 2 | .14 |
| ES I o | 5,318 | - | - | 7,028 | 1 | .01 | 1,459 | 2 | .13 |
| ES I p | 6,445 | 1 | | 7,927 | 4 | .05 | 912 | 4 | .43 |
| ES I q | 4,578 | 1 | | 8,302 | 7 | .08 | 1,447 | 7 | .48 |
| ES I r | 5,938 | 1 | | 7,040 | 5 | .07 | 1,715 | 5 | .29 |
| ES I s | 5,412 | - | - | 7,252 | 6 | .08 | 1,557 | 8 | .51 |
| ES I t | 7,521 | - | - | 10,023 | 7 | .06 | 2,472 | 7 | .32 |
| ES I u | 6,569 | - | - | 7,617 | 5 | .06 | 1,287 | 4 | .31 |
| ES I v | 6,339 | 1 | | 7,693 | 12 | .15 | 1,277 | 4 | .31 |
| ES I w | 6,359 | - | - | 8,055 | 10 | .12 | 1,302 | 4 | .30 |
| ES I x | 5,784 | - | - | 6,939 | 6 | .08 | 1,784 | 3 | .16 |
| ES I y | 5,396 | - | - | 6,548 | 5 | .07 | 1,261 | 5 | .39 |
| ES I z | 5,214 | - | - | 7,900 | 6 | .07 | 1,593 | 6 | .37 |
| | 163,407 | 7 | | 180,942 | 149 | | 41,310 | 126 | |

TABLE I

Experimental Series II (simultaneous controls and treated, 500r or 3000r) is referred to hereafter as ES-II (see Figure 1 for brood pattern and genetic scheme). ES-II, consisting of seven irradiation subseries and five simultaneous control subseries, was conducted at St. Louis University from February, 1959, through May, 1959. The variation in the mutation frequency among the different ES-II subseries which consisted of larger numbers of $F_1$'s than the ES-I subseries, was relatively constant (see Frye, DIS 37). A heterogeneity test was not conducted on these data. ES-II progeny originated from y w In49 f eggs fertilized by $sc^8$ B sperm of two different ages from time of treatment. The first age, 0-2 days, was represented by Male-brood I and all its transfers, while the second age, 2-4 days, was represented by Male-brood II and all its transfers. The same scute-8 stock was used throughout ES-I and ES-II.

At least two counts, sometimes as many as five, were made per bottle between the 10th and 20th day to recover late-hatching mutants among the progeny of ES-I and ES-II. Many of the exceptional yellow females were recovered as virgins. Control counts were made within 24 hours (either before or after) of irradiated counts. Crowding in all series was approximately equal. The over-all mean yields ($F_1$ per bottle) were respectively: 4000r - 198 $F_1$, 3000r - 229 $F_1$, 1000r - 234 $F_1$, and controls - 164 $F_1$. Cultures were never placed in the warm or cold rooms to increase or decrease the rate at which offspring emerged.

The yellow mutants were recovered in the female, since many deletions, many cases of position effect, and perhaps some point mutations would be lethal in the male (as in fact the results did show). The total number of paternal scute-8 chromosomes (spontaneous and irradiated) recovered in the female was 996,294. All the mutants, even the spontaneous ones, arose singly. The absence of mutant clusters would suggest that the induced mutants at the time of irradiation must have arisen in mature spermatozoa or spermatids.

The yellow mutants, if proved to be fertile and transmissible after progeny testing, were then analyzed as soon as possible after stocks of them had been obtained, because of the differential viability of yellows of various structures and the possibility of later accumulation of spontaneous and detrimental mutation. The time interval between recovery and completion of analysis for ES-II yellows (which were analyzed from June, 1959 to August, 1959) was considerably shorter than that for ES-I yellows (which were analyzed from January, 1957 to June, 1958). The minimum number of flies counted for the analysis of ES-I yellows was 227,040 and of ES-II yellows was 77,127.

The scute-8 stock that was used in this study is available to anyone upon request at any of the following Drosophila laboratories (UCLA, Philadelphia, and Bloomington). The author would appreciate a cytological analysis of this stock by anyone who would be interested in this project. (Acknowledgement is made to AEC grants (11-1)-133) and (11-1)-195). This article may be quoted since it is submitted to DIS 38 as a supplement to an article in press - Frye, S. H. A tentative X-ray dose-frequency relation of minute chromosome changes of the yellow region in Drosophila melanogaster.

Iyengar, Shanta V. Nondisjunction in a Drosophila female with the $Y^c:bw^+$ chromosome. A Drosophila female whose father had the genotype $Y^c:bw^+/X^+;bw/bw$ and the mother homozygous for bw was noticed to have wild type red eyes. She was mated to one of her brothers who had the same genotype as their father to investigate if (1) one of her bw genes had mutated back to the $bw^+$ state or (2) whether she carried the $Y^c:bw^+$ chromosome.

Analysis of the offspring of this female by the above testcross showed all her male offspring were red eyed, but among the female offspring 129 (90.85%) had brown eyes and 13 (9.15%) had red eyes. The red eyed female offspring of the above female represent the eggs that carried the $Y^c:bw^+$ chromosome of the mother fertilized by the X-carrying sperm of the father or the fewer number but nevertheless possible nondisjunctional type of egg carrying both the X's of the mother fertilized by the $Y^c:bw^+$ carrying sperm of the father. The morphology of the $Y^c:bw^+$ chromosome is such that it results in a disadvantageous male ratio; in stocks bearing it as few as only 1/5th of the total flies are male. It appears that in exceptional cases of its presence in a female its behaviour is consonant with its atypical distribution at "normal" meiosis in males carrying it.

If this female had one of her bw genes reverted to the wild type, half of her female offspring would have been red eyed. The parent red eyed female was red eyed by virtue of her possessing the $Y^c:bw^+$ chromosome which obviously she inherited from her father and she in her turn passed it on to her offspring, this being phenotypically detectable only among the females as the males carry in this case the $Y^c:bw^+$ chromosome.

Epler, J. L. Ethyl methane sulfonate - induced gonadal mosaicism in D. melanogaster.

Oregon-R adult males (2-4 days old) were injected with $2 \times 10^{-2}$ M ethyl methane sulfonate (EMS) and held for 24 hours. Each male was then mated to 3 virgin females in individual culture vials for three days. Males were discarded at the end of three days and the females were transferred to a fresh culture vial for an additional three day egg laying period. Induced whole body, i.e. whole gonad, lethals and mosaic lethals were detected by the mating procedure described by Brockman, et al. (DIS 34:73, 1960). Of 267 F-1 females tested, 41 (15.36%) were found to be heterozygous for a sex-linked recessive lethal. An additional 29 (10.86%) F-1 females were found to contain gonads that were mosaic for a recessive lethal when all of the daughters from each female were tested for lethal heterozygosity. Thus, gonadal mosaic lethals comprise 41% (29/70) of all lethals induced by this concentration of EMS. In the control series 0.28% (4/1430) F-2 lethals and 0.66% (1/151) mosaic lethals were found.

The occurrence of a high frequency of gonadal mosaic lethals induced with a chemical mutagen stresses the importance of considering this component when calculating the ratio of induced lethal to visible mutations. Gonadal mosaic lethals may also serve as an effective tool for the analysis of mutagen mechanisms and chromosome morphology in Drosophila.

Braver, G. Interaction between Bristled and forked.

During the course of crosses to make a number of X chromosomes co-isogenic, an interesting interaction was observed between the mutants of Bristle and forked. Females that were $f^5/+$; Bl/+ possessed macrochaetes that differed from those of a parallel line of +/+; Bl/+ females. The latter flies showed the normal Bristle phenotype, with macrochaetes somewhat shorter and apparently no thicker than wild type, with the occasional beaded or ragged appearance along the length of the bristle. The $f^5/+$; Bl/+ females had macrochaetes that were generally shorter and thicker than either wild type ($f^5/+$ or +/+) or Bristle, and in addition, were frequently flattened and bent. In a second test, using f in place of $f^5$, the f/+; Bl/+ females showed the same bristle phenotype as $f^5/+$; Bl/+.

Singed$^3$ was also tested for a similar interaction with Bl, with negative results. These observations are consistent with the interactions reported by Lees and Waddington in 1942 (Proc. Roy. Soc. London, B, 131:87-110), in which $sn^3/sn^3$; Bl/+ bristles are not markedly different from those of $sn^3/sn^3$ alone, while f/f; Bl/+ bristles show strong exaggeration, with bristles quite short and inflated.

The f and $f^5$ alleles are apparently not completely recessive to their wild type allele, with heterozygosity detectable in a Bl background.

(Supported by a grant from the National Science Foundation.)

Johansen, Ivar. A D. melanogaster wild type cross with a high yield of hatching.

In a search for D. melanogaster eggs with a high hatchability several stocks have been tested. The hatchability was found to vary considerably from one inbred wild type stock to another. For example, one-third of the eggs from Oregon-R did not hatch, while the hatchability was found to be 94.7 per cent in Ullern wild (a D. melanogaster wild type collected locally in 1959).

Eggs from Ullern wild females mated with Canton-S males were found to have a still higher hatchability; indeed much higher than the reciprocal cross (Table 1).

Table 1. Per cent hatching.

| | Canton-S wild ♂♂ | Ullern wild ♂♂ |
|---|---|---|
| Canton-S wild ♀♀ | 91.3 | 74.5 |
| Ullern wild ♀♀ | 97.4 | 94.7 |

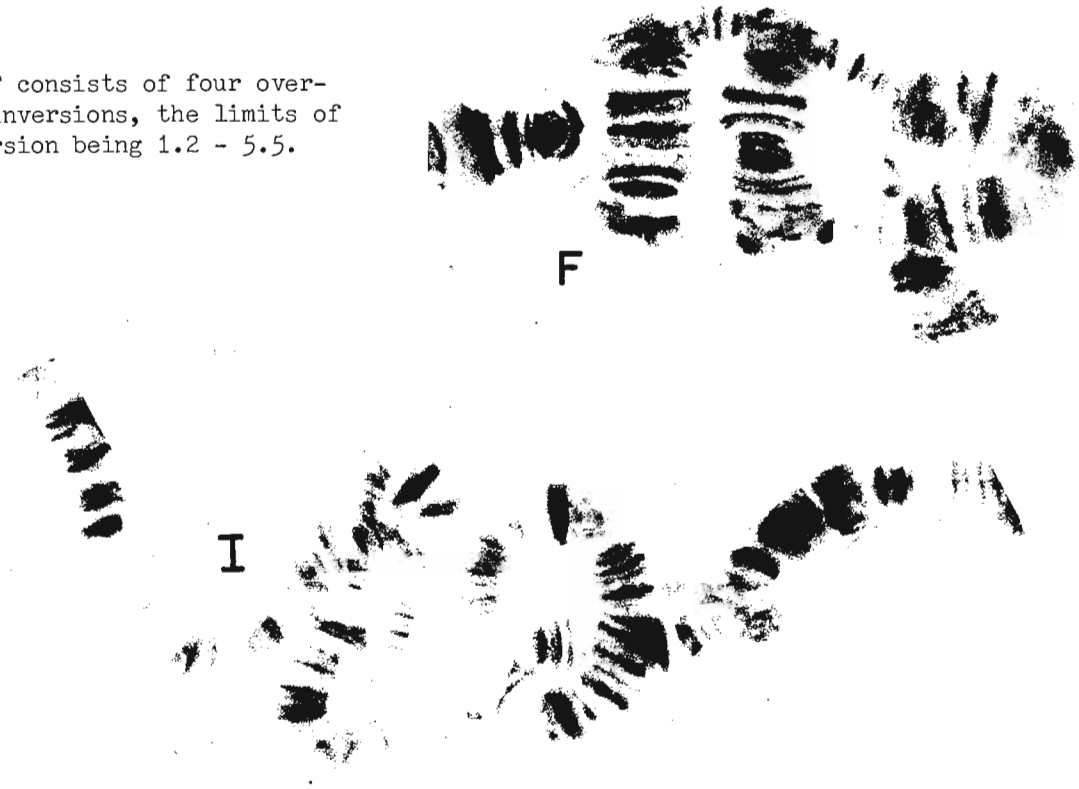
During a period of 15 months the hatchability of eggs from the cross of Ullern wild females by Canton-S males was frequently tested. It was found that the hatchability remained constant during this period. The percentage of unhatched eggs, $2.6 \pm 0.7$, is an average of some 50,000 eggs tested.

(Supported by the Norwegian Cancer Society and the Norwegian Defence Research Establishment.)

Mather, Wharton B. Further inversions in D. rubida.

Since the last report on the inversions of D. rubida (Mather, Wharton B., DIS 37:104, 1963) the analysis of further samples from New Guinea has revealed the presence of two new complex inversions - F on chromosome II R and I on chromosome III (figure 1).

Complex F consists of four overlapping inversions, the limits of the inversion being 1.2 - 5.5.




Complex I also consists of four overlapping inversions, the limits of this inversion being 17.1 and 22.1.

Brink, N. G. The effect of potassium cyanide on the mutagenic activity of heliotrine.

In DIS 37:66 the sentence, "Thus the failure of the eggs to hatch is probably resulting from sperm inactivation or lack of sperm due to the sterilizing action of the alkaloid.", should read "Thus the failure of the eggs to hatch is probably due to the occurrence of dominant lethals, and not merely due to unhatched eggs resulting from sperm inactivation or lack of sperm due to the sterilizing action of the alkaloid."

Burmeister, Maritha A., H. S. Forrest, and J. M. Lagowski. Xanthine dehydrogenase activity from mixed extracts of the rosy and maroon-like mutants of D. melanogaster.

The facts that extracts of rosy mutants of D. melanogaster exhibit a "xanthine oxidase" activity, whereas extracts of maroon-like do not, and that both mutant types lack xanthine dehydrogenase activity led to the suggestion that the wild type enzyme was composed of at least two protein sub-units (Forrest, Hanly, and Lagowski, Genetics, 56:1455, 1961). Attempts to demonstrate the existence of these sub-units were in progress when Glassman (Proc. Nat. Acad. Sci., 48:1491, 1962) reported a small - but definite - reconstitution of xanthine dehydrogenase activity from mixed extracts of $ry^2$ and ma-l, in essence confirming the theory.

Our approach in general was to work with the wild type enzyme and to attempt to dissociate and recombine it using well-established biochemical techniques (e.g. urea, sodium dodecyl sulfate, high concentrations of sodium chloride or Tris buffer, Triton X-100 or tween 80 (non-ionic detergents), acetone, and pH changes). None of these methods was successful. Either the wild type activity

was not completely destroyed or, if lost, it could not be recovered. In view of these results it is not surprising that parallel experiments using the same techniques to effect a reconstitution of xanthine dehydrogenase activity from mixtures of crude extracts of $ry^2$ and ma-l were not successful. Our first clue, albeit a very tenuous one, that different sub-units present in $ry^2$ and ma-l extracts can combine to give some activity was the observation that an enzyme extract made from a mixture of equal weights of $ry^2$ and ma-l adults brought about a very small conversion of 2-amino-4-hydroxypteridine into isoxanthopterin. At this time, Glassman's procedure was reported and we were able to confirm his results. (It may be mentioned at this point that the type of charcoal used to remove endogenous pteridines, etc., before assay of the xanthine dehydrogenase activity is not critical; Norite A (Fisher, neutral), Norite A (Pfanstahl), Norite FQP, and Darco G-60 have all been used without destruction of activity.)

Indirect confirmation of the instability of the $ma\text{-}l^+$ substance reported by Glassman has also been obtained, since xanthine dehydrogenase activity could not be obtained under the conditions used to effect reconstitution when an extract prepared from 1-3 day old $ry^2$ adults that had been stored at -20° for ca. 3 months was used together with an extract from unstored ma-l flies. The ry substance was much more stable to storage under similar conditions, since xanthine dehydrogenase activity was obtained in the reverse experiment (i.e. using 1-3 day old ma-l flies stored at -20° for ca. 3 months plus unstored $ry^2$ flies).

Kroman, R. A., and A. D. Keith* Induction of melanin pigment formation in D. melanogaster.

The addition of 1,2-dihydroxybenzene (catechol) to the food medium of developing larvae of Oregon-R D. melanogaster induces the formation of a dark pigment visible in the pupal case after emergence of the adult fly. Adult pigmentation is not affected. Solubility properties, light absorption spectra, plus the observation that phenylthiourea, a known melanin inhibitor, tends to inhibit the catechol-associated pigment suggests that it is a form of melanin. (Kroman, R. A. and P. A. Parsons, Nature 186, No. 4722, pp. 411-412 (1960) and Parsons, P. A. and R. A. Kroman, Heredity 15, Parts 2 & 3, pp. 301-314 (1960)). A study of the effects of related compounds indicates that 2 ring-adjacent hydroxyl groups are necessary for induction of the pupal case pigmentation.

The capacity of breis to oxidize catechol and 3,4-dihydroxyphenylalanine (dopa), the normal melanin chromagin, has been compared photometrically at several different stages of development. With the exception of eggs, which showed negligible phenolase activity, the oxidation of dopa is always greater than that of catechol measured at the same developmental stage. (All photometric measurements were carried out on a Beckman model B spectrophotometer.) In 4 day old adults, however, catecholase activity was not detectable, but dopa oxidase activity had dropped only slightly, compared to the earlier measured stages. This substrate differentiation may be due to specificity changes of one enzyme or to changes in concentrations of more than a single phenolase. Experiments attempting to correlate observed changes in enzyme activity with in vivo effects of pigment inducing compounds are in progress.

*Supported by a grant from the California Foundation for Biochemical Research.

Keller, E. C., Jr. and Edward Glassman Genetic diversity of xanthine dehydrogenase activity levels in wild strains of D. melanogaster.

98 wild type strains of D. melanogaster were surveyed for their mean xanthine dehydrogenase activity. Statistical analysis of the data revealed that at least 80% of the variation was probably due to genetic differences. The demonstration of the presence of a recessive gene on the third chromosome (lxd) responsible for low xanthine dehydrogenase activity resolved some of the variation attributed to genetic differences. However, the relative smoothness of the distribution of the enzyme activities of the individual flies (excluding lxd flies) indicated that some of the variation is probably due to polygenic effects.

Mettler, L. E. D. mojavensis baja, a new form in the mulleri complex.

The species of the repleta group have served as outstanding examples of Drosophila that exhibit little chromosome polymorphism (Wasserman, M., 1963). Contrary to this general phenomenon, populations of mojavensis in Southern Baja California, Mexico, have recently been found to be polymorphic for at least three banding sequences in chromosome 2, and two in chromosome 3. Collections of mojavensis were made by Mrs. Jean Russel (University of Arizona) at three localities in Baja California (La Paz, Muleje, and near Cabo San Miquel). The flies emerged from pieces of rotting cactus, Machaerocereus pammosus, which were taken to the laboratory. The species was also taken in the State of Sonora, near Sonoita and Magdalena, apparently associated with "saguaro" and "organ-pipe" cactus.

For two decades, one strain (simply identified as Chocolate-Mountains), from the Mojave Desert region of California, has been the exclusive representative stock of mojavensis. This stock is monomorphic for what I will refer to arbitrarily as the Standard banding patterns (ST) of the X, 2, and 3 chromosomes (the 4, 5, and 6 chromosomes have not been predisposed to alteration during the evolution of the repleta group). The newly discovered arrangements are named: La Paz (LP) and Baja (BA), in chromosome 2; and Muleje (MU), in chromosome 3. The breakage points of LP, BA, and MU are marked on the ST arrangements of chromosome 2 and 3 shown in the photograph. The BA sequence is included within that of LP; this, assuming inversions are unique events and multiple breakage does not occur, the chromosome 2 arrangements stand in a phylogenetic relationship, ST ← or → LP ← or → BA.

The collection sites and the chromosome types found to date (based on a small number of flies) are given below:

| Collection Site | X Chromosome | Chromosome 2 | Chromosome 3 |
|---|---|---|---|
| Chocolate Mountains, Calif. | ST | ST -- -- | ST -- |
| La Paz, Baja Calif. | ST | ST LP BA | ST MU |
| Muleje, Baja Calif. | ST | ST LP -- | ST MU |
| Cabo San Miquel, Baja Calif. | ST | ST LP -- | -- MU |
| Sonoita, Sonora | ST | -- LP -- | -- MU |
| Magdalena, Sonora (isofemale) | ST | -- LP -- | ST -- |

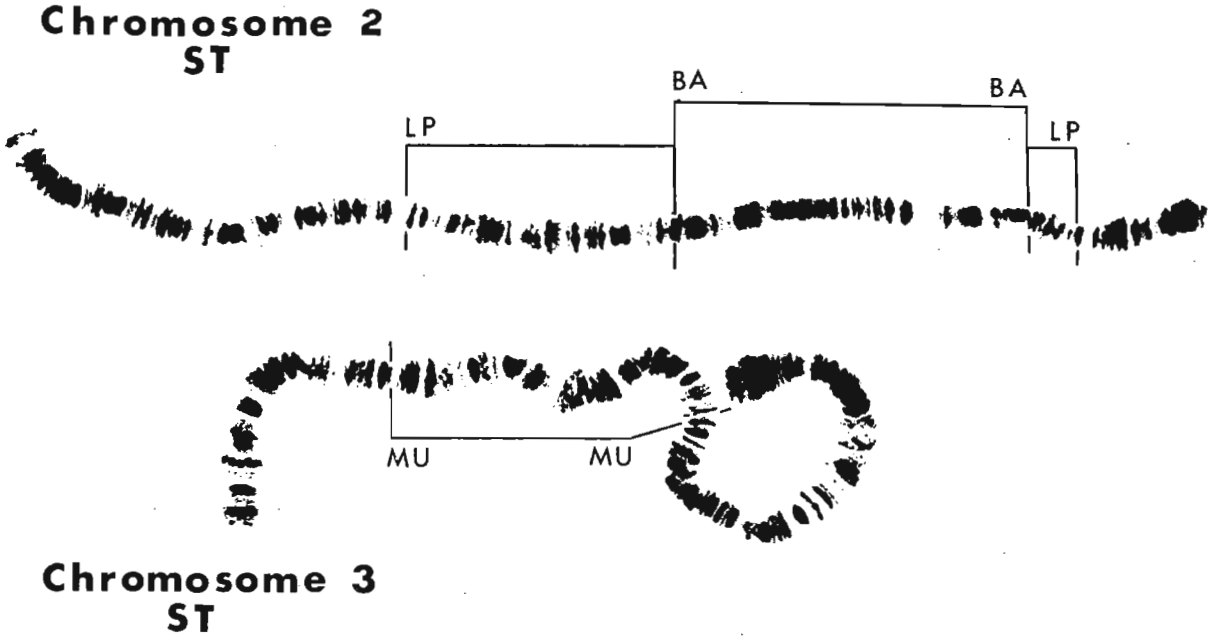
Additional chromosome types and the frequencies of each known arrangement are yet to be determined for every locality. Although the samples are too small to be conclusive at this time, it may turn out, according to the Mayr-Carson-Dobzhansky hypothesis concerning population distributions and the degree of heterozygosity, that the central populations of mojavensis exist in Baja California.

Flies recently collected in the five Mexican localities cross readily and produce fertile offspring with Chocolate-Mountains mojavensis. Their sexual behavior, fertility, and hybrid viability relationships are similar to those of Chocolate-Mountains mojavensis in tests with the semispecies, arizonensis. Thus, the new forms are considered to belong to the species, mojavensis. Additional evidence for this conclusion is the fact that all X chromosomes examined have carried the ST arrangement, which is found in no species of the mulleri complex other than mojavensis.

However, it is suggested that the recently discovered Mexican populations should be recognized as a distinct subspecies, D. mojavensis baja, differing from the Chocolate-Mountains form, D. mojavensis mojavensis. In addition to the obvious chromosome differentiation, the flies of the recent collections are morphologically distinct. They are smaller and darker than the Northern race, and are essentially sibling to arizonensis. Only by careful scrutiny of the abdominal pigment patterns can the baja race be distinguished from arizonensis under low magnification.

On a previous occasion, a fly from the Cape region of Baja California was identified as arizonensis (Wasserman, 1962). The chromosomes were checked eliminating any doubt of sibling species confusion. Thus, it is possible that the two semispecies, mojavensis and arizonensis, exist sympatrically near La Paz. Also, populations of arizonensis and the baja race of mojavensis are now known to be in close proximity, if not actually sympatric, in the border region between

the States of Arizona and Sonora. Work is planned to determine if sympatry exists and if an introgressive hybridization pattern occurs in nature as was found in experimental populations (Mettler, 1957).




(supported by N.I.H. grant GM 06981-04)

Forrest, H. S., J. M. Lagowski, and Maritha A. Burmeister. Studies on xanthine dehydrogenase in D. melanogaster. In a typical xanthine dehydrogenase (XDH) preparation, frozen, 1-3 day old, adult wild type D. melanogaster were placed in a balloon and squeezed to a pulp at 0°. The pulp was extruded from the balloon, thoroughly mixed with two volumes (w/v) of 0.1M Tris buffer, pH 7.45, and then centrifuged at 35,000 r.p.m. for 25 min. The supernatant was treated with Darco G-60 (30 mg./g. flies) and centrifuged at 35,000 r.p.m. for 10 min. The charcoal-treated supernatant was then heated at 60° for 5 min. with exclusion of oxygen (e.g. in a helium atmosphere), cooled to 0°, and centrifuged at 35,000 r.p.m. for 15 min. (4.3 fold purification of XDH with respect to the charcoal-treated preparation; assayed fluorometrically for the conversion of 2-amino-4-hydroxypteridine into isoxanthopterin); this heat-treated supernatant was used for the electrophoretic studies described below. To effect further purification the heat-treated preparation was adjusted to pH 5 with dilute acetic acid, centrifuged at 35,000 r.p.m. for 10 min., and the supernatant readjusted to pH 7.45 (7.5 fold purification with respect to the charcoal-treated preparation). Previous to our attempts to use electrophoretic techniques to study this enzyme, we had achieved a 47-fold purification of the XDH using the following scheme: Darco G-60 treatment, ammonium sulfate fractionation (40-50% saturation; 9.4 fold purification), and fractionation on a DEAE-I cellulose column (column developed with increasing concentrations of Tris buffer, pH 8; XDH eluted with 0.5M Tris; 47 fold purification). In some cases, the charcoal-treated preparation was treated with 2% protamine sulfate prior to ammonium sulfate fractionation, but the overall purification was of the same order of magnitude. Purification using columns of calcium phosphate on cellulose (developed with 0.02M sodium pyrophosphate in 0.1M Tris, pH 8) was explored; ca 50% of the XDH activity and essentially all of the protein placed on the columns could be recovered, but the purification was poor. During dialysis of the XDH-containing fractions, e.g. after ammonium sulfate fractionation, addition of an antioxidant such as β-mercaptoethanol or BAL (to the more dilute solution) decreased the loss of activity.

Heat-treated preparations of the wild-type enzyme were subjected to vertical electrophoresis on Cyanogum 41 (E-C Apparatus Corp.) (0.1M Tris-borate buffer, pH 8.9; 2 hr. at 300 v. constant voltage). The zymograms were stained either for protein (amido black) or for XDH activity (Latner and Skillen, Lancet, ii, 1286 (1961)) using hypoxanthine as substrate. Zymograms of heat-treated preparations that had been stained for XDH activity showed one broad, very strongly staining area plus several (usually four could be detected) weaker and more slowly staining areas, most of which (usually three) had an electrophoretic mobility less than that of the major component. In general the separation achieved on these gels was superior to that obtained on starch gels; the starch gels were stained for protein and/or XDH activity in the same way as the Cyanogum gels.

This general technique was extended to preparative columns of Cyanogum 41. In a typical experiment 0.5 ml. of a heat-treated enzyme preparation (4.6 mg. protein; $10^{-3}$ M with respect to cysteine) was layered on a 9 x 172 mm. column of 5% Cyanogum 41 in 0.1M Tris-borate buffer, pH 8.9; bromphenol blue was used as a marker; and 0.1M Tris-borate buffer, pH 8.9, $10^{-3}$ M with respect to cysteine, was used in the reservoirs. After subjecting the column to a constant current of one milliampere for 20.5 hr. at an external temperature of 4°, the column was extruded, the area which migrated 0.54 the distance of the dye was sectioned out, and the XDH was electrophoretically moved from this section into 0.1M Tris-borate buffer, pH 8.9, $10^{-3}$ M with respect to cysteine, contained in dialysis tubing. About a third of the XDH activity placed on the column could be recovered in this manner. Using larger columns (21 x 200 mm.) the XDH could be separated from 3 ml. of an enzyme extract containing 23 mg. protein. The protein recovered from the column could be concentrated using Carbowax 6000 and then subjected to vertical electrophoresis on Cyanogum 41 slabs. Half of the zymogram was stained for XDH and half for protein; only one band could be detected which stained for XDH, but there was a family of protein bands, no one of which could be unambiguously correlated with the XDH activity. In contrast to the very sharp, narrow protein bands, the bands staining for XDH are broad.

During the course of these experiments it has become apparent that an inhibitor (or inhibitors) is present in the charcoal-treated enzyme preparation. (1) The specific activity (assayed fluorometrically and expressed on a protein basis) of the charcoal-treated preparation is dependent upon the amount of the preparation assayed, e.g. if 0.01 ml. is assayed the calculated specific activity is ca. four times greater than that obtained assaying 0.10 ml. (2) The recovery of XDH activity in the heat-treated step is always in excess of 100%. (3) If the XDH is electrophoretically (Cyanogum 41 column) isolated from the charcoal-treated preparation, it shows a linear relationship with protein concentration in the fluorometric assay.

Whitten, M. J. Variable penetrance and and expression in Witty-eye.

Witty-eye (DIS - New Mutants) has been shown to have variable penetrance and expression. Penetrance is enhanced with maternal age and appears to respond to selection. Further-more, penetrance in most lines is substantially higher in females. Experiments are in progress to isolate any modifiers affecting penetrance. Occasionally background genotype is such that gene activity of single dose surpasses the threshold between wildtype and the mutant form. Since this particular background is lost in the progeny such dominant forms behave as recessive when mated. One dominant form, probably a consequence of a recombinatorial event, was relatively insensitive to background genotype. This stock was lost but it is hoped a similar form will be re-isolated. A study is being made of asymmetry of expression within individuals. Nothing yet is known about the breakdown in normal development of the eye to cause this mutant form.

Strangio, V. A. Further information on radio-sensitive germ cell stages in the spermatogenic cycle of D. melanogaster.

Based on Khishin's technique (1955), a direct attack is in progress on the problem of hypersensitivity in maturing germ cells to the induction of sex chromosome loss. Preliminary evidence of a peak in primary spermatocytes at the early auxocyte stage is being amplified and extended.

Mickey, G. H. Induction of crossing over in males of Drosophila by radio frequency.

Crossing over in the germ cells of D. melanogaster males has been induced by treatment of adult males with radio frequency of 20 megacycles. The treated males were heterozygous for ru h st $p^p$ ss $e^s$ (3 ple) markers and were mated to fresh virgins of the 3 ple stock every 5 days to obtain successive broods. Recominants frequently occurred in clusters and usually without complementary classes. The distribution of exchange regions was nonrandom. The induced crossovers must have occurred in spermatogonial cells rather than in spermatocytes.

Thompson, P. E., and Shuet-Fai Wei. The interchromosomal effect and crossing over in chromosome 4.

One of us reported several years ago (P. T., DIS 28) an instance of 4 chromosome-4 exchanges among 389 progeny of females carrying heterozygous inversions in all the major chromosome pairs. The crossovers were verified by progeny tests, and their occurrence was interpreted as an interchromosomal effect similar to the increases observed in other chromosomes. In later tests, he failed to obtain any chromosome-4 crossovers among 2361 progeny from a supposedly identical series of matings.

The present data represent progeny from Basc/+; SM1/+; $Ubx^{130}$/+; ci gvl $spa^{Cat}$/+ females mated to ci gvl bt males. The inversions are more complex than those used previously, and the chromosome-4 markers cover a larger interval than the ci $ey^R$ combination of earlier crosses. The progeny, with respect to chromosome-4 markers, were:

2894 +/ci gvl bt
2616 ci gvl $spa^{Cat}$/ci gvl bt
44 triplo-4 (+/ci gvl $spa^{Cat}$/ci gvl bt)

Progeny tests with ci gvl (bt) were carried out on a number of what appeared to be crossover progeny. Approximately 44 of these seemed initially to have derived from $ci^+$ $gvl^+$ $spa^{Cat}$ gametes, but on further testing proved to have ci and gvl associated with $spa^{Cat}$. They were probably triplo-4, and their occurrence seems indicative of some heterologous pairing. No actual crossovers were verified.

That the earlier instances of chromosome-4 crossing over were actual crossovers appears likely in view of the progeny testing done, but the present findings indicate strongly that the circumstances of exchange were in some way anomalous.

(Work supported by U.S. Public Health Service grant GM 08912-02.)

Strangio, V. A. Post-irradiation temperature treatment and its effect on sex-chromosome loss in D. melanogaster.

Inseminated y apr ec/y apr ec ♀♀ (previously mated to $X^{c2}$/v f ♂♂) are divided randomly into two approximately equal groups, one of which receives an X-ray dose of 800r. Immediately after irradiation, the treated and untreated groups are halved and in each case, one subgroup placed in a cold room at a temperature of 2±1°C. for twelve hours. The females are then set up individually in culture to provide five consecutive 24-hour broods. Preliminary results are summarized in the following Table. Aberration frequencies (x $10^{-3}$) are expressed in terms of total offspring.

| | Treatment | | | |
|---|---|---|---|---|
| | No X-ray + Warm | No X-ray + Cold | X-ray + Warm | X-ray + Cold |
| Total offspring | 7,466 | 5,662 | 2,719 | 3,701 |
| Sex ratio F/M | 0.93 | 0.96 | 0.88 | 0.83 |
| y apr ec XO ♂♂ | 56(7.50) | 48(8.48) | 25(9.19) | 50(13.51) |
| X $\overline{XY}$ ♀♀ | 9(1.21) | 5(0.88) | 4(1.47) | 5(1.35) |
| $X^{c2}$ v f XO ♂♂ | 0(0.00) | 2(0.35) | 3(1.10) | 4(1.08) |
| $\overline{XX}$ Y ♀♀ | 0(0.00) | 2(0.35) | 1(0.37) | 0(0.00) |
| Mosaic | 17(2.28) | 21(3.71) | 10(3.68) | 12(3.24) |
| Non-Bar ♂♂ | 0(0.00) | 1(0.18) | 2(0.74) | 3(0.81) |
| Yellow-Bar ♂♂ | 0(0.00) | 1(0.18) | 1(0.37) | 0(0.00) |

Epler, J. L., and C. W. Edington. Y-suppressed lethals in immature germ cells of D. melanogaster.

Previous work of Lindsley, et al.(1960) and Edington, et al.(1962) has dealt with the detection and characterization of Y-suppressed lethals induced by X-rays in mature sperm. Because of the nature of such lethals, it was of interest to determine the contribution of Y-suppressed lethals to total lethals induced by X-rays in cells that were at different stages of development at the time of irradiation.

In the present study y v car males (2-4 days old) were exposed to 4000r of X-rays and the frequency of total lethals (orthodox plus Y-suppressed) occurring in daily broods (1 male x 1 female) was determined by the techniques described by Lindsley, et al. (1960). In addition Y-suppressed lethals were classified in each brood as non-heterochromatic or heterochromatic (bobbed) lethals and the contribution of each type of Y-suppressed lethals to total lethals was determined.

From the results tabulated in Table 1, it can be seen that the variation in percentages of recessive lethals recovered in the daily broods essentially parallels that expected from results reported previously in similar experiments. It is clear, however, that the proportion of lethals that are Y-suppressed does change when cells that were spermatogonia at the time of treatment are sampled (broods 14-22). The proportion of Y-suppressed lethals detected in these cells is 0.31 as compared to 0.09-0.17 observed in earlier broods. This increase in the proportion of total lethals that are Y-suppressed can be attributed to the marked increase in the proportion of heterochromatic rather than non-heterochromatic or V-type position-effect lethals.

In cells irradiated as spermatogonia there is a decrease in all types of lethals except heterochromatic Y-suppressed lethals which are induced apparently at the same frequency in all stages of spermatogenesis. This would account for the increase in the proportion of heterochromatic lethals observed in spermatogonia. Since the proportion of non-heterochromatic or V-type position-effect lethals remains relatively constant in all stages of spermatogenesis, it is likely that germinal selection acts equally against these and orthodox sex-linked recessive lethals.

Table 1

| Day | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8-13 | 14-22 |
|---|---|---|---|---|---|---|---|---|---|
| No. Tested | 1171 | 1225 | 1134 | 682 | 407 | 172 | 105 | 388 | 3461 |
| Total Lethals (TL) | 203 | 114 | 110 | 67 | 57 | 22 | 18 | 27 | 26 |
| % TL | 17.33 | 9.31 | 9.70 | 9.82 | 14.00 | 12.79 | 17.14 | 6.96 | 0.75 |
| ± 1 S.E. | ±1.10 | ±0.84 | ±0.87 | ±1.14 | ±1.72 | ±2.54 | ±3.68 | ±1.30 | ±0.15 |
| Orthodox (OL) | 171 | 102 | 98 | 61 | 50 | 20 | 15 | 24 | 18 |
| % OL | 14.60 | 8.33 | 8.64 | 8.94 | 12.29 | 11.63 | 14.29 | 6.19 | 0.52 |
| ± 1 S.E. | ±1.03 | ±0.79 | ±0.83 | ±1.09 | ±1.63 | ±2.44 | ±3.42 | ±1.23 | ±0.12 |
| Y-suppressed (YsL) | 32 | 12 | 12 | 6 | 7 | 2 | 3 | 3 | 8 |
| % YsL | 2.73 | 0.98 | 1.06 | 0.87 | 1.72 | 1.16 | 2.86 | 0.77 | 0.23 |
| ± 1 S.E. | ±0.48 | ±0.28 | ±0.30 | ±0.36 | ±0.64 | ±0.31 | ±1.63 | ±0.44 | ±0.08 |
| Non-heterochromatic | 29 | 11 | 10 | 6 | 6 | 2 | 3 | 3 | 3 |
| % Non-Het. | 2.48 | 0.90 | 0.88 | 0.87 | 1.47 | 1.16 | 2.86 | 0.77 | 0.09 |
| ± 1 S.E. | ±0.45 | ±0.26 | ±0.28 | ±0.36 | ±0.60 | ±0.31 | ±1.63 | ±0.44 | ±0.05 |
| Heterochromatic | 3 | 1 | 2 | 0 | 1 | 0 | 0 | 0 | 5 |
| % Het. | 0.26 | 0.08 | 0.18 | - | 0.24 | - | - | - | 0.14 |
| ± 1 S.E. | ±0.15 | ±0.08 | ±0.12 | - | ±0.24 | - | - | - | ±0.06 |
| YsL/TL | 0.16 | 0.11 | 0.11 | 0.09 | 0.12 | 0.09 | 0.17 | 0.11 | 0.31 |
| Non-het./TL | 0.14 | 0.10 | 0.09 | 0.09 | 0.10 | 0.09 | 0.17 | 0.11 | 0.12 |
| Het./TL | 0.02 | 0.01 | 0.02 | - | 0.02 | - | - | - | 0.19 |

(Work performed under Contract No. AT-(40-1)-2417 from the U.S. Atomic Energy Commission.)

Mukherjee, A. S. Some cytological information about the mutant "sexcombless" in Drosophila melanogaster.

The mutant "sexcombless" (sx, I-?, from X-rayed sy male, Muller) has been known to be associated with an aberration which suppresses all crossing over between v (33.0) and sy (59.2). No recombination has been found between v and sx in females which are without other inversions. However, in the presence of complex inversions in the third chromosomes some crossover products have been obtained.

An attempt has been made to analyze the salivary gland chromosomes for the type of aberration involved. X-chromosomes of salivary glands have been examined in three genotypes carrying sx: 1) y ac $sn^3$ sx $vb^2$ sy/ y ac $sn^3$ v females, 2) y ac $sn^3$ sx $vb^2$ sy/Y males and 3) attached-X females which carry y ac $sn^3$ sx $vb^2$ sy in one arm and $y^2$ $ac^+$ $sn^{3+}$ $sx^+$ $vb^{2+}$ $sy^+$ in the other. Both 1) and 3) show constant inversion loops involving the region between 11D and 15E (figure 1). The type 2) larvae show a single X-chromosome with an inverted band pattern between 11D and 15E (figure 2). The analysis of the loops suggests that the mutant sx is associated with overlapping inversions that have four break-points as follows: Set I: 11E(1-2) - 11E(6) and 15E(1-2) - 15E(4); Set II: 11D(4) - 11D(6) and 14B(7-8) - 14B(9). Due to the complexity and small size of the loops and also due to the fact that the break points involve regions with rather minor bands, it has not been possible to identify the exact interband positions of the break points. No major band seems to be missing or duplicated. However, why such a small, though complex, inverted region suppresses recombination over such a wide range remains unsolved.

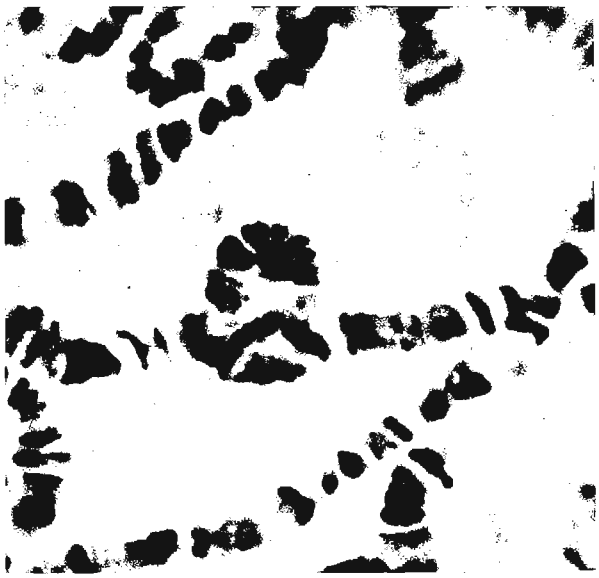


Figure 1. Photograph of the inversion loop in the sx/+ female larval salivary gland chromosomes (approximately 2500x magnified).

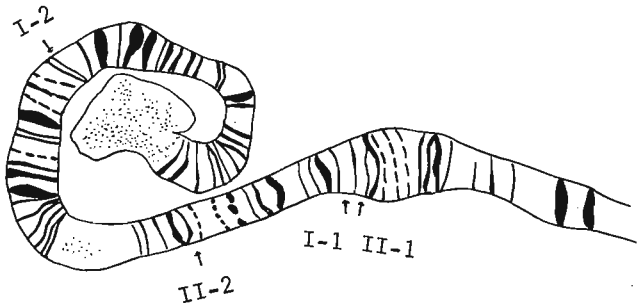



Figure 2. Camera lucida drawing of major band patterns in sx male larval salivary gland chromosome (approximately 2500x magnified). I-1, I-2 = first set of break points and II-1, II-2 = second set of break points.

Volpe, P. A technique to study components of fitness during the growth of Drosophila populations.

A number of experimental techniques have been used to study the effects of natural selection on single gene differences present in artificial populations of D. melanogaster (L'Heritier, Teissier, 1934: C.R.Soc.Biol.,(Paris), 117:1049-51; Wiedemann, 1936: Genetica 18:277-290; Nicoro, Gusiev, 1938: Biol. Zh (Mosc.) 7:197-216; Kalmus, 1945: J.Genet. 47:58-63; Reed, S. C. and E. W. Reed, 1948: Evolution 2:176-186).

But no one of these techniques offers complete information about the more important components of fitness, which would improve the study of the mechanism of natural selection acting during the growth of populations in a restricted environment.

The technique used by Buzzati-Traverso (1947a: Ist. Ital. Idrobiol. 3:67-86) is more laborious than those of the other AA. but provides a wider range of information on the dynamics of the populations under study. By means of this procedure data can be obtained on the number, sex and phenotypic ratios of the flies present in the populations at the time of each transfer to a new vial, and data on the birth rates of the two sexes and of the phenotypes in each vial.

It is possible to modify the method, thereby obtaining more information daily (fig. 1). The population is started by breeding a certain number of ♂♂ and ♀♀ in a vial in which to facilitate counting, the medium has been covered with a black paper disk, imbibed with a yeast suspension. Early the next morning one counts the adults and transfers them to a second black bottomed vial. One counts and transfers to a new vial with fresh food the eggs deposited; then the first black bottomed vial is discarded. Every day this procedure is repeated. The lines, shown in fig. 1, originating from the vials, and the downward arrows indicate that the adult progeny of each vial is added to the population.

By this method results such as those of fig. 2 can be obtained. Disregarding the particular characteristics of this population, as with Buzzati's method, one can collect the following data: 1) - variations in the population size; 2) - variations in the sex ratio of the population as a whole or of its constituent phenotypes; 3) - variations in the gene frequencies; 4) - mean lifespan of adult individuals. In addition, 5) - selection differences for larval and for imaginal stages; 6) - total productivity of the population at any given time; 7) - general oviposition; 8) - fecundity of females, measured by the number of eggs laid per day; 9) - egg hatchability; 10) - fertility, i.e. the percentage of adults emerging from a known number of eggs; 11) - rate of development (average time between the laying of the egg and the emergence of imago).

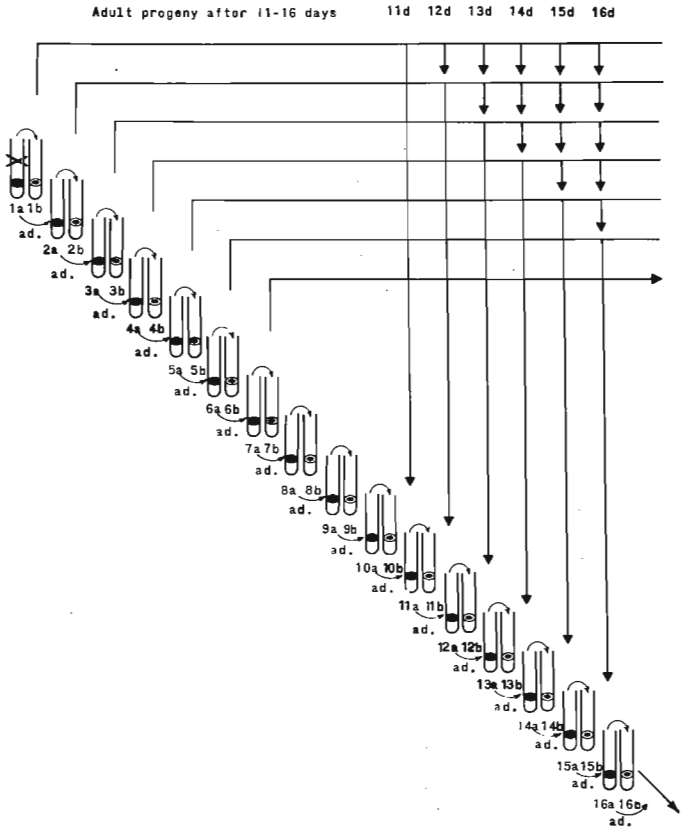



Figure 1
Schematic representation of the breeding procedure used for the experimental populations (The two series of vials a and b are indicated with successive arabic numbers)

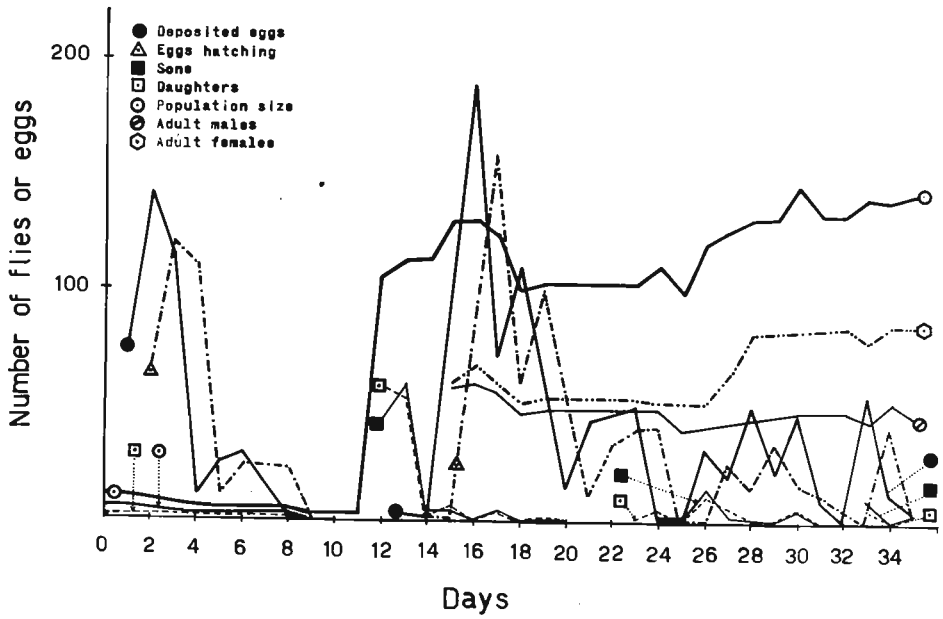



Figure 2
Selection at developmental stages and sex ratio in a Drosophila population.

Clancy, C. W. Is deep orange an allele of l(1)7?

Two well-known, X-linked, recessive mutants of D. melanogaster, hitherto described independently, are here reported to be allelic. One of the mutants is the larval lethal, l(1)7, found by Bridges in 1915; the other is the female-sterile eye-color mutant deep orange (dor), reported by Merrell in 1947. Both mutants are lethal, each in a different way. Both are eye color mutants, l(1)7 by inference based on indirect evidence. Furthermore, each has had its locus determined independently and assigned to the extreme left end of the chromosome, less than one map unit from the terminal marker (see Hildreth, DIS 37:48).

Two different stock carrying l(1)7 were used in the experiments reported here, both obtained recently from the Division of Biology, California Institute of Technology. The parental females of the progeny listed in Table 1 were from the diploid strain, l(1)7/FM-6, and those for the progeny shown in Table 3 from the duplication-bearing (aneuploid) stock, y l(1)7/y l(1)7/Dp(1;f)$X^{c2}$. The dor males used as parents in both series of matings were taken from three different strains of my personal collection of this mutant. These are identified in the tables by the stock numbers B-8, B-82, and C-174. All are derived from an original strain kindly sent to me by D. J. Merrell in December 1947. Two of these stocks, B-8 and B-82, have been isolated from each other for about fifteen years. The C-174 stock was extracted recently from B-82 and is homozygous for a new allele of the eye-color mutant, cardinal ($cd^{63}$), which arose spontaneously in the B-82 stock. These three dor strains differ from each other in certain ways that will not be taken up here. All that might be suggested at this time is that one of the known differences is undoubtedly reflected in the significant deviations from random segregation manifested by some of the progenies. These deviations do not vitiate, however, the main point of this report which is to provide evidence for the allelism of the mutants l(1)7 and dor. For this purpose one need merely note that all of the matings indicated in Tables 1 and 2 result in the production of numerous daughters which are essentially dor in phenotype. Column 3 in each table records the number (1474 in all) of such daughters recovered as adults from each culture of the two series of matings. Their eye color is very similar to the dark orange characteristic of dor females, but appears to be slightly paler in color on direct comparison under a binocular (15X, daylight filter). I have not yet determined whether or not this slight difference in color is due to a difference in amount or kind of eye pigment (see Tables 1 and 2).

Table 1. Segregation frequencies of progenies recovered from single cultures of l(1)7/FM-6 females mated to dor males. Phenotypes enclosed in parentheses.

| Strain of $P_1$ ♂♂ | dor/FM-6 (+/B) | dor/l(1)7 (dor-like) | FM-6 (♂♂) | $\chi^2$ |
|---|---|---|---|---|
| B-8 | 170 | 160 | 175 | 0.69 |
| " | 152 | 118 | 131 | 4.40 |
| " | 143 | 146 | 137 | 0.30 |
| B-82 | 163 | 93 | 165 | 23.96** |
| " | 180 | 115 | 155 | 14.33** |
| " | 121 | 117 | 117 | 0.09 |
| C-174 | 126 | 90 | 98 | 6.82* |
| " | 115 | 111 | 87 | 4.40 |

Table 2. Same as above, but y l(1)7/y l(1)7/Dp(1;f)$X^{c2}$ females by dor males.

| Strain of $P_1$ ♂♂ | y l(1)7/dor/ Dp(1;f)$X^{c2}$ (+)$^{\Delta}$ | y l(1)7/dor (dor-like) | y l(1)7/Y/ Dp(1;f)$X^{c2}$ (+)$^{\Delta}$ | $\chi^2$ |
|---|---|---|---|---|
| B-8 | 101 | 98 | 84 | 1.75 |
| B-82 | 107 | 98 | 76 | 5.43 |
| C-174*** | 152 | 205 | 123 | 21.60** |
| " | 109 | 123 | 64 | 19.26** |

* Deviation significant at 5% but not at 1% level
** Deviation highly significant
***Two successive 10 day broods from the same parents
$^{\Delta}$ Variegation of eye pigments often present

In further comparisons, the dor-like females were mated to dor males, and also to males carrying the wild type allele (FM-6 ♂♂). As is the case with dor females, they are completely sterile in the first combination and produce only daughters when mated to wild type. The daughters from the second type of mating (all carrying the FM-6 chromosome as a marker) were tested individually against wild type males (+ Oregon-R) in order to confirm the l(1)7/dor genotype assigned to their maternal parent.

Two hypotheses are suggested to explain the dor-like phenotype of the l(1)7/dor females. The first, and apparently simpler of the two, assumes that the l(1)7 mutant is the result of a deficiency for the locus. The dor-like phenotype of the heterozygote can then be explained as a case of pseudodominance of the dor allele. The second hypothesis assumes the existence of at least two different mutant alleles (not excluding a pseudoallelic relation) with both shared and different pleiotropic effects. Under this hypothesis, the dor allele complements the lethal phene (a malformation of the gut in the larva) of the l(1)7 allele, but the latter does not complement the lethal phene (abnormal oögenesis) of the dor allele. In addition, both alleles would be considered to have the same or closely similar action in relation to the development of the eye color pigments. (Work supported in part by U.S.P.H.S. Grant GM-09802.)

Miller, Barbara A. and Rhoda F. Grell

Nonrandom assortment of chromosome 3 and a Y

A number of recent investigations utilizing situations which permit recovery of the reciprocal products of meiosis have demonstrated that the X, the Y, chromosome two and chromosome four will segregate from a nonhomologue in a highly nonrandom fashion. In order to determine whether chromosome three can also be involved in this way, females of the following genotype were constructed:

1. $y^2/y^2/sc^8 \cdot Y, y^+$; T(2;3)101, al $sp^2$/TM3, ri $p^p$ sep Sb $bx^{34e}$ $e^s$ Ser
2. $y^2/y^2/sc^8 \cdot Y, y^+$; T(2;3)A, al Bl/TM3, ri $p^p$ sep Sb $bx^{34e}$ $e^s$ Ser
3. $\overline{XY}, y^+/y^2$; T(2;3)101, al $sp^2$/TM3, ri $p^p$ sep Sb $bx^{34e}$ $e^s$ Ser

In each case the females were mated to $\overline{XY}$, y B/Y males. The classes of progeny from the three crosses are shown in Table 1.

| Type ♀ | Total | $y^+$; TM3 | $y^+$; T(2;3) | TM3 | T(2;3) | % TM3, Y Segregation |
|---|---|---|---|---|---|---|
| 1 | 1162 | 76 | 406 | 550 | 130 | 82.3 |
| 2 | 1296 | 67 | 405 | 675 | 149 | 83.3 |
| 3 | 1561 | 333 | 342 | 453 | 433 | 50.9 |

The results indicate that in the presence of a free Y chromosome (Crosses 1 and 2) nonrandom assortment occurs between the multiply-inverted third chromosome, TM3, and the $sc^8$·Y. It is now possible to state that all of the chromosomes of Drosophila melanogaster will, under proper conditions, assort nonrandomly, and thus can be inferred to undergo distributive pairing with a nonhomologue.

Frydenberg, Ove The long term fate of an ebony polymorphism in artificial populations of D. melanogaster.

Various mutant alleles of the ebony locus of D. melanogaster have been reported to persist at stable equilibrium frequencies in different types of artificial populations. This seems somewhat difficult to reconcile with the fact that these mutants never maintain comparable frequencies in natural populations. Since most of the reported investigations have been confined to a relatively modest number of generations, it was considered worth while to follow an e polymorphism for an extended number of generations. An e stock of unknown origin was crossed to cn bw and six Bennett populations were started from homozygous cn bw flies obtained in the F2.

One of these populations, #125, segregated red-eyed flies when first sampled and was then discarded. The five remaining populations have now remained homozygous cn bw during three years, indicating that they have probably not been contaminated by stray flies.

The percentage of e homozygotes observed in these populations were:

| Age at sampling | | Populations | | | | |
|---|---|---|---|---|---|---|
| Days | Generations | 121 | 122 | 123 | 124 | 126 |
| 0 | 0 | 1.0 | 1.0 | 1.0 | 81.0 | 81.0 |
| 100 | 7 | 6.1 | 12.3 | 5.2 | 29.6 | 26.0 |
| 200 | 13 | 4.7 | 11.5 | 9.3 | 39.3 | 23.1 |
| 300 | 20 | - | 8.2 | 8.3 | 29.8 | 11.7 |
| 400 | 27 | 2.5 | 9.8 | 9.2 | 17.2 | 15.3 |
| 600 | 40 | 3.5 | 1.9 | 6.4 | 1.8 | 12.9 |
| 700 | 47 | 1.4 | 3.4 | - | 4.3 | 4.5 |
| 900 | 60 | 2.9 | 3.8 | 1.3 | 3.1 | 0.0 |
| 1000 | 67 | 0.3 | 1.6 | 2.3 | 2.8 | 0.7 |
| 1060 | 71 | 0.2 | 0.0 | 0.3 | 1.4 | 0.3 |

It is seen that the mutant initially showed all signs of approaching an equilibrium. In those populations that had been started at a low e frequency, the equilibrium was reached already after some seven generations and it appeared stable until approximately generation 20. But after this time the mutant frequency declined in all populations, reaching very low levels after 1000 days of observation. Apparently the e polymorphism seen in short-term experiments in artificial populations may decay with time so that these populations, as far as the e mutant is concerned, revert to the state found in natural populations.

Frydenberg, Ove The populational behaviour of a recessive lethal, $Sb^W$, of D. melanogaster

It has been reported earlier (DIS 31:120) that the recessive lethal $Sb^W$ reached apparently stable equilibrium frequencies in populations of the bottle type.

Eighteen strains of the $Sb^W$ mutant have now been followed for three to five years in 35 Bennett populations. These eighteen strains can be divided into two distinct groups.

Some strains give Bennett populations that immediately start to eliminate the mutant. This occurs at a rate even faster than expected for a fully recessive lethal. Other strains yield populations which initially maintain the mutant at frequencies around 20%. Eventually, this apparently stable equilibrium deteriorates and is succeeded by an elimination of the mutant. The break-down of the equilibrium has been observed to occur as early as in generation 10 and as late as in generation 60.

The functional overdominance exhibited by the mutant for a number of generations is explainable in terms of non-random linkage to an inversion, in most cases In(3)Mo. As long as the inversion occurs exclusively in coupling with the mutant, this latter is maintained. As soon as the inversion appears released from the mutant, this is eliminated. While the mutant disappears, the mutant-free inversion is maintained due apparently to an overdominance intrinsic to the reverted chromosome segment.

Computer studies have shown that the proposed explanation accounts satisfactorily for the available quantitative observations. It was specially striking to see that stochastical processes, inherent in populations of limited size, could actually be responsible for differences in break-down times of the order of fifty generations such as has actually been seen in the experimental populations.

Armentrout, H. C. Chromosomal polymorphism in D. robusta populations on Unaka Mountain, Tennessee.

Acetocarmine squashes were made of 812 $F_1$ female larvae of D. robusta collected at 15 stations on Unaka Mountain, in northeastern Tennessee. The complete structural karyotype was determined for each of the female larvae from which the mean index of free crossing over was calculated for the population taken at each collecting station. Chi-squares testing the goodness of fit between observed and expected values of various karyotypes were calculated. Several of the samples of D. robusta from Unaka Mountain showed a relatively high degree of free recombination, which characterized them as either marginal or intermediate populations. With some exceptions there were indications that the amount of free recombination increased at higher elevations. The effects of a changed ecology (caused by fires and logging operations) were also suggested as contributing factors for the increased amount of free recombination. Some of the inversions normally associated with northern populations were conspicuously abundant. The Chi-square tests showed that significant P values were usually associated with the X chromosome.

Bodenstein, D. and R. C. King Nonautonomy of $ap^4$ ovarian implants.

Females homozygous for apterous (2-55.4) may be viable and fertile as in the case of $ap^{56f}$, or they may be sterile and die during the second or third day of adult life (as in the case of $ap^4$ and $ap^{49j}$). $ap^4/ap^{56f}$ and $ap^{49j}/ap^{56f}$ are viable and fertile; whereas $ap^4/ap^{49j}$ are poorly viable and sterile. Vitellogenesis is greatly retarded in the sterile apterous mutants. Females of genotype $ap^4/ap^4$ or $ap^{49j}/ap^{49j}$ or $ap^4/ap^{49j}$ have no oocytes containing yolk in their ovaries during the first two days of life. The few flies which survive to the third day sometimes contain a few yolky oocytes in their ovaries, but the total volume of yolky ooplasm per ovary is no more than 1% that found in wild type flies of similar age. However, ovaries of $ap^4/ap^4$ genotype will produce abundant yolk when implanted into wild type abdomens. Thus it is the abdominal environment of $ap^4$ which is at fault, rather than a malfunctioning of the ovary. Ovarian implants from $ap^4/ap^4$ donors produce four times more yolk when left for three days in the abdomens of fes/fes females than when left for an equal time in wild type abdomens. This finding suggests that the host ovary and the implant are competing for nutrients. Since the host ovaries offer little if any competition in the case of fes, the implant is more successful. As one would expect implants produce more yolk the longer they are left in the host. Thus five day implants were found to have 3.5 times more yolky ooplasm than implants left for three days in the abdomens of + females. Ovaries from $ap^4/ap^4$ females also produce yolk when implanted into the abdomens of wild type males or tra/tra "males". However, the amount of yolk is at least 10 times greater when transformed females serve as hosts than when normal males are hosts. (Research supported by the U.S. P.H.S. grant RG-9694 and NSF grant G 21759).

Pasternak, Luise Mutagenic activity of nitrosamines

Further experiments were carried out to test the mutagenicity of Methyl-vinyl-nitrosamine, Methyl-benzyl-nitrosamine and N-methyl-N'-nitrosopiperasine. The first two substances were able to induce tumors of the esophagus in

rats; as regards N-methyl-N'-nitrosopiperasine there is at this time no final result.

It is known that nitrosamines will be broken up in vivo enzymatically. This process is followed by an alkylation of nucleic acids.

The substances which were diluted in an aqueous solution were fed to Drosophila males for 3 days. Their mutagenic activity was investigated by the M-5 test.

| Substance | Dose | Brood I | Brood II | Brood III |
|---|---|---|---|---|
| Methyl-vinyl-nitrosamine | 0.0002 % | 2.9 (455)* | 5.0 (400) | 1.7 (240) |
| Methyl-benzyl-nitrosamine | 0.006 % | 2.7 (408) | 3.0 (135) | 4.0 (276) |
| N-methyl-N'-nitrosopiperasine | 0.005 % | 2.2 (808) | 5.0 (474) | 5.6 (624) |

*Numbers in parentheses are those of tested chromosomes

The doses used were shown to be non-toxic. As to be seen from the table, there is a mutagenic activity for all three compounds. The values presented for each brood are percentages of the mutation rates. Control experiments were negative.

DeMarinis, F. Action of some synthetic amides on the Bar larvae of Drosophila. It has been shown, in general, that amides, ureids, and nucleotides all tend to increase the facet number in the Bar eye Drosophila. However, some of these compounds have been found more toxic than others and thus have delayed their further study.

The present work reports on the results of some of the toxicity tests made on some of these compounds. The compounds were mixed with standard Pearl's food mixture, eggs were deposited and permitted to develop on this mixture. The following toxic concentrations were determined for each of the following: N-methylformamide-0.50%, ethylurea-0.50%, allylurea-0.50%, acetylurea-0.50%, biuret-0.50%, methylurea-0.50%, uracil-1.00%, n-propionamide-1.00%, n-butyramide-1.00%, acetamide-1.25%, hydantoin-1.25%, malonamide-4.00%, glutaramide-4.50%. While adipamide, succinamide, oxamide, uric acid, allantoin, and biurea show no apparent toxic effect when used in higher concentrations. The double amides, $NH_2CO(CH_2)_n CONH_2$, are found to be least toxic and more effective than the single amides, $R\text{-}CONH_2$, in increasing the number of facets in the Bar eye. In general, the different concentrations of these compounds which permit the complete development of the fly also delay its developmental period: Urea-10 days, methylurea-10 days, allylurea-10 days, adipamide-10 1/2 days, malonamide-11 days, N-methylformamide-12 days, ethylurea-14 days, hydantoin-15 days, biuret-15 days, n-valeramide-16 days, n-hexanamide-22 days, uracil-24 days, control-9 days. Some of these amides seem to interfere with the very early stages of the normal physiology of development while others seem to interfere during the late pupa stage. Ethylurea and methylurea increases facet number and at the same time modifies forked bristles to normal-appearing bristles.

Jacobs, M. E. Amino acid utilization of ebony and non-ebony melanogaster. $C^{14}$ labeled amino acids were injected into the hemocoels of early pupae. e/e showed marked deficiency of deposition of beta-alanine derived $C^{14}$ in pupal sheaths as compared with +/+, while +/e was intermediate. Gamma amino butyric acid (1-C-14) and uniformly labeled aspartic acid, tyrosine, and phenylalanine showed similar results, though much less marked. It is postulated that accumulation of beta-alanine and, perhaps, gamma amino butyric acid in e/e (due to failure of deposition in the pupal sheath) may help explain the abnormal behavior of e/e (these amino acids are neural inhibitors). It is also postulated that accumulation of beta-alanine may inhibit transformation of phenylalanine to beta-alanine through aspartic acid, with consequent conversion of phenylalanine to melanin. Chromatographic analysis of acid digested pupal sheaths of flies injected with $C^{14}$ labeled aspartic acid showed $C^{14}$ at the beta-alanine as well as the aspartic acid positions, thus supporting this postulate.

Mittler, Sidney and A. Hampel Enhancement of radiation induced crossing over in male Drosophila. In the study of sulfhydryl compounds as possible protection against radiation induced genetic damage, one of the compounds AET (2-aminoethyl isothiouronium) was found to increase crossing over in the male. Approximately 1 X $10^{-7}$ l containing 3 X $10^{-4}$ mg of AET buffered to pH of 7 with NaOH was injected into 12-24 hour old males heterozygous for al b c and exposed to

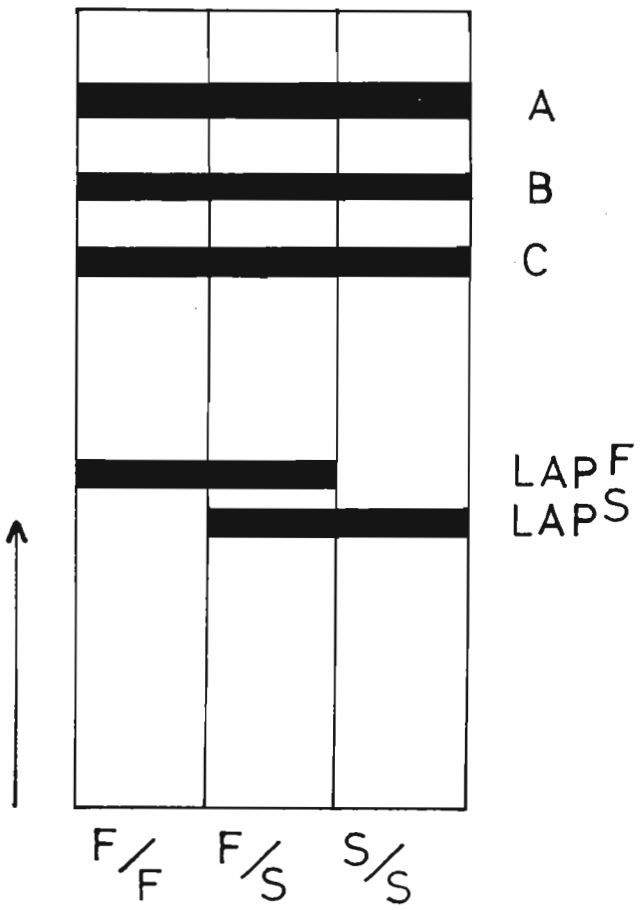
2000R of 100KV X-rays. The males were mated individually to al b c at ratio of 1 male to 3 females and transferred to a new group of virgins every 4 days. The data from 12-16 day brood represent spermatogonia that were exposed to AET and radiation. The $r_1$ and $r_2$ below are single and two mutant recombinants.

| Treatment | No. of treated ♂♂ | No. of ♂♂ which produced recombination offspring ($r_1$) | No. of ♂♂ which produced recombination offspring ($r_2$) |
|---|---|---|---|
| Saline injected and irradiated (control) | 77 | 9 | 6 |
| AET injected and irradiated | 71 | 26 | 14 |

Beckman, L. and F. M. Johnson Variations of leucine aminopeptidase in pupae of D. melanogaster.

Homogenates of Drosophila individuals of different developmental stages were investigated for leucine aminopeptidase (LAP) activity by means of the zymogram technique. Single individuals were frozen and thawed and then homogenized in a small drop of distilled water. After starch gel electrophoresis of the samples, the gels were stained in Tris-maleate buffer pH 6.0 using L-leucyl-β-naphthylamide as substrate and Garnet GBC salt as dye coupler.

The aminopeptidase zones were rather faint and variable except in the pupal stage where a quite sharp red-staining zone was observed. Pupae from a number of different stocks were then examined for variation in this LAP zone (the stocks were obtained from Dr. I. Oster, The National Science Foundation Stock Center at The Institute for Cancer Research, Philadelphia). Two different electrophoretic variants of pupal LAP were found. From the Philadelphia stock b232, 42 individual larvae were tested and all were found to have the slow component only. Forty-five individuals from stock b313 had only the fast component. Individuals from the reciprocal crosses between stocks b313 and b232 showed both the slow and the fast components. The present preliminary results suggest that the fast and slow pupal LAP variants are controlled by two autosomal alleles.

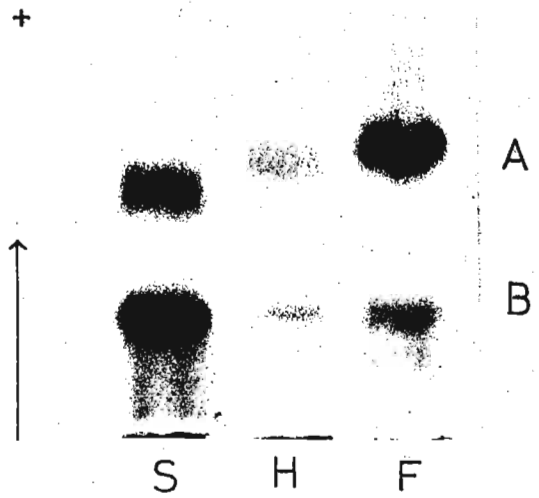



Figure

Schematic picture showing the electrophoretic mobilities of some proteins in homogenates of pupae of D. melanogaster. A, B and C are the major protein fractions revealed by means of amidoblack staining. $LAP^F$ and $LAP^S$ are the two different variants of leucine aminopeptidase. Three different LAP types F/F, F/S and S/S are shown in the picture. The arrow shows the direction of mobility.

Beckman, L. and F. M. Johnson Genetic variations of phosphatases in larvae of D. melanogaster.

Homogenates of single individuals of D. melanogaster were examined by means of starch get electrophoresis. The resulting gels were stained for alkaline phosphatase activity using α-naphthyl phosphate and Fast Blue RR salt. The electrophoretic enzyme patterns varied between different developmental stages. One particular zone, found only in the larval stage, showed variations between different laboratory stocks (the stocks were obtained from Dr. I. Oster, The National Science Foundation Stock Center at The Institute for Cancer Research, Philadelphia). Larvae from the Philadelphia stocks d32, g119 and b232 had a fast moving variant of the larval phosphatase, whereas larvae from stocks f41, b317 and b313 had a slow moving variant. Mixtures of homogenates from larvae with fast and slow variants showed the presence of both components. Larvae obtained from reciprocal crosses between stocks d32 x f41 and b232 x b317 had only one phosphatase zone of intermediate mobility. The present preliminary results indicate that the fast and slow variants of larval phosphatase are controlled by two autosomal alleles which in a heterozygous conbination interact to form a hybrid substance of intermediate mobility.




Figure

Zymograms of multiple larvae homogenates (six larvae in each sample) showing slow (S), hybrid (H) and fast (F) variants of the phosphatase zone A. The slow moving zone B is nonvariable and occurs in all developmental stages. The arrow indicates the direction of the migration.

Butler, Daniel R. and L. E. Mettler Ecological and cytological notes on D. sigmoides.

An association has been observed between D. sigmoides and Tripsacum dactyloides, a relative of Zea mays. The Tripsacum inflorescence is of the panicle type, though lateral branches are sometimes lacking. Florets are unisexual. The pistillate florets are borne singly on the lower one-fourth of the central spike and lateral branches. The staminate florets occupy the terminal three-fourths of the central spike and lateral branches and are borne in spikelets, two florets per spikelet and two spikelets per node.

Observations were made at Raleigh, N. C. during the week of July 14-19, 1963. Adult flies were seen flying near or resting on all parts of several Tripsacum plants, though they were most numerous in the vicinity of the inflorescence. Infrequently, flies were observed on adjacent plants of other species, but never at a distance of more than three feet from a T. dactyloides plant.

Close examination of two stems and associated leaves and inflorescences revealed the staminate portion of the inflorescence to be the site of egg deposition and larvae development. On one staminate spike with eighty-four spikelets, twenty-six empty egg cases were observed. On the other staminate spike, with seventy-four spikelets there were twenty-one empty egg cases. Only one egg case was found in the pistillate portion of the inflorescences.

At the time of egg deposition the anthers have not been exerted and are approximately one-third to one-half full size. Upon hatching, the larvae enter the staminate florets where they feed upon the anthers until pupation. One staminate spike bearing seventy-four spikelets possessed twenty-five larvae. All flies eclosing from a segment of this spike were identified as sigmoides.

Cytological preparations of salivary glands and brain ganglion of a few larvae revealed that 2N = 12. The karyotype consists of five pairs of rods and a pair of very small dots. One pair of rods is larger than the others. No differences could be found between male and female karyotypes. Pairing was regular in the five large arms of the salivary chromosomes (for the few individuals examined).

Novitski, E. The origin of mosaics.

In experiments involving the irradiation of the doubly marked Y chromosome, $y^{+}YB^{S}$, Novitski, Cohen and Laird (1962) have shown that mosaics occur with an appreciable frequency. Among a total of 58 changes in which Bar was lost, but the normal allele of yellow was not, 15 proved to be mosaics, either after closer examination of the individual which proved to have one eye Bar and the other not, or after progeny tests which indicated that the apparent non-mosaic in fact must have carried both a changed and an unchanged chromosome. Since such changes must originate from chromosomal breaks rather than from point events, the arguments which have been advanced to explain mosaicism at the genic level should be generalized (if possible) to include mosaicism for gross chromosomal abnormalities.

Muller, Carlson and Schalet have proposed rotational substitution of base pairs as an explanation for whole body (gene) mutations, the remainder then affecting usually only one strand and resulting in mosaicism. It does not seem likely, however, that such an explanation could be extended to include whole body and mosaic changes brought about by breakage events. We would like to suggest an alternative which will account for both genic and chromosomal types of mosaicism. Assume that one of the first two cleavage products in the egg can lead to complete development even if the other is lethal, and that the chromosomes are in a doubled state in the mature sperm. A mosaic might arise, then, when one of the two strands is affected, followed by their separation at the first cleavage division into two viable products, each making a contribution to the soma of the developing individual. A whole body mutation arises, similarly, when only one of the two strands is affected, but when one of the two first division cleavage products (the one not carrying the change) is lethal. This lethality could arise from dicentrics or inviable chromosomal combinations induced by the treatment in any of the other chromosomes.

On this basis, then, the grossly different frequencies of mosaicism found when X-rays and chemical mutagens are compared could be attributed, for the most part, to the different frequencies with which lethal changes (killing off one of the two first division products) are induced by the two types of agents.

Nirula, Satya, R. P. Sharma, M. S. Swaminathan, and A. T. Natarajan. Incidence of crossing over in male Drosophila fed on irradiated medium.

An experiment was designed with the aid of second chromosome recessive markers (b cn bw) to see whether the indirect effects of radiations through feeding irradiated medium could induce crossing over in male Drosophila.

Freshly laid eggs of the genetic constitution + + +/b cn bw were transferred onto the basic medium (comprising glucose, agar and yeast) irradiated with 150,000 rads of $Co^{60}$ $\gamma$ rays. A comparable number of controls were transferred to the unirradiated basic medium. The larvae completed development in the same culture vial without further transfer. The adult males (of the constitution + + +/b cn bw) emerging out of these larvae were mated individually to untreated virgin b cn bw/b cn bw females. The progenies were scored for crossovers between these loci. In a total of 100 crosses studied, recombinations which could be ascribed either to somatic or meiotic crossing over in the male germ line, were recovered only twice.

Crossing over occurred in one case between b and cn bw resulting in the expected parental and crossover classes viz., + + +/b cn bw, b cn bw/b cn bw and b + +/b cn bw, + cn bw/b cn bw.

In the second vial, besides the parental types, offspring resulting from crossing over between b and cn bw and cn and bw could be distinguished. These were as follows: i) b + +/b cn bw; + cn bw/b cn bw and ii) + + bw/b cn bw; b cn +/b cn bw. Out of a total of 137 flies, the former types were 14 and 10 and the latter 7 and 9 respectively; the rest belonged to the two parental classes.

Though the possibility of translocations having occurred in the material cannot be ruled out, it seems likely from the total absence of sterility or other abnormalities in the material that only crossing over is involved.

Rinehart, R. R., R. M. Valencia, and J. I. Valencia. Comparative lethal rates from X-irradiated D. melanogaster males and females mated together or to unirradiated partners.

The possibility was considered that irradiated D. melanogaster stage 14 oocytes might be less able to repair X-ray-induced damage in fertilizing sperm than was the cytoplasm of eggs receiving no X radiation. Either irradiated D. melano-

gaster Oregon-R females, matured for 5-1/2 to 6 days, or similar unirradiated females, were mated to irradiated or unirradiated males, matured for the same length of time, according to the mating scheme presented in Table 1. The introduction of the flies into the irradiation chamber, as well as all pair matings, were performed without etherization so that mating and fertilization would occur as soon as possible after the X-rays were delivered. The techniques used in pair mating a large number of flies quickly, without the use of ether, are presented in the technical notes of this issue. Even though insemination usually started within 15 minutes after the males and females were put together, fertilization, as measured by egg laying, usually did not start until 5 to 8 hours after the mating. The number of eggs laid by these females was counted and all vials that contained fewer than 25 eggs were matured at 25±1°C. and were scored for pupal development. A ratio of the number of pupae developing to the total number of eggs laid gave the percent survival for the test. One minus the percent survival gave the lethal rate, the parameter of X-ray damage used.

Table 1 demonstrates that in this system females irradiated with either 250 or 500r of X-rays are as capable of repairing the X-ray damage of sperm given X-ray exposures of 1000 or 2000r, as are unirradiated eggs. Therefore either the X irradiation of these eggs had no effect upon repair mechanisms, or, more likely, even in this system, specifically designed to bring sperm and eggs together as quickly as possible, the damage to repair systems in the oocyte already had been repaired at the time of sperm entry.

| X-Ray Exposure (r) | Mating Scheme: X-irradiated ♂ and ♀ — Pupae/Eggs | % Lethal (observed) | S.E. | % Lethal (expected)* | Mating Scheme: X-irradiated ♂ Unirradiated ♀ — Pupae/Eggs | % Lethal | S.E. |
|---|---|---|---|---|---|---|---|
| ♂ 1000 ♀ 500 | 186/606 | 69.3 | ±1.87 | 73.4 | 514/772 | 33.4 | ±1.70 |
| ♂ 2000 ♀ 500 | 105/707 | 85.1 | ±1.34 | 85.5 | 380/899 | 57.7 | ±1.65 |
| ♂ 1000 ♀ 250 | 259/624 | 58.5 | ±1.97 | 58.8 | 210/314 | 33.1 | ±2.66 |

| Cont. | Mating Scheme: Unirradiated ♂ X-irradiated ♀ — Pupae/Eggs | % Lethal | S.E. | Mating Scheme: Unirradiated ♂ and ♀ — Pupae/Eggs | % Lethal | S. E. |
|---|---|---|---|---|---|---|
| ♂ 1000 ♀ 500 | 316/791 | 60.1 | ±1.74 | 553/602 | 8.1 | ±1.11 |
| ♂ 2000 ♀ 500 | 214/625 | 65.8 | ±1.90 | 767/830 | 7.6 | ± .92 |
| ♂ 1000 ♀ 250 | 360/584 | 38.4 | ±2.02 | 449/497 | 9.7 | ±1.33 |

*Calculated from the survival rates of tests in which only females or males were irradiated

Fahmy, O. G. and Myrtle J. Fahmy

Nomenclature of 'visibles' in relation to mimicry and pleiotropism.

In the analysis of the new visible mutations induced mainly by the alkylating compounds, two difficulties arose in relation to nomenclature. The first concerned the naming of mutants of comparable phenotype but with different genetic position (mimics). The second related to highly pleiotropic mutations where several of the external features of the fly were slightly affected, but with no single feature sufficiently pronounced to characterise the mutant.

Mimics were most frequent among the mutations affecting the macrochaetae

(thin-bristle), the body size (small-body), and wing position (upheld). So far we have endeavoured to give each mutant a distinctive name, but this has become increasingly difficult and cumbersome. We accordingly propose to distinguish between mimics by adding to the symbol of the general type the genetic position of the locus (to the nearest unit) in inclined arabic numerals joined with a hyphen. This is a slight modification of the customary system now in use for mimics, with the inclusion of the genetic positions of the mutants instead of an arbitrary series of letters. The symbol would thus indicate the position of the mutation without recourse to the literature or the detailed records. The first mutant we named "thin-bristle" (DIS 32:76) will accordingly become thb-48. In the section on 'new mutants' of this or the next issue of DIS, we shall also describe two "small-body" mutants sby-61 and sby-62. These proved non-allelic in spite of their close proximity.

Mutants with multiple slight deformities will be designated according to essentially the same system used for mimics. The general phenotypic type will be termed "polyphene, symbol pph" and the different mutant loci will be differentiated according to genetic position: ex. pph-61

Polyphenes are of limited use in genetic experimentation since they are less obvious than pronounced single character mutants; this is further indicated by dropping their rank by a point for difficult classification. Nevertheless, they are of value in the indication of sites of mutagenic action, as well as in deficiency coverage tests.

Kim, K. W. Drosophilidae of Tol-San Island, Korea.

Collections were made in 1959, 1961, and 1963 at Mt. Kum-O (223m) of Tol-San Island. A total of 1036 specimens belonging to 16 species were collected by trapping and sweeping, and they were represented in Table 1.

Table 1. Drosophilid flies collected in Tolsan Island from 1959 to 1963

| Species | Total | Species | Total |
|---|---|---|---|
| Leucophenga magnipalpis | 1 | D. auraria (C-type) | 204 |
| Mycodrosophila splendida | 1 | D. rufa | 107 |
| Mycodrosophila koreana | 1 | D. brachynephros | 2 |
| Scaotimyza graminim | 2 | D. angularis | 131 |
| D. coracina | 23 | D. bizonata | 26 |
| D. suzukii | 280 | D. (D.)sp. of quinaria section | 17 |
| D. lutea | 9 | D. immigrans | 229 |
| D. melanogaster | 1 | D. virgata | 2 |
| | | Total flies | 1036 |

Among them D. rufa was collected in this Island and Huk-San Island of Korea; so far, this species seems to be distributed only in the southern islands of Korea. 2 males of D. virgata were collected for the first time, and this species is new to the Korean Drosophilid fauna. D. auraria was collected only C-type, and D. suzukii, D. auraria, D. rufa, D. angularis, and D. immigrans were dominant species in the island.

Di Pasquale, A. and L. Zambruni New data on the manifestation of the "brown spots" (bsp) character.

New investigations on "brown spots" of D. melanogaster - a character whose manifestation is caused by copulation - showed that the frequency of spotting increases with the age of the copulating females, while no relation is found between aging of the male and spotting. Other observations seem to emphasize an activity of the sperm fluid: mating with males lacking paragonial fluid owing to repeated copulations, causes only very few spots in the bsp/bsp females.

Ichida, H. and E. Ohnishi Decreased tyrosinase activity in tumor-bearing individual of $tu^g$ in D. melanogaster.

The melanotic tumor mutant, $tu^g$, is controlled by a recessive gene (or genes) located on the second chromosome. When reared on 20% yeast-sucrose-agar medium, its penetrance is not complete (70 - 90%).

Tyrosinase activity of the prepupal extract was compared between tumor-bearing individuals and false-normal individuals sampled from the same inbred line. Enzymatic activity was determined by the colorimetric method of Horowitz and Shen (1952), following the process of activation at 0°C. The time required to attain half the maximal activity

was taken as a measure for comparing the rate of activation process of the enzyme.

Typical data are shown in the table. Extracts from tumor-bearing individuals showed always lower tyrosinase activity than those from false-normal individuals. No significant difference was seen in the rate of the activation process. The decrease in enzymatic activity is apparently due to the metabolic disturbance caused by the presence of melanotic tumor.

| Sex | Phenotypic expression | Maximal tyrosinase activity (O.D./mg protein/min.) | Time to attain half maximal activity (min.) |
|---|---|---|---|
| Male | normal | 0.162 | 48.0 |
| | tumor-bearing | 0.091 | 51.3 |
| Female | normal | 0.150 | 44.0 |
| | tumor-bearing | 0.089 | 49.0 |

Moriwaki, D. and Y. Fuyama Responses to selection for the rate of development in D. melanogaster.

Several authors have performed selection experiments with regard to the rate of development in Drosophila. However, there still remains ambiguity as to whether the selection is effective for fast development. To clarify this, two selection experiments were carried out on Drosophila melanogaster.

In Exp. 1, two selection lines for fast and slow development (F- & S-line respectively) and a control line, each consisting of ten cultures, were started from the $F_2$ generation of the cross between two wild strains, Renfrew and Gabarros. While in Exp. 2, a heterozygous cage population was used to make the foundation population, from which two replicate lines in either direction of selection with six cultures in each were started. Time in hours needed from oviposition to pupation was measured as the rate of larval development and individual selection methods were applied by picking pupae with selection intensities of 20%. Only in Exp. 2, selections were practiced every second generation. Selected parent flies were permitted to oviposit during four hours and newly hatched 50 larvae were introduced into each of culture bottles. All experiments were carried out at 25° using boiled yeast medium for measurement.

As a result of selection, considerably rapid responses were observed in both F- and S-line throughout Exp. 1 and Exp. 2. Difference between F- and S-lines, diverging, attained about 17 hours after nine generations in Exp. 1 and about 19 hours after nineteen generations in Exp. 2. In Exp. 1, responses per generation of S-line were approximately two times as large as that of F-line. However, when the responses were plotted against cummulative selection differentials, there was no indication of such asymmetry of response, and so this discrepancy would be accounted for larger phenotypic variations found in S-line.

Estimates of realized heritability ($h^2$) were 0.19 and 0.20 for F- and S-line respectively in Exp. 1, and $h^2$ = 0.11 & 0.14 for two F-lines and 0.17 & 0.10 for two S-lines in Exp. 2.

From these results it seems to be concluded that selection for the rate of development is effective in both fast and slow directions with realized heritability of 0.1 - 0.2. This, also, shows that relatively small but consistent additive genetic variances are present in this character.

Okada, T. Adaptive differentiation of the respiratory horns of drosophilid larvae and puparia.

The gross structure of the larval and puparial respiratory horns (anterior and posterior) were comparatively studied with about fifty species of Drosophilidae. The anterior horns are found to exhibit general complication in accordance with the systematic positions of the possessors (general caenogenesis). Although the same is generally true also for the posterior horns, the general trend in this case tends to be obscured probably by the development of special adaptation to the specific environment (special caenogenesis). For example, the sap eaters show two opposite directions of structural differentiation: 1, posterior horns are extremely short and divergent, while the anal tubercles are well developed (Steganinae in general, Chymomyza); 2, posterior horns are extremely long and parallel, while the anal tubercles are small or degenerated (some of Drosophilinae, esp. Paradrosophila). Both features, though opposed to each other, can be interpreted as highly adaptive to the sequence to prevent the larvae or puparia to sink in the soft media. That the sap

environment is softer than the fruit or fungus environment is accounted for by the fact that the mouth hook dentition is less developed in the sap eating larvae than in the fruit or fungus eaters (cf. DIS 34:98).

The differential adaptational trends observed between the anterior and posterior horns suggest their different respiratory functions: inspiration by the posterior and expiration by the anterior horns.

Paik, Y. K. A comparative survey of genetic variabilities between second and third chromosomes from Korean populations of D. melanogaster.

An earlier study of Korean populations of D. melanogaster showed an extremely low frequency of lethals, in contrast to the findings of Ives (1945) in North America and of others elsewhere. This author then suggested that Korean populations are different from others, in respect to population size. In the present investigation, 138 second and 151 third chromosomes collected in mid-July of 1962 in Seoul were analysed by Cy and TM3 balancer chromosomes. The numbers of lethals and semi-lethals are 13.0% for second and 18.5% for third chromosomes. 160 second and 164 third chromosomes were taken in mid-October and 17.5% second and 16.6% third chromosomes were lethals and semi-lethals. Another sample was collected in rural Kwangnung in mid-September and 16% of second (176 chromosomes) and 20% of 221 third chromosomes taken were lethals and semi-lethals. Differences in lethal concentration between second and third chromosomes are not significant in time and space. However, on the average slightly more third chromosomes than second chromosomes carry lethals. Additional data showed a high value of homozygous viability of both second and third chromosomes above semi-lethals and of quasi-normals: on the average the mean viability of chromosomes above semi-lethals appears to be 30.4 ± 0.3% for second and 31.0 ± 0.5% for third chromosomes. Tests showed comparatively high frequency of allelism among the strict lethals in both second and third chromosomes, showing no significant differences between collections in the same location or between locations (1 to 3% for III and 1% for II chromosomes). The total incidence of visibles was low in each collection, but more than half of the different visibles occurred more than once within each population. The mutants bw and p which appear to be allelic, respectively, to the visibles marked on our Cy and TM3 chromosomes occurred in high frequency. Many of the wild males tested, which carried either bw or p, were further proved to have more than one lethal and/or other deleterious genes. The frequencies and allelism of second and third chromosomes of lethals and other genetic variabilities agree well each other, and a bunch of the results falls within the expected range showing smallness of population size. It may be safe to conclude that the Asiatic populations of D. melanogaster are really different from other geographical populations so far reported, in respect to population size and genetic structure.

Reitan, P. J. and M. E. Annan A study of the influence of the degree of desiccation on the frequency of X-ray induced developmental failure in Drosophila.

A previous study (DIS 36:111) indicated that the dehydration of Drosophila females prior to their exposure to irradiation increased the frequency of developmental abnormalities in the embryonic stages of subsequent offspring. This particular study was undertaken to test the previous work with increased numbers of embryos and to study the effects of different degrees of desiccation.

Virgin Drosophila females were treated with 4,000 r of X-ray following dehydration at 25% humidity in a desiccation chanber. Flies were dehydrated for 0, 4 and 8 hours. Eggs were collected on the 5th, 6th and 7th days following treatment and were allowed to develop for 14-16 hours before they were fixed for embryological examination.

The data indicate that the frequency of developmental failure is increased with an increase in the severity of desiccation. The increase noted is one which occurs in the number of eggs which show a complete lack of development and will include unfertilized eggs as well as those in which very early failures have taken place.

| Hours of desiccation | No. of eggs examined | % showing developmental failure | % completely undeveloped |
|---|---|---|---|
| 0 | 236 | 54 | 39 |
| 4 | 621 | 66 | 47 |
| 8 | 860 | 72 | 58 |

(This work was supported by grant GM 11645-06 from the National Institutes of Health)

Erickson, John Cytological study of a case of meiotic drive.

Novitski and Hanks (1961 Nature 190:989-990) reported a case of meiotic drive in which there was regularly an excess of X over Y sperm, resulting in a sex ratio of approximately $2\varphi\varphi:1\sigma$.

A cytological study of spermatogenesis has revealed a number of meiotic abnormalities. The most significant aberration involves breakage of the Y chromosome at the anaphase of the second meiotic division. In some 63% of Y-bearing second division cells at least one Y shows a break, usually in the long arm; the fragment remaining on the spindle (Fig. 1). Sometimes both Y's are broken, and two or more fragments are seen (Figures 2, 3 and 7). If it is assumed that the fragments lead to gametic death, as distinct from zygotic death, then this may represent the major mechanism in differential production of X and Y sperms. The proportion of affected Y second divisions is consistent with the degree of disturbance of the sex ratio.

Genetic studies show that breakage of the Y is brought about by a factor on the X, near the euchromatic-heterochromatic junction. This implies that the definitive action precedes first anaphase, as is also indicated by time-temperature experiments (Erickson and Hanks, 1961 Amer. Nat. XCV:247-250). The appearance of the fragments at AII might, therefore, be a delayed effect. Some support for this suggestion is offered by the following observations: A broken Y is seldom seen before AII, but they do occur. Demonstration of the earlier breakage is facilitated by the use of a ring Y. Figure 4 shows two sister MII cells; in one, both Y ring-chromatids are broken, the X chromatids in the other nucleus being normal. In figure 5 both Y chromatids are deficient, but since no fragments are present, it must be inferred that they were excluded on the first division spindle.

Legend →

Figs. 1,2,3 AII cells showing Y chromosome breakage. Fig. 4 Sister MII cells showing ring Y breakage at left and a normal X cell on the right. Fig. 5 AII with two deficient Y chromatids. At upper pole the Y is partially obscured by an autosome. Fig. 6 AI with anaphase bridge in an autosome pair. Fig. 7 AII with an extra autosome resulting from AI non-disjunction and showing Y chromosome breakage. (Object at left of two fragments is an artifact). Fig. 8 AII deficient for one autosome.

Further irregularities also occur in this stock. These include AI bridges of an autosome pair (fig. 6) or of the sex chromosomes. Rarely, AII bridges are seen. Some AI's are grossly abnormal. Non-disjunction is high - some 15% of cells being affected. Autosomal non-disjunction is shown in figures 7 and 8: Figure 7 also shows Y chromosome fragmentation. The high non-disjunction and other abnormalities cited undoubtedly contribute to the poor fertility of this stock, and possibly to the observed sex ratio.

Sironi, G. P., E. Gallucci and S. Giavelli Low temperature effect on X-ray induced translocations.

In order to study whether low temperature has an effect on the repair of X-ray damage to chromosomes, we irradiated Drosophila b cn vg heterozygous males with a single 2000r dose or two 1000r fractionated doses, submitting the flies to temperatures of 25°C or 0°C during the fractionation interval. We planned to study the radiation damage and repair in haploid male germ cells; we irradiated adult males soon after eclosion and we crossed each male with three Cy L Pm H Sb virgin females every day, for 8 days.

At the same time we scored the first X-ray induced crossing over recombinants from irradiated b cn vg heterozygous males, crossed to three b cn vg females every day with the daily brood technique. We found the first diploid cells at the seventh day of the X-ray sensitivity pattern.

We scored the progeny of the crosses for translocations, but did not find any difference between the sensitivity patterns obtained after single, and fractionated doses at normal temperature. A higher frequency of induced translocations was observed in the 4th - 5th and 6th day of the experiments with low temperature in the interval fractionation time.

Krimbas, C. B. Drosophila species captured in Greece.

Another two species should be added to the list of Drosophila species in Greece (Krimbas, C. DIS 37:95 and Tsacas, L. DIS 37:135): D. kuntzei, collected in Karpenisi in forests and D. obscura, collected in Karpenesi in forests. (In the notes of Krimbas DIS 37:95-96 please read D. ambigua instead of D. obscura). The total number of Drosophila species captured in Greece until now reaches 18, three of which belong to the obscura group (D. subobscura, D. obscura and D. ambigua).

Novitski, E. The origin of mosaics.

In experiments involving the irradiation of the doubly marked Y chromosome, $y^{+}YB^{S}$, Novitski, Cohen and Laird (1962) have shown that mosaics occur with an appreciable frequency. Among a total of 58 changes in which Bar was lost, but the normal allele of yellow was not, 15 proved to be mosaics, either after closer examination of the individual which proved to have one eye Bar and the other not, or after progeny tests which indicated that the apparent non-mosaic in fact must have carried both a changed and an unchanged chromosome. Since such changes must originate from chromosomal breaks rather than from point events, the arguments which have been advanced to explain mosaicism at the genic level should be generalized (if possible) to include mosaicism for gross chromosomal abnormalities.

Muller, Carlson and Schalet have proposed rotational substitution of base pairs as an explanation for whole body (gene) mutations, the remainder then affecting usually only one strand and resulting in mosaicism. It does not seem likely, however, that such an explanation could be extended to include whole body and mosaic changes brought about by breakage events. We would like to suggest an alternative which will account for both genic and chromosomal types of mosaicism. Assume that one of the first two cleavage products in the egg can lead to complete development even if the other is lethal, and that the chromosomes are in a doubled state in the mature sperm. A mosaic might arise, then, when one of the two strands is affected, followed by their separation at the first cleavage division into two viable products, each making a contribution to the soma of the developing individual. A whole body mutation arises, similarly, when only one of the two strands is affected, but when one of the two first division cleavage products (the one not carrying the change) is lethal. This lethality could arise from dicentrics or inviable chromosomal combinations induced by the treatment in any of the other chromosomes.

On this basis, then, the grossly different frequencies of mosaicism found when X-rays and chemical mutagens are compared could be attributed, for the most part, to the different frequencies with which lethal changes (killing off one of the two first division products) are induced by the two types of agents.

Nirula, Satya, R. P. Sharma, M. S. Swaminathan, and A. T. Natarajan. Incidence of crossing over in male Drosophila fed on irradiated medium.

An experiment was designed with the aid of second chromosome recessive markers (b cn bw) to see whether the indirect effects of radiations through feeding irradiated medium could induce crossing over in male Drosophila.

Freshly laid eggs of the genetic constitution + + +/b cn bw were transferred onto the basic medium (comprising glucose, agar and yeast) irradiated with 150,000 rads of $Co^{60}$ $\gamma$ rays. A comparable number of controls were transferred to the unirradiated basic medium. The larvae completed development in the same culture vial without further transfer. The adult males (of the constitution + + +/b cn bw) emerging out of these larvae were mated individually to untreated virgin b cn bw/b cn bw females. The progenies were scored for crossovers between these loci. In a total of 100 crosses studied, recombinations which could be ascribed either to somatic or meiotic crossing over in the male germ line, were recovered only twice.

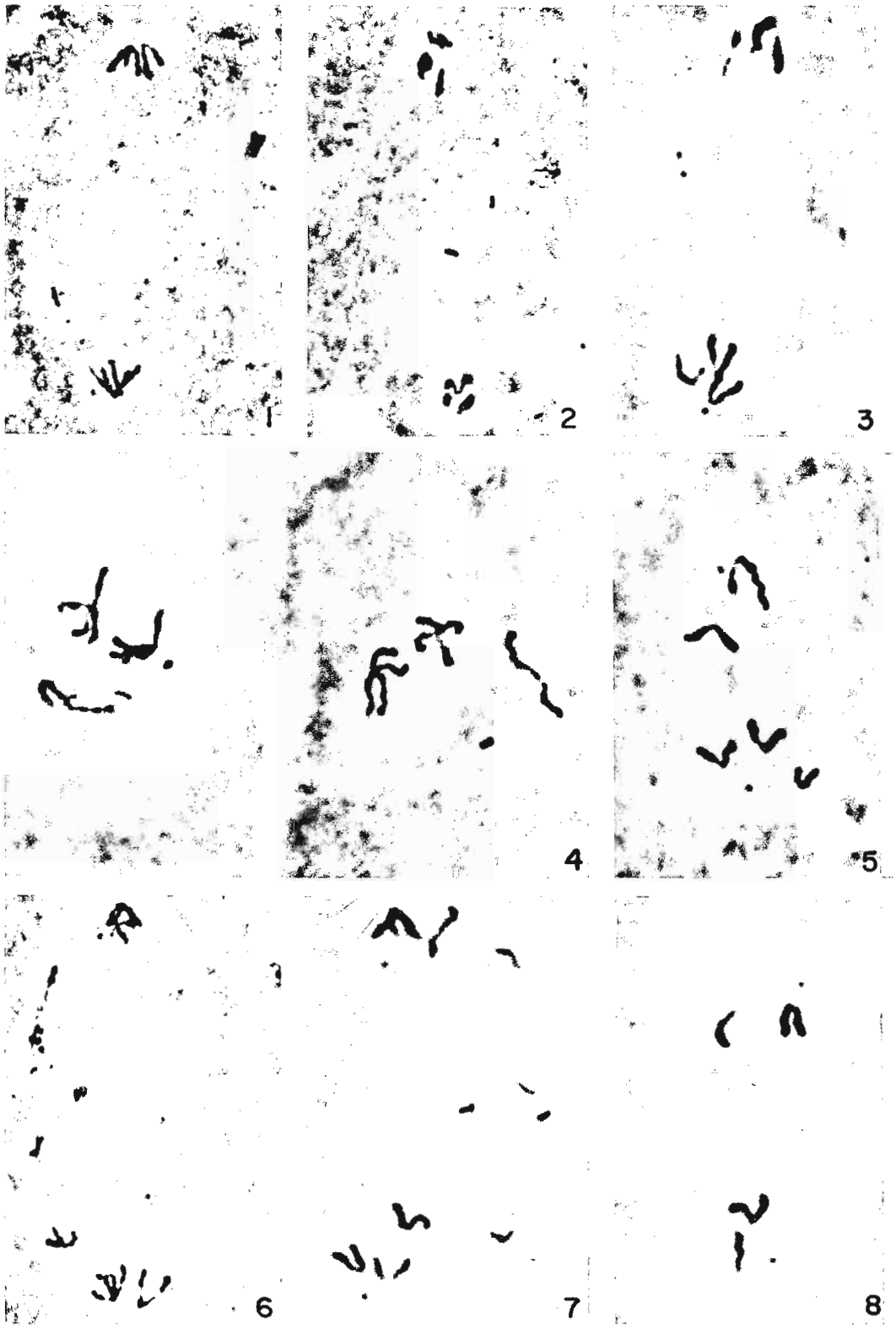
Crossing over occurred in one case between b and cn bw resulting in the expected parental and crossover classes viz., + + +/b cn bw, b cn bw/b cn bw and b + +/b cn bw, + cn bw/b cn bw.

In the second vial, besides the parental types, offspring resulting from crossing over between b and cn bw and cn and bw could be distinguished. These were as follows: i) b + +/b cn bw; + cn bw/b cn bw and ii) + + bw/b cn bw; b cn +/b cn bw. Out of a total of 137 flies, the former types were 14 and 10 and the latter 7 and 9 respectively; the rest belonged to the two parental classes.

Though the possibility of translocations having occurred in the material cannot be ruled out, it seems likely from the total absence of sterility or other abnormalities in the material that only crossing over is involved.

Rinehart, R. R., R. M. Valencia, and J. I. Valencia. Comparative lethal rates from X-irradiated D. melanogaster males and females mated together or to unirradiated partners.

The possibility was considered that irradiated D. melanogaster stage 14 oocytes might be less able to repair X-ray-induced damage in fertilizing sperm than was the cytoplasm of eggs receiving no X radiation. Either irradiated D. melano-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Carnes, Roger W. Allelism of sex-linked "biochemical" lethals.

Eleven lethals of different origin, all of which are viable when present in individuals mosaic for normal tissue as well as the lethals, are located at the extreme left end of the X-chromosome and are covered by the tip of the X carried on the scute-8·Y-chromosome. It is possible, therefore, to make allelism tests of these and the results show that 192 and 162 are allelic; also, that 152, 1403, 139 and 1402 are allelic. The other lethals, 113, 187, 1147, 1168, 128, 131 and 133 did not appear to be allelic to each other or to the other two groups.

The high incidence of allelism in these randomly derived lethals suggests that the total collection of "biochemical" mutants now available in the Novitski laboratory represents a substantial majority of the total that will ever by found on the X-chromosome.

Keith, A. D. Analysis of the fatty acids of Drosophila melanogaster.

The fatty acids of D. melanogaster have been analyzed by gas liquid chromatography using a Perkin-Elmer model 800 gas chromatograph equipped with a 15 foot resoflex packed column. The present report will give only the relative integrator response of each fatty acid present in Oregon R that falls between 12 and 18 carbons because fatty acids that have a carbon number of less than 12 occur in minute quantities and are extremely variable, and compounds with carbon numbers greater than 18 occur only in trace amounts. Traces of a 20 carbon fatty acid have been found in some stocks (e.g. yellow).

Larvae were raised on yeast and the late third instar larvae were washed free of yeast and homogenized in glass. Lipid was extracted from this homogenate by using a modification of the Folch method (Folch, J., M. Lees and G. H. Sloane Stanley, JBC 226:497, 1959). The total lipid content was fractionated and the fatty acid fraction was methylated for gas liquid chromatography. Identification of the peaks appearing on the gas chromatograms has been done by using appropriate standard mixtures of methylated fatty acids and by calculation of the equivalent carbon chain length (Miwa, T. K., K. L. Mikolajzak, E. R. Fontaine and I. A. Wolff, Anal. Chem. vol. 32, no. 13:1739, 1960).

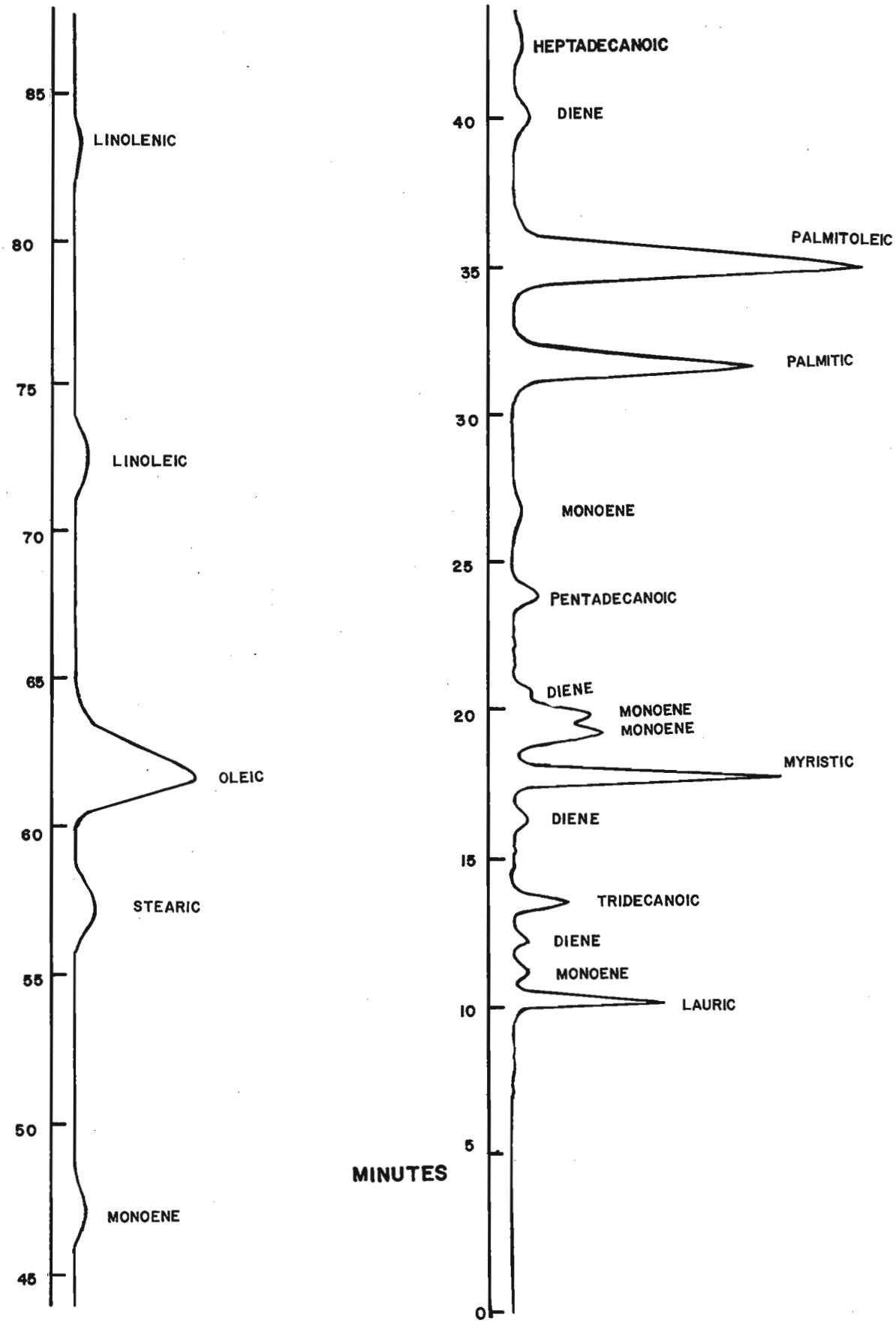
The percentage of the total integrator response contributed by each fatty acid is shown in table 1. Integrator response is a measure of the area under a peak and is proportional to the molar quantity.

| Compound | Relative percentage of total integrator response | Compoundd | Relative percentage of Total integrator response |
|---|---|---|---|
| 12 lauric acid | 3.53 | 15:1 monoene* | .19 |
| 12:1 monoene* | .46 | 16 palmitic acid | 19.58 |
| 12:2 diene* | .30 | 16:1 palmitoleic acid | 33.78 |
| 13 tridecanoic acid | 1.59 | 16:2 diene | .38 |
| 13:2 diene* | .38 | 17 heptadecanoic acid | .19 |
| 14 myristic acid | 10.44 | 17:1 monoene* | .30 |
| 14:1 monoene* | 7.83 | 18 stearic acid | 1.68 |
| 14:1 monoene* | | 18:1 oleic acid | 17.58 |
| 14:2 diene* | | 18:2 linoleic acid | .72 |
| 15 pentadecanoic acid | .99 | 18:3 linolenic acid | .10 |

Table 1: The number before the colon refers to the number of carbons in the fatty acid and the number following the colon refers to the number of unsaturated positions. *Identified by mathematical calculations only (standards are soon to be available for their confirmation). It must also be mentioned that the column being used does not distinguish between hydroxy-branched compounds and other fatty acids with the same chain length.

Figure 1 shows a tracing of a chromatogram taken from a sample of Oregon R. This chromatogram was made by injecting 0.1 microliter of sample in petroleum ether into the instrument. The peaks made by petroleum ether occur immediately after injection and are not shown on the tracing.

Investigations show that the relative quantity of various fatty acids changes in different strains. These changes usually fit some pattern such as a shift to more or less unsaturated fatty acids or of a shift to more or less of the 12-14 carbon or 16-18 carbon fatty acids and various other changes of this general type. Usually a group of fatty acids change in such a way as to suggest a functional relationship between them.


85
LINOLENIC
80
75
LINOLEIC
70
65
OLEIC
60
STEARIC
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50
MONOENE
45
HEPTADECANOIC
40
DIENE
PALMITOLEIC
35
PALMITIC
30
MONOENE
25
PENTADECANOIC
DIENE
20
MONOENE
MONOENE
MYRISTIC
DIENE
15
TRIDECANOIC
DIENE
MONOENE
10
LAURIC
5
0
MINUTES

Welshons, W. J., and Nicoletti, B. Pseudoallelic recombination of the mutant white-coffee ($w^{cf}$).

The origin, gross cytology, localization, and phenotypic expression of $w^{cf}$ was previously reported by B. Nicoletti (DIS 34:52, 1960). In order to see if $w^{cf}$ would recombine with pseudoalleles at the white locus, several experiments utilizing $w^{cf}$, w and $w^{a}$ were performed in which second and third chromosome inversions were used to enhance crossing over.

In the cross $y^{2} w^{cf}/y\ w\ fa^{no}$; SM1 Cy/+; $Ubx^{130}$/+ females by y w $fa^{no}$ males, one y+++ recombinant chromosome was recovered as a yellow female heterozygous for $Ubx^{130}$. The total number of tested chromosomes was 21,200. In another cross, differing from the previous one only by having the single autosomal inversion $Ubx^{130}$, another yellow +++ recombinant chromosome was recovered in a total of 21,300. The third cross, in which only SM1 Cy was used, yielded no recombinants in 20,000. Thus, for a total of 62,500 chromosomes in the three experiments, one can estimate a recombination value of 0.0064%, with the $w^{cf}$ allele occupying a position to the left of w.

Since the mutant $w^{a}$ is also localized to the left of w, an attempt was made to separate $w^{cf}$ and $w^{a}$. In the cross $w^{cf}/y\ w^{a}\ fa^{no}$; SM1 Cy/+; $Ubx^{130}$/+ females by y $w^{a}$ $fa^{no}$ males, one y+++ chromosome (recovered as a yellow female), and one recessive lethal white recombinant carrying the marker $fa^{no}$ and heterozygous for SM1 Cy were found in a total of 123,000 tested chromosomes. The single y+++ recombinant would place $w^{cf}$ an estimated 0.0016 units to the left of $w^{a}$. The white recombinant is probably of the type described by Judd (Genetics, 46:1687, 1961) and Green (Genetics, 44:1243, 1959) and interpreted to be the consequence of asymmetric pairing and crossing over. However, Judd using $w^{a}$ and either $w^{Bwx}$ or $w^{bf}$, both to the left of $w^{a}$, finds the asymmetric pairing always to be in one direction, with a region to the left of $w^{a}$ asymmetrically paired to a region right of $w^{Bwx}$ or $w^{bf}$, whereas the white deficiency recovered in our crosses involving $w^{cf}$ and $w^{a}$ could be explained only by an exchange after asymmetrical pairing with reversed direction.

The possibility of separating by crossing over $w^{Bwx}$ or $w^{bf}$ and $w^{cf}$ has not yet been tested and therefore no conclusion can be drawn about the exact site of $w^{cf}$ among the alleles to the left of $w^{a}$. (Work performed in the Biology Division, Oak Ridge National Laboratory, operated by Union Carbide Corporation for the United States Atomic Energy Commission).

Tokunaga, C. A study of Extra sex combs$^{2}$ mutant in D. melanogaster.

The mutant $esc^{2}$ (extra sex combs$^{2}$) was found by Dr. O. Stroemnaes of the University of Oslo. When this mutant stock was tested after several months inbreeding in our laboratory, it turned out to be not recessive but dominant.

$Esc^{2}$ is homozygous lethal. Expressivity and penetrance is variable according to the genetic and environmental background. The data presented in the table show the dominant manifestation of this gene and the variability of the penetrance in males from different crosses tested under similar cultural conditions. Simultaneous work with the original esc allele confirmed its recessive nature.

| Cross | Penetrance in $Esc^{2}$ male offspring | | | Number of pair matings |
|---|---|---|---|---|
| | Average | Minimum | Maximum | |
| $Esc^{2}$/+ ♀ X Wild-type ♂ ($+^{3}$ stock) | 19% | | | 1 |
| y ac; $Esc^{2}$/+ ♀ X Wild-type ♂ ($+^{3}$ stock) | 36%± | 1% | 68% | 7 |
| y ac; $Esc^{2}$/+ ♀ X T(1,2) $sc^{19}$/y f:=, fes $sc^{19i}$ b pr/Cy dp pr ♂ | 85%± | 65% | 95% | 8 |
| y ac; Cy/Pm ♀ X y ac; $Esc^{2}$/+ ♂ | 67%± | 53% | 95% | 6 |
| y ac; Cy/$Esc^{2}$ ♀ X y ac; Cy/$Esc^{2}$ ♂ (both from the original cross y ac; Cy/$Esc^{2}$ X y ac; Cy/Pm) | 74%± | 24% | 95% | 9 |

Crossing over occurs between $Esc^{2}$ and Cy. Linkage tests of $Esc^{2}$ with the markers Bl, lt and $stw^{3}$ indicate that $Esc^{2}$ is located between Bl and lt. Data from x-ray induced somatic crossing over experiments also suggest that $Esc^{2}$ is located in the left arm of the second chromosome. Salivary chromosome tests show no inversions associated with $Esc^{2}$ in contrast to esc which is associated with an inversion.

Moree, Ray An effect of sodium propionate on viability.

Since a number of mold inhibitors used in culture medium have, in some degree, a toxic effect on Drosophila, or have other undesirable effects, I have used sodium propionate ($Ch_3CH_2COONa$) for a number of years in preference to other inhibitors. This substance is used extensively in the baking industry to inhibit mold and "roping" in bread and pastries and it was this that first suggested its use in fly cultures. That is to say, low toxicity was assumed on the basis of its industrial use. While its general toxicity may be relatively low for man and Drosophila, data now being compiled indicate that in the concentrations at which it has been used (about 0.8% by volume of dry powder), sodium propionate does lower the preadult viability of $e^{11}/e^{11}$ flies. The tabulated data, given as mean percentages of $F_2$ $e^{11}/e^{11}$ adults per half-pint culture bottle, were obtained from 24 cultures; each culture was initiated with 30 $+/e^{11}$ flies of each sex and each yielded a total of about 650 adult flies.

| Replicate # | 1 | 2 | 3 | 4 | Average/replicate |
|---|---|---|---|---|---|
| No propionate | 21.2 | 20.5 | 22.0 | 19.6 | 20.8 |
| Propionate | 12.1 | 11.0 | 16.4 | 11.9 | 12.9 |

It is obvious that the reduction of viability reported by Moree and King (Genetics 46:1735-1752, 1961) is influenced by the presence of sodium propionate. But it is also obvious that this reduction is not all due to sodium propionate, for viability varies with food supply and approaches its theoretical maximum when food is plentiful, even though propionate is present. Also, when propionate is lacking and there is some degree of food shortage, some reduction in viability still occurs, as the above data show. Of course, in considering any of our work on $e^{11}$ and its alleles, the effect of soduim propionate as an environmental factor will have to be kept in mind.

The situation which led to these results was as follows. Previously, medium contained sodium propionate and was autoclaved. When an autoclave was no longer available, Acid Mix "A" of Lewis (DIS 34:117) was added to the medium, which then contained both sodium propionate and propionic acid. When recently it was decided to omit the propionate, the above results were obtained. (Work supported by funds from the State of Washington Initiative Measure No. 171 for the Support of Biological and Medical Research.)

Krimbas, C. B. Inversion polymorphism in southern Greek populations of D. subobscura.

In addition to the five populations investigated and reported in DIS 37:95 two new ones (Vytina and Karpenisi) have been examined and two of the previously reported have been followed. The analysis of the data leads to several conclusions:

1) The inversion polymorphism in all these populations estimated by IFR values (using either Stumm-Zollinger and Goldschmidt or Kunze-Mühl and Sperlich tables for inversions' length) is as high as in other Central European populations. This fits well with the new data on geographic distribution of D. subobscura, which is found in North West Africa and Iran (Burla, personal communication): Greece is by no means a marginal population but rather a central one. These data are in accordance with the Dobzhansky-Carson hypothesis. A small sample from Dutch populations shows a small amount of polymorphism and a similarity with the Scotish ones. These places are in the north margin of the species distribution.

2) There is no obvious correlation between diversity of vegetation and inversion polymorphism in Greek populations.

3) Creta population differs from the other six in frequency of inversion types. The study of the Greek populations makes obvious the existance of geographic lines of inversions going from north to south: In chromosome A inversion A2 increases going south. In chromosome J, J ST is replaced by J1 and this last one by J 3+4. In chromosome U, U ST is replaced by U 1+2 and this by more complicated structural types as U 1+2+4 (in Creta U 1+2+8). In chromosome O, O ST is replaced by O 3+4 and in Creta this one by O 3+4+8. In chromosome E, E ST is replaced by E 1+2 and E8 and these by E 1+2+9 and E 1+2+9+12.

4) Within a year (1963) the changes of inversion frequencies in Parnes population is significant to the 5% level for chromosomes E and U. The full data will be reported soon elsewhere. (This research is supported by a grant of the Royal Hellenic Research Institution).

Siegel, I. M. and J. Hirsch Selection and threshold determination for the optomotor reflex.

Using a modification of apparatus and techniques developed by Selig Hecht and George Wald (J. Gen. Physiol. 17:511, 1934), bidirectional selection for extremes and stabilizing selection over 30 generations have produced three strains of D. m. differing in their optomotor responses. Luminance thresholds for eliciting the optomotor response at five different visual angles have been determined with a method of limits for the three strains developed by selection.

Their threshold functions have been fitted with straight line equations having identical slope parameters but different ordinate intercept parameters.

Schalet, A. Marked Y chromosomes.

From X-rayed $Y^{S} \cdot X$ InEN $y \cdot Y^{L}$ $sc^{8}$ $y^{+}$ crossed to y v f ma-$l^{bz}$ females. Two fertile $y^{+}$ v f ma-$l^{bz+}$ sons were recovered.

1) This $y+Yma\text{-}l^{+}$ #1 chromosome contained the normal alleles of car, Mn, sw, ma-l, $su^{w}$-f, but not of su-$w^{a}$. In addition it contained the normal alleles of at least 5 lethal loci located between car and ma-l and at least 2 lethal loci located between ma-l and $su^{w}$-f. Lost.

2) This $y+Yma\text{-}l^{+}$ #2 chromosome contained normal alleles of sw, ma-l, $su^{w}$-f, but not of $sc^{J1}$, su-$w^{a}$ and Mn. In addition it contained the normal alleles of at least 2 lethal loci (one of which is allelic to sw) located to the left of ma-l in the normal sequence and 6 lethal loci located between ma-l and $su^{w}$-f. Derivatives of this $y+Yma\text{-}l^{+}$ chromosome were obtained by irradiating males carrying the $y+Yma\text{-}l^{+}$, crossing them to y v $ma\text{-}l^{2}$ females, and selecting fertile $y^{+}$ v ma-l sons. Of 13 Y chromosomes obtained in this manner 12 proved to be deficiencies (as judged by their lethality with previously covered lethals to the left and/or right of ma-l). One of the Y chromosomes covered all the lethals but appeared ma-$l^{+}$ in combination with $ma\text{-}l^{1}$ or $ma\text{-}l^{bz}$ and may represent a non-complementary mutation of $ma\text{-}l^{+}$ rather than a deficiency for the locus.

Schalet, A. Additional information on $ma\text{-}l^{3}$: maroon-like$^{3}$

The mutant $ma\text{-}l^{3}$ reported in DIS-35 as a possible aberration appears to be a deficiency by genetic tests. The deficient region includes at least one lethal locus to the left of sw, sw, ma-l, at least 5 lethal loci between ma-l and $su^{w}$-f, and $su^{w}$-f, but not bb. The deficiency is covered by the $y+Yma\text{-}l^{+}$ #2 chromosome reported above.

Schalet, A. Location of ma-l: maroon-like

The mutant bz was reported by Fahmy and Fahmy (DIS 32) to be located on the X chromosome at 64.9 ± 0.2. Glassman (DIS 33) presented evidence of allelism between ma-l and bz. He also isolated f $Bx^{3}$ ma-l $su^{w}$-f recombinant flies which indicated that ma-l was located to the left of $su^{w}$-f. The report by Brosseau et al. (1961) that Bar Stone Y, $B^{S}Y$, did not carry $ma\text{-}l^{+}$ or $sw^{+}$, though it was known to carry $su^{w}\text{-}f^{+}$ (Zimmering 1959) confirmed the position of ma-l as being to the left of $su^{w}$-f. Brosseau et al. (1961) and also Grell (1962) state that sw is to the left of ma-l, although they do not indicate how this information was obtained.

Since the localization reported by the Fahmys did not utilize markers on both sides of ma-l, the localization was rechecked and the results which were obtained confirmed the map position previously reported.

Females which were y v f car $su^{w}$-f/$ma\text{-}l^{bz}$ were crossed to $ma\text{-}l^{bz}$ males. $F_1$ males carrying at least f car, representing crossovers between car and $su^{w}$-f, were selected and crossed to $ma\text{-}l^{bz}$ to test for the presence of $ma\text{-}l^{bz}$. Because of the maternal effect, early hatching $F_1$ males which are car $ma\text{-}l^{bz}$ usually do not show the mutant phenotype associated with $ma\text{-}l^{bz}$. Of 24 f car males, 16 also carried $ma\text{-}l^{bz}$. Since $su^{w}$-f is to the left of and extremely close to bb, the standard car to bb interval of 3.5 map units may be used to place ma-l at 64.8 (16/24 of 3.5). Furthermore, complementation tests with various chromosome duplications and deficiencies are consistent with the previously reported placement of ma-l to the right of sw. These tests also show that at least 5 lethal loci are located between ma-l and $su^{w}$-f.

Schalet, A. Attached X carrying maroon-like y v f Df(1)ma-l$^{3}$/y v f l ma-l$^{bz}$ From X-rayed y v f Df(1)ma-l$^{3}$·Dp(y$^{+}$ sc$^{V1}$)/ y v f l ma-l$^{bz}$ crossed to y v f ma-l$^{1}$ su$^{w}$-f· Dp(y$^{+}$ sc$^{V1}$). Arose as a single (y v f ma-l) female. Attachment was probably the result of a pseudocrossover where one break was to the right of the centromere and to the left of y$^{+}$ in one strand, the other break was to the left of the centromere and to the right of su$^{w}$-f$^{+}$. The lethal present in the arm carrying ma-l$^{bz}$ had previously been located between car and sw and to the left of the ma-l$^{3}$ deficiency.

Sperlich, D., G. Jaksch and A. Karlik Recessive lethals in island and continental populations of Drosophila melanogaster. Males of Drosophila melanogaster were collected at Formia (continent of Southern Italy) and on the small islands Ponza and Ventotene, situated at a distance of 33km from the coast, in autumn 1960 and in spring 1961 and 1962. The frequency of second chromosome lethals was investigated by using the L Cy/Pm method. All cultures producing less than 3.33% normals in the $F_2$ were counted as lethals. Table 1 shows the number of tested chromosomes and the frequency of lethals for the three localities examined. There is no significant difference between the frequency of lethals from the different localities. Further it is evident that there is no seasonal difference at Formia and Ponza.

To determine the rate of allelism, the strains from autumn collection containing a lethal second chromosome were crossed with one another. The rate of allelism is given in table 2. We found no significant difference between the island and the continental populations.

The frequency of lethals and the rate of allelism for the tested samples agree with the results found for American populations of Drosophila melanogaster but are different from those found for the populations from Korea and Japan.

Table 1
Frequency of second chromosome lethals for autumn and spring samples from Formia and the Isles of Ponza and Ventotene

| | Autumn | | Spring | |
|---|---|---|---|---|
| | number of tested chromosomes | frequency of lethals % | number of tested chromosomes | frequency of lethals % |
| Formia | 121 | 25.6 ± 4.0 | 107 | 33.6 ± 4.6 |
| Ponza | 126 | 23.8 ± 3.8 | 50 | 22.0 ± 5.9 |
| Ventotene | 64 | 25.0 ± 5.4 | - | - |

Table 2
Rate of allelism in the autumn samples from Formia, Ponza and Ventotene

| | number of crosses | rate of allelism |
|---|---|---|
| Formia | 210 | 0.95 ± 0.67 |
| Ponza | 284 | 1.41 ± 0.70 |
| Ventotene | 91 | 0.00 |

Tobari, I. and M. Nei Genetic effects of x-rays on the quantitative characters in a heterogeneous population. A number of studies have indicated that in a genetically homogeneous population the variation of quantitative characters is increased by radiations. From the standpoint of radiation hazard to man, however, it is important to examine the genetic effects of radiations in a heterogeneous population, since human populations are all heterogeneous. In a heterogeneous population the variation of quantitative characters might not necessarily be increased, because the polygenes responsible for these characters are mutable in both positive and negative directions.

One thousand virgin females and one thousand males taken at random from the base population, composed of 24 wild strains of D. melanogaster, were exposed to 2000r of 170 KVP X-rays. Immediately after irradiation, the flies were allowed to mate at random in a population cage. After three generations of random mating the effects of X-rays on the

abdominal bristle number were examined. This examination was conducted in two different ways, i.e., sib-analysis and artificial selection. The results of sib-analysis are tabulated as follows:

| Components of variance | | Irradiation | Control |
|---|---|---|---|
| Additive genetic | $V_A$ | 10.18 ± 2.81 | 7.47 ± 2.18 |
| Dominance | $V_D$ | - 0.11 ± 3.30 | 2.15 ± 2.68 |
| Environmental | $V_E$ | 6.90 ± 1.53 | 4.38 ± 1.33 |
| Phenotypic | $V_P$ | 16.97 ± 0.54 | 13.99 ± 0.44 |
| Heritability | $h^2$ | 0.599 | 0.533 |

It is seen that the total phenotypic variance ($P_V$) has increased considerably by the irradiation of X-rays, the difference between "irradiation" and "control" being statistically significant at the 5% level. The partitioning of phenotypic variance into its components shows that the increase of phenotypic variance has been brought about mainly by the increased amount of additive genetic and environmental variances, though neither of these increments is statistically significant. The estimates of heritability are almost the same both for the irradiated and control populations. Upward and downward selections have been carried out for 13 generations and the results obtained so far indicate no significant difference in the response to selection between the irradiated and control populations.

Kang, Y. S., Y. J. Kim, C. C. Lee and J. J. Choi. The frequency of sex-linked recessive lethal mutation in D. melanogaster irradiated with 1500r of X-rays.

The determination of the frequency of sex-linked recessive lethal mutations in D. melanogaster (Seoul strain) males irradiated with 1500r of X-rays was carried out in this study. The frequency was determined at three intervals during spermatogenesis after irradiation.

In 1963, Ives observed different frequencies in several mutants which were irradiated with the same dosages of r-rays. Reviewing works of various authors, mutation rates seem to be different from one material to another even when the same doses are given.

Drosophila melanogaster (Seoul strain) males and M-5 females were used for the material in the present experiment. The results are summarized in tables 1 and 2.

Table 1

The frequency of sex-linked recessive lethal mutation at intervals after irradiation.*

| | Time mating was made after irradiation (days) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 1 - 2 | | | 3 - 4 | | | 5 - 6 | | |
| Time eggs were laid after mating (days) | No. of chromo. tested | No. sex-linked mutations | % | No. of chromo. tested | No. sex-linked mutations | % | No. of chromo. tested | No. sex-linked mutations | % |
| 1 - 2 | 299 | 25 | 8.36 | 299 | 20 | 6.68 | 154 | 15 | 7.73 |
| 3 - 4 | 310 | 27 | 8.70 | 307 | 24 | 7.81 | 70 | 6 | 8.57 |
| 5 - 6 | 306 | 19 | 6.20 | 310 | 19 | 6.12 | 105 | 8 | 7.61 |
| Total | 915 | 71 | 7.75 | 916 | 63 | 6.87 | 369 | 29 | 7.85 |

* New males and females were used for crossing in every mating time.

The mean frequency of sex-linked lethal mutation appeared in 1-2 days after irradiation was 7.3% in D. melanogaster Seoul strain.

At three intervals tested, no significant difference in the frequency was observed in this study, in contrast with the result of Reddi and Mathew (1963) which showed 7.65% on the first day after 3000r irradiation followed by a decrease to 4.95% on the second day.

The frequency of lethal mutation during spermatogenesis showed 7.02% on 1-2 days and increased to 9.32% on 5-6 days. It seems that the rate of mutation in spermatocyte is higher than that of mature spermatozoa. According to Ives' report (1963), the frequency increases suddenly from 1-3 days to 5-6 days during spermatogenesis.

Table 2. The frequency of sex-linked recessive lethal mutation during spermatogenesis after irradiation.**

| | Time mating was made after irradiation (days) | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | 1 - 2 | | | 3 - 4 | | | 5 - 6 | | |
| Time eggs were laid after mating (days) | No. of chromo. tested | No. sex-linked mutations | % | No. of chromo. tested | No. sex-linked mutations | % | No. of chromo. tested | No. sex-linked mutations | % |
| 1 - 2 | 299 | 25 | 8.36 | 313 | 20 | 6.38 | 94 | 8 | 8.51 |
| 3 - 4 | 298 | 20 | 6.71 | 301 | 31 | 10.29 | 24 | 3 | 12.5 |
| 5 - 6 | 300 | 18 | 6.00 | 308 | 30 | 9.74 | -- | -- | -- |
| Total | 897 | 63 | 7.02 | 922 | 81 | 8.75 | 118 | 11 | 9.32 |

** Just the same males were only used for crossing in all mating times.

Sävhagen, Ruth and Barbro Kristofferson
Induced translocations in spermatogonia and oogonia of D. melanogaster.

17 hour old Canton-S larvae were irradiated with 900r in air. The dose was chosen in order to ensure good viability and fertility of the adults which have been treated as larvae. After eclosion the males and females were separated and individually mated respectively to 2-3 cn bw; $e^{11}$ ♀♀/cn bw; $e^{11}$ ♂♂ for 3 days.

On the 14th day after the mating period started 15 $F_1$ ♂♂ heterozygous for the irradiated genome were picked up from each vial. These males were then tested individually for the occurrence of induced translocations. $F_1$ ♂♂ from males which were irradiated as larvae were only tested for the occurrence of II-III translocations, while $F_1$ ♂♂ from irradiated female larvae were tested for I-II; I-III; II-III and I-II-III translocations. All $F_2$ cultures which did not yield an adequate number of flies to enable determination of whether or not a translocation was present were retested.

Several repeats were made. The results are presented in Tables 1 and 2.

Table 1. Frequency of induced translocations in spermatogonia.

| Exp. # | Number of irradiated P larvae | Number of observed translocations/ tested sperm | |
|---|---|---|---|
| 1. | 53 | 4/767 | (0.52%) |
| 2. | 33 | -/475 | |
| 3. | 52 | -/727 | |
| 4. | 21 | -/299 | |
| 5. | 53 | -/763 | |
| 6. | 24 | -/347 | |
| Total | 236 | 4/3378 | (0.12%) |

The 4 translocations observed in experiment #1 are observed among the offspring from 4 different larvae and are thus not due to some clustering effect.

Table 2. Frequency of induced translocations in oogonia.

| Exp. # | Number of irradiated P larvae | Number of observed translocations/ tested egg |
|---|---|---|
| 1. | 25 | -/363 |
| 2. | 6 | -/ 88 |
| 3. | 56 | -/828 |
| 4. | 60 | -/843 |
| Total | 147 | -/2122 |

Sävhagen, Ruth The frequency of induced genetic damages after irradiation of D. melanogaster males in $N_2O$ atmosphere.

0-1 day old $y^{16}:sc^{8}Y$ males were irradiated with 1100r in $N_2O$ atmosphere. The flies were kept 15 minutes in $N_2O$ before irradiation. Immediately after irradiation the males were transferred to a mating cage. Every second day the males were transferred to a new mating cage, and thus, up to the 5th day a.i. the males were kept with an excess of virgin females. From the 6th day a.i. to the 10th day a.i. the mating periods consisted of 24 hours, and the irradiated males were mated to y w sn ♀♀. The offspring from this cross would give y ♀♀ and w sn ♂♂. The occurrence among the offspring of y w sn ♂♂ was taken as an indication of induced losses of the X and/or Y chromosomes or at least the marker gene $sc^{8}Y$ (=XO males), while wild type females (y w sn/$y^{16}$ w sn$^{+}$:$sc^{8}Y$) were the result of induced non-disjunction between the paternal sex-chromosomes. Since the wild type females may also originate from induced crossing-over between the X- and Y-chromosomes each wild type female was tested individually.

Several repeats were made. The data are pooled in the tables. The heterogenity between the experiments is measured through chi-square tests.

Table 1. 0-1 day old $y^{16}:sc^{8}Y$ males irradiated with 1100r in $N_2O$ atmosphere. Number of XO males, total number of offspring (n) and frequency of XO males in successive mating periods are given.

| | Time after irradiation | | | | |
|---|---|---|---|---|---|
| | 6th day | 7th day | 8th day | 9th day | 10th day |
| | XO♂♂ n | XO♂♂ n | XO♂♂ n | XO♂♂ n | XO♂♂ n |
| Control air$^{XX}$ | 10/23044 | 12/23577 | 14/22601 | 4/11729 | 5/10388 |
| | = 0.04 % | = 0.05 % | = 0.06 % | = 0.03 % | = 0.05 % |
| Control, $N_2O$ | 11/23924 | 3/32161 | 11/28475 | 5/29666 | 12/31021 |
| | = 0.05 % | = 0.01 % | = 0.04 % | = 0.02 % | = 0.04 % |
| 1100r, $N_2O$ | 75/41904 | 222/79707 | 276/61702 | 163/34463 | 67/24315 |
| | = 0.18 % | = 0.28$^{X}$ % | = 0.45$^{X}$ % | = 0.47 % | = 0.28 % |
| 1100r, air$^{XX}$ | 328/24735 | 400/18100 | 463/13350 | 186/ 7772 | 152/27946 |
| | = 1.33 % | = 2.21$^{X}$ % | = 3.47$^{X}$ % | = 2.39$^{X}$ % | = 0.54 % |
| 1100r, $N_2^{XX}$ | 60/24542 | 158/32715 | 56/17086 | 53/14201 | 38/24900 |
| | = 0.24$^{X}$ % | = 0.48$^{X}$ % | = 0.33$^{X}$ % | = 0.37 % | = 0.15 % |

Table 2. 0-1 day old $y^{16}:sc^{8}Y$ males irradiated with 1100r in $N_2O$ atmosphere. Number of wild type females, total number of offspring (n) and percentage of wild type females in successive mating periods are given.

| | Time after irradiation | | | | |
|---|---|---|---|---|---|
| | 6th day | 7th day | 8th day | 9th day | 10th day |
| | XO♂♂ n | XO♂♂ n | XO♂♂ n | XO♂♂ n | XO♂♂ n |
| Control air$^{XX}$ | 3/23044 | 6/23577 | 2/22601 | 1/11729 | 1/10388 |
| | = 0.01 % | = 0.03 % | = 0.01 % | = 0.01 % | = 0.01 % |
| Control, $N_2O$ | 22/37624 | 15/32161 | 13/28475 | 11/29666 | 6/31021 |
| | = 0.06 % | = 0.05 % | = 0.05 % | = 0.04 % | = 0.02 % |
| 1100r, $N_2O$ | 14/41904 | 25/79707 | 47/61702 | 25/34463 | 15/24315 |
| | = 0.03 % | = 0.03 % | =0.08$^{X}$ % | = 0.07 % | = 0.06 % |
| 1100r, air$^{XX}$ | 2/24735 | 40/18100 | 68/13350 | 15/ 7772 | 49/27946 |
| | = 0.01 % | =0.22$^{X}$ % | = 0.51 % | = 0.19 % | = 0.18 % |
| 1100r, $N_2^{XX}$ | 12/24542 | 25/32715 | 25/17086 | 9/14201 | 13/24900 |
| | = 0.05 % | = 0.08 % | = 0.15 % | = 0.06 % | = 0.05 % |

X = heterogeneity within the material. XX = from Sävhagen 1961.

From Table 1 it is seen that the number of XO males increases from the 6th day, reaches a maximum on the 8-9th day, and then decreases towards the 10th day after treatment. This is in agreement with earlier findings (Sävhagen 1961).

When examining the frequency of induced wild type females an increase in the rate is observed from the 7th day a.i. to the 8th day, thereafter the rate is essentially constant.

These data support the hypothesis that irradiation performed in $N_2O$ atmosphere, like irradiation in $N_2$ atmosphere reduces the yield of XO males as well as non-disjunction females.

Schwinck, Ilse Non-autonomy of Malpighian tube function in transplantation experiments.

In earlier experiments, transplantation of rosy imaginal eye discs into maroon-like hosts and vice versa had shown that there is no complementation with respect to drosopterin formation in the implant or in the host eyes, although both mutants are non-autonomous in drosopterin formation in reciprocal transplantation experiments with wild type (Hadorn und Schwinck, 1956, Z.F. Vererbungslehre 87 p. 528; Schwinck, 1960, DIS 34). Recently, reciprocal transplantation studies with Malpighian tubes between rosy and maroon-like were carried out in order to study possible complementation of subunits released by the mutant cistrons in this tissue. According to Ursprung and Hadorn (Experientia 17, p. 230, 1961), the wild type Malpighian tube show xanthine dehydrogenase activity in homogenate supernatants. No complementation in eye pigment formation or in the morphological aberrations of the Malpighian tubes was found when Malpighian tubes of both mutants were combined in rosy or in maroon-like hosts. Moreover, reciprocal Malpighian tube transplantations between rosy and wild type demonstrated, that tubule implants dissected several days after emergence of the flies, behave non-autonomously with respect to the excretion of fluorescent globules and cell morphology. This means, that implanted $rosy^+$ Malpighian tubes release the $ry^+$ factor and induce increased drosopterin formation during metamorphosis in the host eyes; however, in older flies they appear like rosy excretory organs. The rosy Malpighian tubes implanted in wild type hosts look like the wild type; however, after reimplantation in rosy larvae and going through a second metamorphosis they appear again like rosy tubules and do not induce increased drosopterin formation in the host eyes.

Lee, William R. Combination of the "Maxy" chromosome for detecting specific locus mutations with $sc^8 \cdot Y \cdot B^S$ for detecting loss of the Y or X chromosome.

In many studies of mutagenesis it is desirable to measure from the same treated individual both chromosomal aberrations and mutations at specific loci. This was done by I.I. Oster (1963, Repair from Genetic Radiation Damage, Ed. Sobels, F. H. 454 pp. Pergamon Press, N.Y.) in a multi-purpose stock in which the $sc^8 \cdot Y \cdot B^S$ chromosome was used to detect certain structural changes along with autosomal translocations, sex-linked recessive lethal mutations, and visible mutations at the dumpy locus. Absence of $sc^8(y^+)$ and/or $B^S$ markers in the male progeny was an efficient measure of certain types of chromosomal aberrations.

It would be desirable to combine this method of detecting structural changes in the Y or X chromosome with the "Maxy" stock for detecting mutations at specific loci (1957, Muller and Schalet, DIS 31 : 144); however, this is not feasible because loss of the $sc^8$ region of the Y chromosome would uncover the 1J1 region of the X chromosome in the "Maxy" stock and kill the male. This difficulty was overcome by using Fl (1961, Zimmering and Muller, DIS 35: 103-104) in place of 1J1 to kill the unwanted class of females. Fl has normal viability in the male, though unfortunately it has reduced viability in the heterozygous female due to its partial dominance. Zimmering and Muller (1961) found that its partial dominance was influenced by modifiers. To overcome its partial dominance a number of chromosomes was made containing Fl within In49 (fortunately Fl was originally found in In49) and suitable markers. Two of these chromosomes showing high viability when heterozygous in females were carried as follows: "Stock M" In49 v ptg oc Fl $B^{Ml}$ & y f := and "Stock Y" y In49 v Fl g $B^{Ml}$ & y f :=. By carrying these two chromosomes in this way there was constant selection against lethals and detrimentals. Males from Stock M are then crossed with $sc^8(y^+) \cdot Y \cdot B^S$/y f := virgin females (Stock f75 Philadelphia Stock List DIS 35:23). Progeny from nonvirgin parental females can be readily detected and eliminated. The $sc^8 \cdot Y \cdot B^S$/In49 v ptg oc Fl $B^{Ml}$ males of this cross are then treated (or their sperm may be treated) and mated to automatic virgins y In49 v Fl g $B^{Ml}$/ "Maxy" produced by crossing "Stock Y" males to "Maxy" virgins. The "Maxy" stock has had the $sc^8 \cdot Y \cdot B^S$ chromosome substituted for the $1J1^+ \cdot Y$ chromosome, so that females containing the Y chromosome because of nondisjunction can be eliminated.

The progeny in the first generation following treatment are then scored. The only class of normally produced males $sc^8 \cdot Y \cdot B^S$/y In49 v Fl g $B^{Ml}$ is checked for loss of the $sc^8(y^+)$ and/or, $B^S$ markers on the Y. Males produced by nondisjunction in the mother can be detected by the absence of g. The only class of normally produced females In49 v ptg oc Fl $B^{Ml}$/ "Maxy" is checked for visible mutations at the 14 loci of the "Maxy" chromosome. Females produced by nondisjunction can be detected by the presence of $B^S$, and the vermilion eye of

the female makes the other eye color mutants (except pn) easier to detect. The normal female may then be tested for sex-linked recessive lethals. Viability of all these stocks has been found to be good under our laboratory conditions. (I wish to express my appreciation to Dr. H. J. Muller for advice during the construction of these stocks. This investigation was supported by Public Health Service Research Grant GM 11449-01 from the National Institute of General Medical Sciences).

Inagaki, H. and Y. Nakao Modification of X-ray induced visible mutation frequencies by chromosome structure.

48 hr old males of Canton-S and y $sc^{S1}$ In49 $sc^{8}$; bw; st (from Institute of Animal Genetics, Edinburgh and abbr. 0-1) were X-irradiated with the dose of 3000r (x-ray apparatus was operated at 200 KvP and 20 mA), and mated immediately with y, w, m, f ♀♀ (originally provided from the Zoological Institute, Kyoto Univ., and these two mutant strains are pseudoalleles with respect to y locus). The visible mutation frequencies at 4 loci were determined examining the $F_1$ females.

| Strain | Dose | No. of tested $F_1$ females | No. and percentages of mutants y | w | m | f |
|---|---|---|---|---|---|---|
| 0-1 | 3000r | 7924 | 36 (0.45) | 5 (0.06) | 2 (0.03) | 0 ( - ) |
| | 0r | 7671 | 2 (0.03) | 1 (0.01) | 1 (0.01) | 0 ( - ) |
| Canton-S | 3000r | 6845 | 3 (0.04) | 8 (0.12) | 3 (0.04) | 0 ( - ) |
| | 0r | 13665 | 0 ( - ) | 0 ( - ) | 0 ( - ) | 0 ( - ) |

In the above table, there were no difference at 3 loci, w, m, and f. However, the rate at the irradiated y locus of the 0-1 strain was almost 10 times higher than that of the Canton-S strain, and in these cases the number of males was found to be about half that of th females. It may be said that the mutations at the y locus may represent very small deficiencies with hemizygous lethal effects.

The visible mutation frequencies were compared between the 0-1 and M-5 strains to clarify whether inversions may be responsible for the differences, since 0-1 has an inversion in the X chromosome.

| Strain | Dose | No. of tested females | No. and percentages of mutants y | w | m | f |
|---|---|---|---|---|---|---|
| M-5 | 3000r | 8111 | 16 (0.22) | 2 (0.03) | 4 (0.05) | 1 (0.01) |
| | 0r | 2835 | 0 ( - ) | 0 ( - ) | 0 ( - ) | 0 ( - ) |
| 0-1 | 3000r | 7924 | 36 (0.45) | 5 (0.06) | 2 (0.03) | 0 ( - ) |
| | 0r | 7671 | 2 (0.03) | 1 (0.01) | 1 (0.01) | 0 ( - ) |

Since the mutation frequency at the y locus is fairly high in M-5, we may interpret this to mean that the inversion may be part of the reason, but it is very difficult to explain why this is not the case for other loci.

Finally, reciprocal crosses were examined, that is, males of y, w, m, f, and Canton S strains were irradiated and mated to 0-1 ♀♀. The results were as follows:

| Strain | Dose | No. of tested females | No. of y mutants | Mutation freq. (%) |
|---|---|---|---|---|
| y, w, m, f | 3000r | 8582 | 12 | 0.140 |
| | 0r | 1947 | 2 | 0.103 |
| Canton-S | 3000r | 6116 | 11 | 0.180 |
| | 0r | 1910 | 2 | 0.105 |

In this case, there are little differences between irradiated groups and control groups in the two. Of course, irradiated groups always have higher frequencies than those of controls.

Kikkawa, H. An agar-gel electrophoretic study on amylase in D. virilis.

By using agar-gel electrophoresis improved by Ogita (DIS 37:142), two different types of amylase were found among various strains of D. virilis. They were named $Amy^F$ (amylase fast) and $Amy^S$ (amylase slow) respectively. The $F_1$ flies between the two types showed mixed bands of parent patterns on the zymogram.

Genetic and biochemical analyses gave the conclusion that $Amy^F$ and $Amy^S$ might be allelic with each other, and that they were located on very near the locus of scarlet gene (67.5) on the fifth chromosome. As shown in the previous issues (DIS 32:142; 34:89 and 37:94), the amylase gene in D. melanogaster is located on the right arm of the second chromosome. Based on the gene-analogy, it has been assumed that the fifth chromosome of D. virilis may be homologous to the right arm of the second chromosome of D. melanogaster (Chino, M., 1936, 1937, Jap. J. Genet., 12:187, 257; 13:100). Now the chromosome-homology in the two species has also been shown in the biochemical standpoint.

The hybrid between D. virilis ($Amy^S$) and D. novamexicana ($Amy^F$) gave both F and S bands on the zymogram, showing that each amylase gene produces its own product (amylase protein) even in the hybrid cytoplasm.

Rakha, F.A., and A.O. Tantawy, Genetic variance in natural populations of D. melanogaster and D. simulans.

Natural populations of D. melanogaster and D. simulans captured from Alexandria University Farm were used in the present experiments to study the genetic variance, i.e. heritability of wing length, thorax length, lifetime egg production and longevity of adult flies, as well as genetic and environmental correlations between any of two characters. Different progeny tests were carried out (mating at random) and the regression of offspring on mid-parent or the intrasire regression methods were used. The results are presented in Table I.

Table 1. Heritability (%) for the four different characters studied in D. melanogaster and D. simulans.

| Character | D. melanogaster | D. simulans |
|---|---|---|
| Wing Length (W) | 21.62 + 1.27 | 21.80 + 7.86 |
| Thorax Length (T) | 20.34 + 1.06 | 23.30 + 6.30 |
| Longevity (L) | 3.57 + 9.50 | 11.10 + 8.80 |
| Egg Prod. (G) | 11.29 + 12.7 | 14.60 + 10.10 |

The results indicate clearly that body size in both species shows higher heritability estimates than egg production and longevity. It is interesting to note that the characters with lower heritability estimates are associated with greater phenotypic variance. Fitness characters are affected more by environmental agencies rather than metric ones. Table 2 includes all possible correlations between the four different characters.

Table 2. Genetic ($r_G$), phenotypic ($r_P$) and environmental ($r_e$) correlations between different characters.

| Characters | D. melanogaster | | | D. simulans | | |
|---|---|---|---|---|---|---|
| | $r_G$ | $r_P$ | $r_e$ | $r_G$ | $r_P$ | $r_e$ |
| W - T | 91.10 | 57.75 | 49.04 | 53.05 | 58.40 | 60.15 |
| L | 34.30 | 21.97 | 22.60 | 16.40 | 21.43 | 11.95 |
| G | 0.74 | 24.70 | 30.59 | 0.86 | 21.89 | 24.84 |
| T - L | 35.62 | 33.15 | 34.41 | 32.40 | 11.90 | 8.24 |
| G | 1.30 | 50.55 | 60.97 | 0.92 | 25.08 | 30.81 |
| L - G | 1.35 | 75.68 | 83.83 | 2.26 | 40.35 | 45.92 |

The results of the genetic correlation between different characters indicate clearly that selection for either wing or thorax length should be accompanied by increasing or decreasing both characters. On the other hand, longevity may show change but to a lesser degree while egg production will not show any change in relation to wing or thorax length.

The path coefficient method will be applied to the data to demonstrate the relationship between different characters. The results will be published soon.

Kuroda, Y. In vitro cultivation of single cells from the embryonic blastoderm of D. melanogaster

An attempt was made to dissociate the embryonic blastoderm of D. melanogaster into single cells and to culture these single cells in synthetic medium. Newly laid eggs of an isogenic wild strain (Oregon-R) were collected and immersed in 3% sodium hypochloride solution for 6 minutes. The chorions were removed without apparent harm to the embryos. The embryos at the stage of blastoderm formation were selected under a binocular microscope. Trypsin (Difco), lipase (N. B. C.), lysozyme (N. B. C.), chymotrypsin (N. B. C.), pancreatin (Yasuei, Japan), proteinase (Nagase, Japan) and pectinase (Sigma) were tested for their ability to dissociate the blastoderm into single cells. None of these enzymes could break out the vitelline membrane of embryos.

After the dechorionated embryos were sterilized in 70% ethyl alcohol for 10 minutes, they were rinsed in sterile Carlson's physiological salt solution. The following

procedures for cultivation were carried out under sterile conditions. The embryos were placed on a slide glass and covered with a coverslip, then they were pressed slightly over the coverslip with a needle. The vitelline membranes of embryos were broken and the naked embryos and their fragments on the slide glass were collected in a test tube by letting flow with the culture medium. The embryos and fragments were dispersed into single cells by flushing them briskly through the tip of a fine pipette. The single cell suspension obtained as above was cultured in synthetic media in short test tubes at 25° C.

Various concentrations of lactalbumin hydrolyzate (N. B. C.) added in Carlson's medium were tested to obtain the optimum level of total amino acids in synthetic medium. 10,000 mg, 8,000 mg, 6,000 mg, 4,000 mg, 2,000 mg and 0 mg/100 ml of Carlson's medium were tested and the highest increase in the number of cell nuclei was obtained in 6,000 mg of lactalbumin hydrolyzate/100 ml of medium after 7 days of cultivation.

Various concentrations of yeast extract (Difco) were tested to obtain the optimum level of total vitamins in synthetic medium. 800 mg, 400 mg, 200 mg, 100 mg, 50 mg and 0 mg/100 ml of Carlson's medium with 6,000 mg of lactalbumin hydrolyzate were tested and the highest increase in the number of cell nuclei was obtained in 800 mg of yeast extract/100 ml of medium after 7 days of cultivation.

Various pH's of the media were tested. Among pH's 6.0, 6.2, 6.8, 7.4, 7.8 and 8.0 tested the highest increase in the number of cell nuclei was obtained at pH 7.8 of Carlson's medium containing 6,000 mg of lactalbumin hydrolyzate/100 ml of medium after 4 days of cultivation and at 7.4 after 7 days of cultivation.

In Carlson's medium containing 6,000 mg of lactalbumin hydrolyzate and 800 mg of yeast extract/100 ml of medium with pH 7.8 the number of cell nuclei increased four times after 4 days of cultivation at 25° C.

Nakao, Y., E. Yamaguchi and I. Machida

The effect of chronic irradiation on sex-linked lethal mutation frequencies in D. melanogaster.

The changes of lethal mutation frequencies after long term chronic irradiation were examined. 7 bottles which contain 17 pairs of OrK ♂♂ and M-5 ♀♀ were prepared. About two weeks later $F_1$ flies were collected and mixed among bottles, then 7 new bottles each of which contained 17 $F_1$ pairs were set up. When the $F_1$ emerged, wild ♂♂ were randomly picked out and these were mated to M-5 ♀♀ as the first crosses. This procedure was repeated as a routine operation for about a year. Every generation of this series was exposed to γ-rays from $Co^{60}$ (5 curie) at the dose rates of 4.3r/day for the first 7 months and 10.3r/day for the following 4 months. By this mating scheme, the induced lethals on the X chromosome were thrown away and only non lethal X chromosomes were retained throughout the generations. Similar crosses without irradiation were also kept as a control series. Both series were kept at 25° C.

When the total dose reaches about 2000r (30th generation), ♂♂ from both irradiated and control series were irradiated with 3000r of X-rays (X-ray apparatus operated at 200 KvP, 20 mA, with 0.5 mm Al + 0.5 mm Cu filter, dose rate about 90r/min), and sex-linked recessive lethal frequencies were examined by the M05 method. After 32 generations lethal mutation frequencies at the dose of 1500r, and after 34 generations, spontaneous mutation rates were tested for both series, respectively. The results are shown in the table.

| Generations | Series | Dose | No. of tested chromosomes | Lethals | Semi-l* | Mutation rate (%) |
|---|---|---|---|---|---|---|
| 30 | Irrad. | 3000 | 5211 | 297 | 31 | 5.70 (6.30) |
| 30 | Control | 3000 | 5428 | 251 | 30 | 4.62 (5.18) |
| 32 | Irrad. | 1500 | 5761 | 151 | 25 | 2.62 (3.06) |
| 32 | Control | 1500 | 5467 | 151 | 20 | 2.76 (3.13) |
| 34 | Irrad. | 0 | 6694 | 16 | 4 | 0.24 (0.30) |
| 34 | Control | 0 | 6678 | 12 | 6 | 0.18 (0.27) |

*: Semi-lethals

20 $F_1$ pairs were examined from each irradiated and control male.

From the table, it can be seen that the mutation frequencies in chronically irradiated series are higher than those in the control series, with the exception of those at 1500r. That is to say, contrary to expectation, the X chromosome acquires no resistance against the mutagenic effect of radiations. The chronic irradiation is still continued, and from the 50th generation attached X females were used instead of M-5 females to simplify the scheme.

Tsukamoto, M. Attempts to calculate more precise loci for genes with poor penetrance of characters.

The phenotype of some mutants of Drosophila, such as bo (bordeaux), gp (gap), ld (loboid), etc., unfortunately overlaps that of the wild alleles even under the best rearing conditions. Furthermore, the character of some mutants such as s (sable), ba (balloon), Dfd (Deformed), spa (sparkling), or sw (short-wing), Sp (Sternopleural), pys (polychatous), etc., is best at lower or higher temperatures. These mutants are considered by many investigators as less useful mutants in the recombination tests such as three-point method, and loci of these mutants on the linkage map are somewhat obscure because of their poor penetrance of the character.

In the usual calculating method, therefore, some fraction of the flies bearing such a mutant gene, hereafter designated x, may always be miscounted as wild type flies, and this gives an incorrect map distance for estimating the locus of the gene x.

To eliminate these unknown and unavoidable effects of poor penetrance in the usual calculating method, a new and more precise method has been derived. This method is based on the following assumptions: 1) that a certain portion, y, of genotypically x flies are always and equally miscounted as the wild phenotype, and hence only (1 - y) fraction of the x genotype flies can be recognized as the mutant phenotypically; and 2) that no specific interaction exists among the genes used or tested.

(I) The a-x-b arrangement

When the x mutant is assumed to be located between two good marker mutants, a and b, the following backcross system may be used to calculate the recombination values between a and x or x and b:

$$\frac{a\ x\ b}{a\ x\ b} \times \frac{+\ +\ +}{+\ +\ +}$$

$$\downarrow$$

$$F_1\left(\frac{a\ x\ b}{+\ +\ +}\right)\varphi \times \frac{a\ x\ b}{a\ x\ b}\sigma$$

From such a backcross system, therefore, the following eight phenotypes might be expected to occur in the backcross progeny:

| Phenotype | Observed Number | | Estimated Constitution |
|---|---|---|---|
| a x b | A | = | $n(1 - y)$ |
| a x + | B | = | $n\left(\frac{R_2}{100 - R_2}\right)(1 - y)$ |
| + x b | C | = | $n\left(\frac{R_1}{100 - R_1}\right)(1 - y)$ |
| + x + | D | = | $n\left(\frac{R_1}{100 - R_1}\right)\left(\frac{R_2}{100 - R_2}\right)(1 - y)$ |
| a + b | E | = | $n\left(\frac{R_1}{100 - R_1}\right)\left(\frac{R_2}{100 - R_2}\right) + ny$ |
| a + + | F | = | $n\left(\frac{R_1}{100 - R_1}\right) + n\left(\frac{2R_2}{100 - R_2}\right)y$ |
| + + b | G | = | $n\left(\frac{R_2}{100 - R_2}\right) + n\left(\frac{R_1}{100 - R_1}\right)y$ |
| + + + | H | = | $n + n\left(\frac{2R_1}{100 - R_1}\right)\left(\frac{R_2}{100 - R_2}\right)y$ |

where n is the basic number for the non-crossover wild type flies (+ + +), $R_1$ and $R_2$ are percent recombination values for the a-x and x-b regions, respectively.

From these equations, the recombination values

$$R_1 = \frac{100}{1 + \sqrt[4]{\frac{DE - Cf}{BG - AH}}}\ (\%) \ldots\ldots (1)$$

and

$$R_2 = \frac{100}{1 + \sqrt[4]{\frac{DE - BG}{CF - AH}}} (\%) \ldots\ldots (2)$$

are calculated where any influence of the poor penetrance of the x gene, 1 - y, and the unknown symbol n are eliminated.

(II) The x-a-b arrangement

When the x-a-b arrangement is assumed, the following equations will be estimated in the backcross progeny from the crossing system of

$$F_1(\frac{x\ a\ b}{+\ +\ +})♀ \quad X \quad \frac{x\ a\ b}{x\ a\ b}♂$$

| Phenotype | Observed Number | | Estimated Constitution |
|---|---|---|---|
| x a b | A | = | $n(1 - y)$ |
| x a + | B | = | $n(\frac{R_2}{100 - R_2})(1 - y)$ |
| x + b | C | = | $n(\frac{R_1}{100 - R_1})(\frac{R_2}{100 - R_2})(1 - y)$ |
| x + + | D | = | $n(\frac{R_1}{100 - R_1})(1 - y)$ |
| + a b | E | = | $n(\frac{R_1}{100 - R_1}) + ny$ |
| + a + | F | = | $n(\frac{R_1}{100 - R_1})(\frac{R_2}{100 - R_2}) + n(\frac{R_2}{100 - R_2})y$ |
| + + b | G | = | $n(\frac{R_2}{100 - R_2}) + n(\frac{R_1}{100 - R_1})(\frac{R_2}{100 - R_2})y$ |
| + + + | H | = | $n + n(\frac{R_1}{100 - R_1})y$ |

where $R_1$ and $R_2$ are percent recombination values for the x-a and a-b regions, respectively. Consequently, unknown symbols n and y are eliminated from the final equations:

$$R_1 = \frac{100}{1 + \sqrt[4]{\frac{ACFH}{BDEG}}} (\%) \ldots\ldots (3)$$

and

$$R_2 = \frac{100}{1 + \sqrt[4]{\frac{ABGH}{CDEF}}} (\%) \ldots\ldots (4)$$

When the mutant to be tested is dominant over the wild type, more possible combinations of crossing systems may of course be expected than in the case of a recessive. However, the recombination values, $R_1$ and $R_2$, are also calculated from the formulae (1) and (2) for the a-X-b arrangement or from the formulae (3) and (4) for the X-a-b arrangement, respectively.

It should be noted, as is obvious from these formulae, that the observed number of zero for any phenotype, especially such as double crossover classes, should not be included in the data. Most investigators report their linkage data in DIS without any description of the phenotypes, in a condensed form such as 0=670+703; 1=32+37; 2=92+83; 1,2=3+6; N=1626; $R_1$=4.8; $R_2$=11.3, because the condensed form was proposed by the editor of DIS-1 (1934) and DIS-9 (1938) issues. According to the viewpoint of the new calculating method described above, however, it is impossible to calculate the more precise map distance from such an inadequate or incomplete expression of linkage data. Therefore, I would like to propose that the complete data including the phenotypes should be reported in the future, even in the section of "linkage data" of DIS!

In addition to eliminating the influence of poor penetrance of the mutant character upon the recombination values, the influence of lower or higher viabilities of the mutant gene tested or marker genes used was also effectively arranged to be eliminated in these new formulae. (The procedure for eliminating the influence of viability of each mutant gene is based on an unpublished method by Hiroyoshi and Tsukamoto).

The principle of this method is effectively applicable not only to Drosophila but also to other organisms, and not only to morphological characters but also to various physiological and/or biochemical characters, such as insecticide resistance, radiation tolerance, enzyme activities, lethal or semi-lethal, etc.

Ogaki, M. and E. Tanaka Genetical analysis of resistance to gamma radiation in D. melanogaster.

Two or three days old flies from an inbred wild strain of Hikone-H, together with that of bw; st ss and six special synthetic strains of D. melanogaster were subjected to $Co^{60}$ gamma radiation in doses ranging from 80,000r to 180,000r at approximately 90,000r/hr. The flies were kept 40/vial at 24 ± 0.25°C. Survival and mortality rates were counted on seven days after irradiation. The interpolated $LD_{50}$ of Hikone-H is near 150,000r for females, 130,000r for males, and that of bw; st ss is about 120,000r for females, 100,000r for males. The $F_1$ generation of a reciprocal cross between these two strains showed almost the same resistability as that of the Hikone-H strain, thus the $LD_{50}$ of hybrid females was near 150,000r. It is clear that the radiation tolerance is dominant over susceptibility, and maternal or cytoplasmic effect is negligible. In order to analyse which chromosome is responsible for tolerance, six special synthetic strains, for instance, I(Hikone-H);bw; st ss, I;II(Hikone-H); st ss, I(Hikone-H); bw; III(Hikone-H) etc. were constructed. Comparison of resistability to radiation of these six stocks revealed that the resistance to gamma rays is mainly due to the 3rd chromosome.

According to the preliminary crossover data the major gene (or genes) locates on the right hand beyond st(3-44.0), but the precise locus is not decided yet. It is noticeable that the degree of resistance varies strictly according to the age of the flies, so we will limit more precisely the time of egg laying and the age of the flies in subsequent experiments.

Sheldon, B. L. Specificity of a canalization system.

The genotype selected for a degree of canalization in a scute stock (Rendel and Sheldon 1960) has so far been tested by appropriate backcrossing, on three other mutants, ci, cv and ey. Preliminary analyses indicate that it also produces a degree of canalization in ci and cv flies but has the opposite effect on ey flies, which become more variable in the presence of the low variance scute background. It would appear that the genetic system controlling canalization of scutellars has a more general sphere of influence extending to all or part of the rest of the thorax including the development of the wings. Other studies indicate that the low variance scute genotype is polygenic, chromosomes I, II and III each contributing, with some interaction between II and III.

References: Rendel, J.M. and Sheldon, B.L., 1960. Aust.J.biol.Sci., 13(1): 36-47.

Sheldon, B. L. and Rendel, J. M. Canalization changes in homozygous lines.

A set of 18 different but highly homozygous lines were derived by an isogenizing procedure from an Oregon RC stock. About half of them were more strongly canalized for scutellar bristle number than the base population, and the other half were less strongly canalized. It is concluded that heterozygosity is not essential in the genetic system controlling canalization. This is supported by similar results obtained in a series of inbred lines derived by brother-sister mating. A scute population, weakly canalized at 2 bristles by artificial selection for low variability (Rendel and Sheldon, 1960) has become still less variable after a total of over 100 generations of selection. A similar series of homozygous lines to the above were derived from this low variance (L.V.) scute population. Canalization was reduced in most of these lines but to a variable extent. In all lines some canalization remained. While the results of this series indicate that artificially selected canalization has involved dominance, at least in part, again there is no support for the notion of heterozygosity being essential for developmental stability. All the lines referred to here are still available if anyone wishes to use them.

References: Rendel, J.M. and Sheldon, B.L., 1960. Aust.J.biol.Sci., 13(1): 36-47.

Koch, Elizabeth A. and R. C. King

The ultrastructure of fes ovarian tumors.

Hereditary ovarian tumors occur in female Drosophila melanogaster homozygous for the recessive gene females sterile (fes, 2-5±). Each ovariole usually contains 3 or 4 chambers each of which is surrounded by an envelope of follicle cells. Most chambers are filled with hundreds of tumor cells, but some may also contain cells which on the basis of their nuclear morphology resemble normal nurse cells (3, 5).

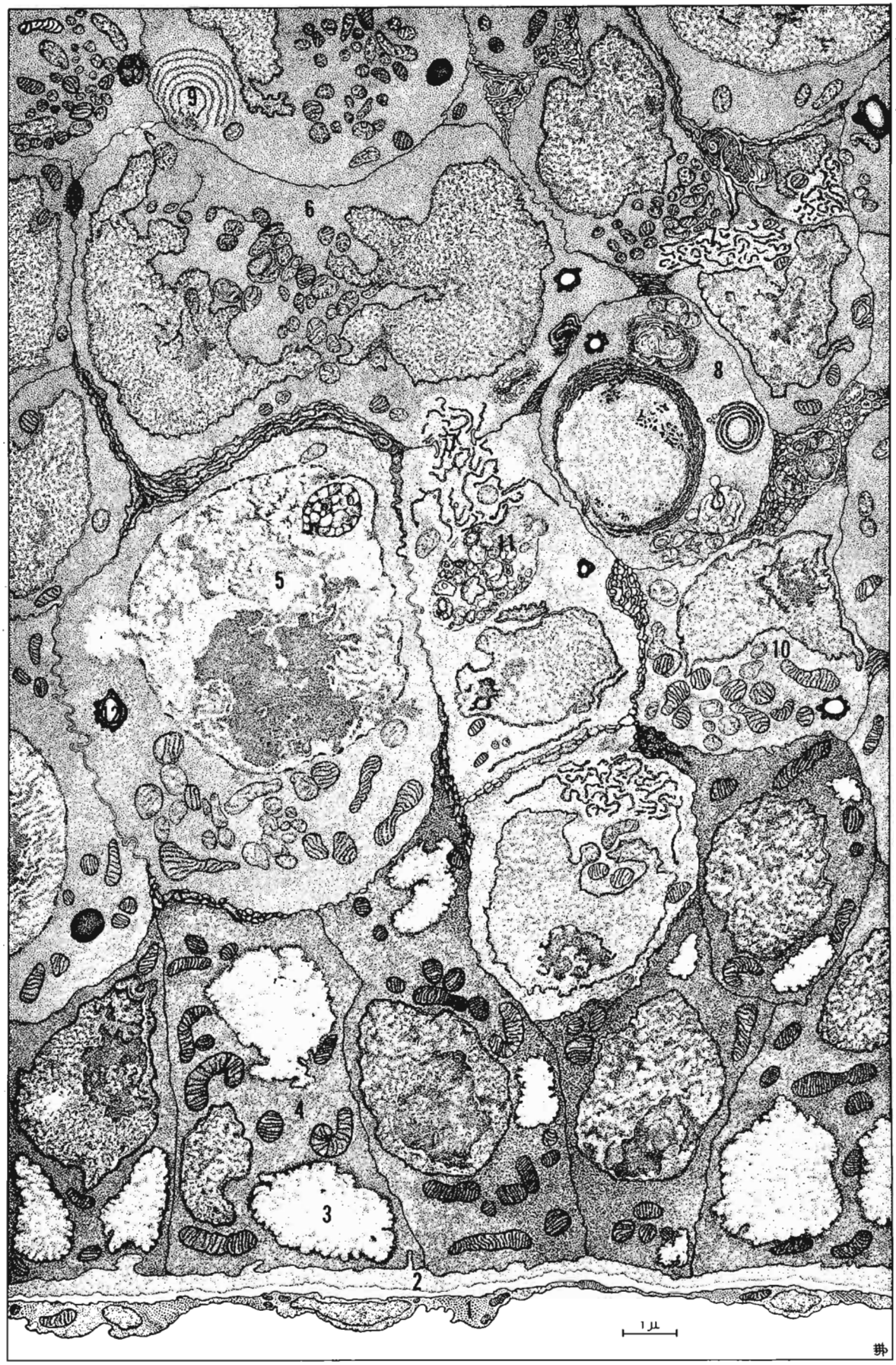
The drawing, which is a composite made from about 50 electron micrographs, shows a portion of a tumorous chamber. The follicle cells contain large carbohydrate deposits which are not seen in normal follicle cells. We have shown previously that the vitelline membrane (which contains carbohydrate) is a product of the follicle cells (2). It may be that in those fes follicle cells that are prevented from forming vitelline membrane precursors of the carbohydrate component accumulate as the deposits mentioned above. The follicle cell cytoplasm is usually more dense than that of the tumor cells. This difference in density is probably a function of the concentration of the ribosomes.

We have never seen any particles in either the nucleus or the cytoplasm of the tumor cells which resemble viruses. The cytoplasm of tumor cells lacks endoplasmic reticulum and in this respect resembles normal, rapidly dividing cells such as those observed in the germarium. Quite an elaborate endoplasmic reticulum normally develops in follicle cells at the time of the secretion of the vitelline membrane and chorion (2). Among the population of mitochondria in the tumor cells one sees bodies of similar dimensions that do not show clear-cut cristae. Such bodies may be mitochondria that do not show cristae because of the plane in which they are sectioned, or they may be A bodies. A bodies, which are normally found in the cells of the germarium and in the cells of normal chambers, are the size of mitochondria but do not show cristae (4).

Certain cells in the chamber increase markedly in volume and develop large, banded, polytene chromosomes. These cells are considered nurse-like in their behavior since the nuclei are undergoing endomitotic duplication of their DNA. The nuclei of normal nurse cells contain multicompartmented bodies (4). Multicompartmented bodies have also been seen in relatively small nuclei of certain cells in these tumorous chambers. Thus these nuclei are nurse-like at the ultrastructural level.

The 16 cells in a normal chamber are connected by 15 ring canals arranged in a characteristic pattern (1). These ring canals are believed to be permanent structures which arise at the time of cell division when the centripetal advance of the cleavage furrow is stopped by the spindle fibers. Since cells which are connected by ring canals are daughters, an analysis of the canals within a chamber gives information as to the lineage of the cells. Ring canals similar in structure to those found in + ovaries connect adjacent tumor cells or adjacent nurse-like cells in fes chambers. However, in chambers containing both tumor and nurse-like cells, ring canals have never been seen to connect cells of these two types. This observation indicates that two different cell lineages are represented within a single chamber. A tumor cell is often seen to be connected by ring canals to two or three other tumor cells, and we therefore conclude that some canals are stable structures which can persist for at least two additional mitotic cycles. In a normal chamber a system of persistent ring canals is characteristic of the nurse cell-oocyte complex. The demonstration of relatively persistent canals linking tumor cells suggests that the tumor cells are of germ cell origin.

A drawing showing a portion of a tumorous chamber as it appears under the electron microscope. 1, epithelial sheath. Note the parallel clusters of myofilaments in the cytoplasm, the nuclei, and mitochondria. 2, the tunica propria. 3, carbohydrate deposit. 4, follicle cell containing mitochondria and a nucleus. Note that the cytoplasm of the follicle cells is denser than that of the tumor cells. 5, the nucleus of a nurse-like cell containing a multicompartmented body. 6, tumor cell at telophase. Note that this cell is connected by a ring canal to an adjacent tumor cell. 7, ring canal between two tumor cells. Note the remnants of the mitotic spindle lying within the canal. 8, degenerating cell. 9, annulate lamellae. 10, tumor cell. Note the irregular shape of the nucleus. 11, cluster of vacuoles interpreted as phagocytized cellular debris. 12, lipid droplet with extracted center.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King, R. C. Trends towards ovoviviparity in Drosophila.

In the DIS 32 Sang and I reported experiments in which mated Oregon-S, Crianlarach-6, and hybrid females were compared with respect to their ability to synthesize yolk from larval reserves when reared upon an inadequate adult diet. Ore-S females were found to store the majority of synthesized eggs in their ovarioles; whereas C-6 and hybrid females laid most of the eggs synthesized at once. In the case of Ore-S one ovariole (out of 2300) was found to contain an active, first instar larva (which subsequently developed into a fertile male). Obviously the egg from which this larva arose was fertilized in the ovariole.

In the DIS 37 Gregg and Day reported experiments which compared egg retention in adult, virgin females (of differing genotypes) all of which were fed an adequate diet. The females came from a variety of wild type stocks as well as from interstrain crosses. Females from the strain characterized by the lowest egg retention averaged one mature egg per ovariole; whereas an average of three eggs were observed in each ovariole of females from the strain showing the greatest tendency to store eggs.

Thus in Drosophila there exist genotypes which facilitate egg storage, and ovariolar fertilization does take place on rare occasions. Egg storage and ovariolar fertilization are departures from oviparity which must accompany the transition to ovoviviparity, a method of reproduction common among muscoid flies ( see H. R. Hagan's Embryology of the Viviparous Insects). Studies like those cited above can give some indication of the genotypes available for selection during the evolution of ovoviviparity.

Stalker, H. D. and H. L. Carson A very serious parasite of laboratory Drosophila. Second report.

In 1956 workers in this laboratory found what appeared to be a microsporidian infection of D. parthenogenetica, (DIS 31:170). The spores of this parasite were strikingly consistentin size and shape, and covered by a think, rigid capsule. They were ovoid and 4 - 5 micra in length. The spores were readily seen in heavily infected individuals dissected in saline and were found to be associated with the reproductive organs, fat bodies and gut. Heavily infected individuals were sterilized, and frequently killed. The parasite was transmitted by feeding on feces and bodies of infected individuals, by mating, and apparently through eggs. Infection could result in death of larvae and pupae as well as adults. It was proven that the parasite was easily transferred to species such as D. melanogaster, D. robusta and D. melanica. The infected strain of D. parthenogenetica was discarded, and the infective agent seemed to disappear from the laboratory.

When the parasite was discovered in D. parthenogenetica, Dr. M. Wheeler examined some of the stocks in the University of Texas species collection, and while he did not find the type of parasite we had seen in St. Louis, he did find (in a species related to D. parthenogenetica) what appeared to be a different type of microsporidian, with a much larger banana-shaped spore, about 20 micra in length.

In 1958 a mass collection of D. melanogaster from the St. Louis area produced an abundant $F_1$, then the $F_1$ individuals developed a high degree of sterility and mortality. The $F_1$ were shown to have the banana-shaped spores. The stock was discarded.

In the fall of 1963 both the large and small types of spores were discovered in stocks in the St. Louis laboratory. The small spores were found in two of four strains of Buonostoma (Scaptomyza) originally collected in Hawaii, cultured there for a number of months, and then sent to St. Louis. The two strains known to be infected were rather quickly eliminated through death and sterility of adults, and a very high degree of mortality in young larvae. The large (banana-shaped) spores came from three mass collections of D. melanogaster from three localities in the St. Louis area (November 1963). The $F_1$ from the wild-caught flies appeared healthy, then within a few days the young $F_2$ larvae showed 100% mortality, followed by complete sterility of all adults, and their death at about 2 weeks of age.

We know of no good method for eliminating these parasites from infected stocks. However, we do have the impression that they have appeared in quantity following crowding of adults, and it is possible that spreading out lightly infected stocks, and carrying them through a rapid change schedule might be helpful. We also have reason to believe that change from low to high temperature may be associated with build-up of the parasite, and thus low temperature might be helpful in preventing infection or eliminating it when established.

Enloe, J. Alton, and C. A. Norman.

Egg collector, fly mixer, and techniques developed for studies on melanotic tumor formation in Drosophila melanogaster.

Introduction: Metallic ion chromophores may be involved in the formation of melanin pigments. Much basic research on the pigment system involved in malignant melanoma has been done with D. melanogaster. Kanehisa[6] and Kuroda et al.[7] have suggested relationships between some trace metal patterns and melanotic tumor formation in Drosophila. Studies of melanotic tumor formation in D. melanogaster were initiated at this institution to advance our investigations on the trace metal patterns in human malignant melanoma[10].

Because of difficulties in obtaining sufficient quantities of biological materials for trace metal investigations on biochemical systems of interest, only one Drosophila strain was used initially for intensive study. A suppressor-erupt, suppressor-tumor strain* (Su-er tu bw; st er su-tu) was chosen for these studies, since tumor induction could be controlled. This strain responds to x-radiation and high oxygen tensions by producing melanotic masses[4,9]. The hypothesis was that some trace metal patterns might be correlated with the etiology and development of melanotic tumors[3] created in Drosophila by x-radiation and/or chemical agents.

The purpose of this paper is to describe the development and use of an egg collector, a fly mixer, and some effective techniques developed in studies of melanotic tumor formation in Drosophila melanogaster.

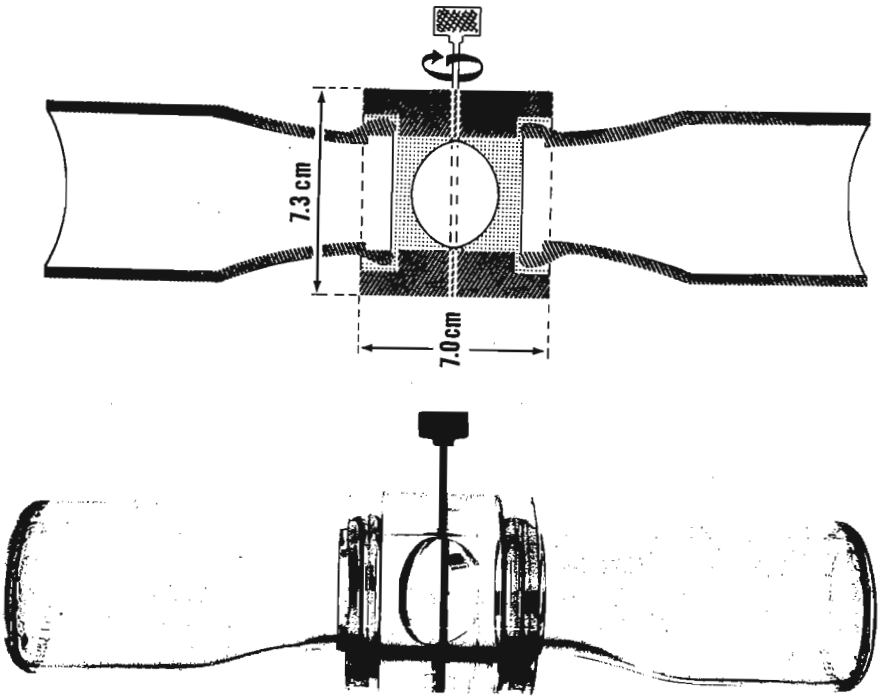
Drosophila egg collector: Egg Collecting Methods Described in the Literature: Sonnenblick and Spencer[2] described a number of techniques for satisfactory collection of Drosophila eggs. Materials such as the inner surface of banana skins, yeast-food spoons, and bottle caps plated with agar were suggested as media for egg deposition. Allen[1] has collected satisfactory numbers of eggs for various experiments by an adaptation of the bottle cap technique for use on ordinary chemical reagent bottles.

Development of a Drosophila Egg Collector: Although the studies were limited to a single Drosophila strain, improved techniques for collection of large quantities of eggs had to be developed in order to attain the objectives of the project. The applicability of the Drosophila Egg Collector which we developed was suggested by Plaine's technique[8]. This technique[8] utilizes nongrowing brewer's yeast as the sole medium for growing Drosophila larvae and uses Buchner funnels and filter paper for sterilization and treatment of Drosophila eggs[9]. (In metabolic studies, it is necessary to use chemically-defined media, such as the complete media developed by Sang[11] and Hinton[5] or the minimal medium developed by Allen[1].)

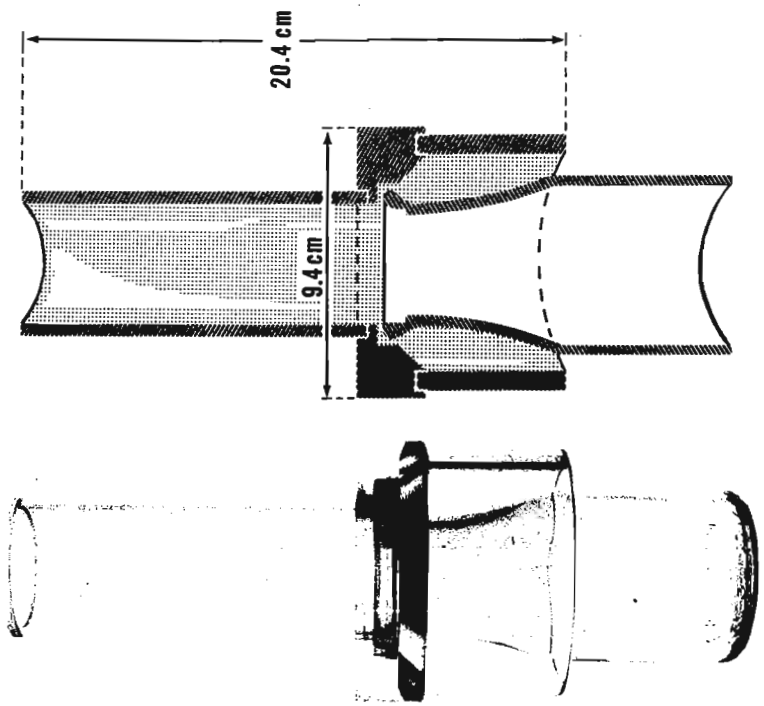
Plaine[8] showed that larvae could be grown on filter paper in Petri dishes with only yeast as food. Our collector was designed to allow egg deposition directly into such containers. This device eliminated, (1) the need for agar bases which must be cooled after pouring; (2) the insufficient surface available to laying adults in ordinary collecting bottles; and (3) the time required to move eggs from agar surfaces to larval growth media. This apparatus, developed for collection of large quantities of Drosophila eggs directly on filter paper in 9 cm. Buchner funnels or Petri dishes, is shown in Fig. 1.

The egg collector was constructed in the Physics Department of this institution from standard lucite stocks. The apparatus consists of (1) a barrel 12.4 cm. in length, 5 cm. in diameter, and closed at the top with a 3 mm. lucite plate; (2) a barrel 6.9 cm. in length, 8.9 cm. in diameter, with 6 mm. walls; (3) a piece of lucite stock 9.4 cm. in diameter and 2.5 cm. deep. The two barrels are joined together through a bore in the piece of lucite stock. The piece of stock is grooved to fit closely around the lip of a pint bottle and is beveled inside so that adult Drosophila slide into the smaller barrel when the collector is inverted. An egg collector of these dimensions is spacious enough for flies from 3 heavily populated pint bottles. Eggs can be collected at predetermined intervals by positioning the larger barrel of the collector into the 9 cm. Petri dish.

Use of Egg Collector: Adult flies from 3 bottles containing regular banana medium were collected daily at 1:00 p.m. in an empty pint bottle where they had no nourishment for one hour. This preliminary gathering, although not essential, seemed to stimulate the laying of greater numbers of eggs. With the suppressor-erupt, suppressor-tumor strain, it was advantageous to pregather the sticky adults. This pregathering protected the egg collector from extremely rough handling, since an unusual amount of jarring was needed to remove sticky adults kept in a particular bottle for 2 or more days.




DROSOPHILA MIXER




DROSOPHILA EGG COLLECTOR

During the 1-hour fasting period, sterile Petri dishes containing 3 sheets of 9 cm. Whatman No. 1 filter paper were moistened with 3.5 ml. of 2% acetic acid in distilled water. A cup (4.5 mm. in diameter and 1.5 mm. deep) filled with dried brewer's yeast was emptied in the center of the moistened filter paper. The yeast absorbed water rapidly and adhered to the paper even when the Petri dish was inverted. With this preparation, there was little hazard for adult flies as they moved about in the egg collector.

At 2:00 p.m., a bottleful of adults was transferred to each of the egg collectors, which were then inverted. After the removal of its lid, a Petri dish was quickly positioned on the egg collector to replace the fly bottle. The inverted egg collector was placed in a dark cabinet. At 8:00 p.m., the adults were transferred to 2 or 3 bottles (according to losses sustained) of new food. After the removal of all dead adults, a lid was replaced on the Petri dish which contained the eggs.

Since eggs can be laid throughout the collector, it must be washed thoroughly after each use. The collector must be sterilized if different flies will be used and there will be danger of contamination. If many adults are in the egg collector, excessive moisture will accumulate in the top of it after a few hours. Great losses can be expected over longer collection periods. Good yields have been obtained with our egg collector when it is used as we have described. Optimum yields were obtained when adults were transferred to new media 1 day before they were placed in the egg collector. Excellent yields can also be obtained on small pads of 3% agar poured into the center of 9 cm. Petri dishes if one wishes to gather the eggs for particular experiments.

Drosophila Mixer: The device shown in Fig. 2 has been useful for mixing new and old adults and for equating adult populations in different bottles. The apparatus consists of a lucite cylinder 7.0 cm. in length and 7.3 cm. in diameter. Holes (1.7 cm. deep) were bored on each end of the cylinder to accommodate 2 pint bottles. A lucite plate of 5 mm. thickness was fitted in the 4.0 cm. bore of the fly mixer and was positioned by a 9.0 cm. pin attached to a knurled knob. When the passage is open, adult flies can be mixed and divided between the 2 bottles as desired. Rotation of the knurled knob closes the passage between bottles and allows transfer without loss of adult flies. This apparatus was used routinely in these studies to insure the homogeneity of populations in different bottles.

Techniques: Larval Growth: When the larvae began to move about in the Petri dishes, 5 small drops of brewer's yeast (.55 gm. of yeast per ml. of distilled water) were placed at random locations on the filter paper. Since the yeast mixture interferes with hatching if it is placed directly on the eggs, the drops were not placed on large masses of eggs. These small drops of moistened yeast provide adequate nourishment and moisture, without excesses which take nutrients from the yeast, for even the most heavily populated plates for 1 day. The drops also attract the young larvae so that large amounts (usually 5 heaping spoonfuls) of the mixture can be added to vacant areas 1 day later. The second addition of food was sufficient for the larvae until pupation.

Harvest: Grown at approximately 25°C, 4-day old larvae were washed from the yeast with several changes of distilled water. Since the larvae sank in this suspension, only very fine yeast particles could be separated from them. A saturated solution, containing reagent grade NaCl was added to the mixture of yeast and larvae. In the saturated NaCl solutions, the larvae floated and were separated rapidly from the large yeast masses. Yeast particles slightly heavier than the larvae usually could be removed by slight dilutions of the NaCl solutions in which the larvae would still float. The floating larvae were poured into another container, which was immediately filled with an equal volume of salt solution. The process was repeated and the larvae were decanted. The third change of salt solution was diluted at once with deionized water until the larvae sank. The larvae were then washed 6 times with deionized water. In contact with the salt for less than 3 minutes, the larvae were alive after the final washing in deionized water.

Summary: Included in this paper are illustrations and descriptions of an egg collector and a fly mixer developed for and found useful in studies on melanotic tumor induction in Drosophila melanogaster. Techniques which were used in the growth and harvest of larval material for trace metal analysis are also described. Larvae that are grown and cleaned by these techniques are being used in x-ray studies on melanotic tumor induction. The results of these studies will be reported in future communications.

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Seecof, R. L. An apparatus for anaesthetizing Drosophila with $CO_2$.

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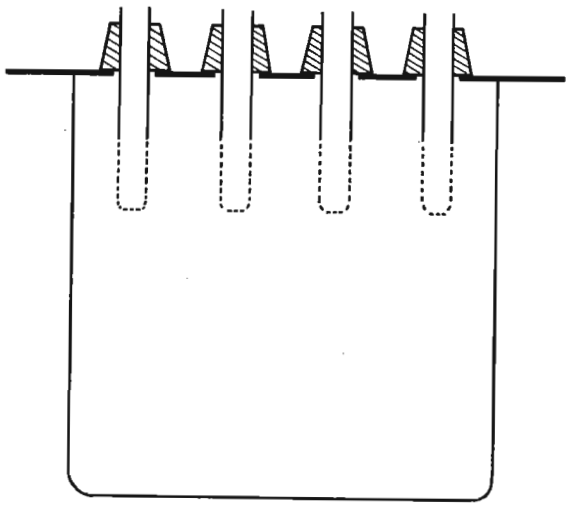
Drosophila are routinely tested for $CO_2$ sensitivity by exposure to pure $CO_2$ for 15 minutes at 14° C. This procedure, calling for compressed gas and appropriate apparatus, is described in: An alternative procedure, utilizing dry ice, has proved to be quick and simple and is described below.



A cross section of the dry ice anaesthetizer is shown in the accompanying figure. The container is a thin-walled stainless steel pot, 6 in. in diameter and 7 in. deep. The lid is Plexiglas (9 x 9 x 3/16 in.), pierced by 16 circular holes (diameter 5/8 in.) arranged in 4 rows above the container mouth. There are 16 perforated glass or plastic tubes (13 x 100 mm) suspended into the chamber by means of number 4 rubber stoppers. The tubes project 60 mm. below the lid.

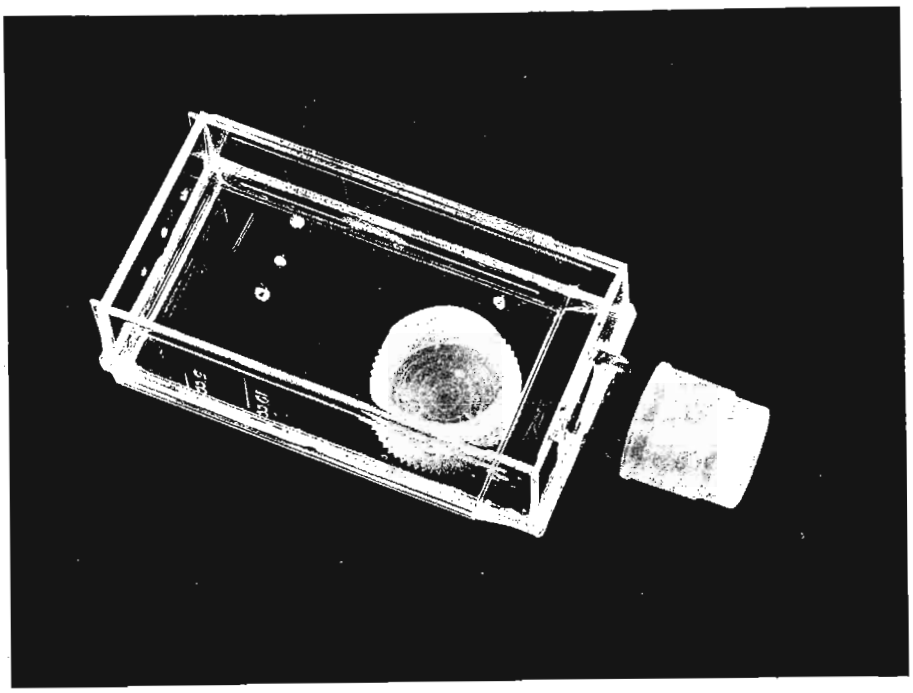
About 300 grams of crushed dry ice are placed in the container, the lid and tubes set in place, and a thermometer placed in one of the tubes. The evaporation of the dry ice will cool the tubes to 12°C. in about 5 minutes. After this time, flies may be shaken into the tubes through a funnel and kept in the tubes during the standard 15 minute test for $CO_2$ sensitivity. The tubes can then be withdrawn from the container, emptied of flies, and then returned to the container for testing another batch of flies. The slowly-evaporating dry ice will keep the tubes below 17° C. for about an hour.

The $CO_2$ content of the atmosphere within the container has not been determined. However, the test distinguishes $CO_2$-sensitive from $CO_2$ resistant flies unequivocally between 12° and 17° C. The rate of evaporation of the dry ice depends upon the size and composition of the container and the temperature and humidity in the room. Therefore, a standard quantity of dry ice to be used must be established by trial and error. This quantity will serve for all future determinations unless the room conditions change drastically.

Walker, G. W. R., and J. F. Dietrich. A miniature population cage.

A population of the order of 50 flies may be reared for extended periods by the inexpensive and quickly constructed population cage shown below. It consists of a 25-ml polystyrous disposable tissue culture flask (catalogue no. 3004, Falcon Plastic, 5500 W. 83rd St., Los Angeles 45, Calif.). A circular hole is readily punched in the bottom with a moderately heated cork-borer (#9, 17 mm. outside diamter), for insertion of the food container, a plastic cap from a 2-dram Opticlear shell vial (catalogue no. 60930-51902; Kimble Glass Co., Toledo 1, Ohio). Ventilation holes are also readily punched in the wall with a heated dissecting needle. Etherization is quickly achieved by replacing the food container with an etherizing container.

Population Cage: As Seen from Above



Rinehart, R. R., and R. M. Valencia. A method for pair mating Drosophila without etherization.

A Kimax No. 58 glass funnel, 6 cm. in diamter, a piece of clear plastic 6 cm. in diameter containing a 2 cm. hole in its center, and several feet of 1/4" x 1/8" Tygon clear flexible vinyl tubing were used. The plastic top was permanently fixed to the funnel, and the flies were introduced unetherized through the small hole which could be plugged with a polyurethane foam plug. The Tygon tubing was attached to the stem of the funnel, which was placed in a ring stand. The flies could be manipulated easily in the tubing since they could be seen at all times. One investigator handled the tubing containing the flies and was able to separate the Drosophila and drop them individually into vials held by a second person. Males were placed in each vial this way and, by repeating the process, females were added. Very few of the males escaped during the subsequent introduction of the females. With a minimum of practice we were able to make approximately 120 pair matings per hour. The system also can be used to separate males and females without etherization.

Fuscaldo, K. E., and A. Tambornino. The resolution of complex protein extracts of D. melanogaster by means of disc electophoresis.

Crude extracts of D. melanogaster have been subjected to electrophoresis in a polyacrylamide gel. A high degree of resolution of the proteins into clearly defined bands has been obtained. The apparatus and materials for this technique may be obtained from Canalco (N.Y.) under the trade name Disc Electrophoresis.

The method, originally developed by Ornstein (1961), permits the separation of large molecules by passage through a polyacrylamide gel of a specified pore size. A standardized procedure has been developed in this laboratory in which reproducible patterns of from twelve to sixteen bands have been obtained.

Flies were collected, lyophilized and homogenized in buffered saline (2% in 0.85% NaCl buffered at pH 7.4 with 0.5 M phosphate). The homogenate was centrifuged at 3000 X g 20 minutes at 4°C. The resulting extract was concentrated 10 fold by dialysis, lyophilization and resuspension in 1/10 the original volume of buffered saline. A Lowry protein determination on this material indicated a protein concentration of about 5.4 mg/ml.

Approximately 400γ of protein (0.037 ml of the crude extract) was suspended in 0.037 ml of water and 0.075 ml of the sample gel. A gel with a pore size of 50° A was prepared. This get permits the separation of proteins having a molecular weight of between 30,000 and 300,000. A spacer gel was applied over the polymerized lower gel. The gel containing the sample was then carefully suspended over the spacer gel and both layers were polymerized by U.V. As the proteins descended through the spacer gel, well defined discs were formed at the interphase between the gel layers.

Electrophoresis was carried out in Tris buffer, pH 8.4 at 5 milliamps for 35 minutes. The gel was stained in 1.0% buffalo black in 7.5% acetic acid and destained by running the gel column at 10 milliamps in 15% acetic acid.

Drosophila proteins of various genotypes may thus be analyzed. With a well standardized procedure, protein bands of particular interest may be isolated and further characterized.

Sang, J. H. and B. Burnet

The importance of agar.

Because Japanese girl divers now find more congenial employment making transistors, than in collecting seaweed, supplies of Kobe No. 1 agar are now only rarely available in the U.K. Mediterranean and New Zealand agars have taken their place, sometimes with disastrous results even when used to make corn-meal, molasses media. Reports show that Drosophila females may then lay inviable eggs and, in one instance, that pupae of many selected lines fail to eclose. Our own experience is summarised below.

Since agars can be made from different seaweeds (often a mixture of species) and are subject to different kinds of processing, one might expect this natural product to be very variable. The following inorganic analyses show that this is indeed so.

| Agar | % Ash | $Ca^{++}$ | $Mg^{++}$ | $Na^{+}$ | $K^{+}$ | $Fe^{+++}$ | $SO_4^{++}$ |
|---|---|---|---|---|---|---|---|
| Oxoid Kobe No. 1 | 3.9 | 0.34 | 3.8 | 2.5 | 0.9 | 0.03 | 16.9 |
| " No. 3 | 3.6 | 1.43 | 0.3 | 3.7 | 0.7 | 0.15 | 17.6 |
| " Ionagar No. 1 | 1.4 | 1.88 | 4.0± | 6.2 | 0.8 | 0.07 | - |
| Davis New Zealand | 1.6 | 2.75 | 3.2 | 4.0 | 1.5 | 0.10 | 7.1 |

All values in mg per gm agar.

Tests of different agars using defined media (Sang, 1956) showed that available samples of Davis and Difco "Bactoagar" would not support larval growth. Frequency of melanotic tumors also proved a sensitive indicator of agar differences, as follows:

| | tu bw; $+^{se-tu}$ | tu bw; st su-tu |
|---|---|---|
| Live yeast | 10 % tumors | 95 - 100 % tumors |
| Kobe No. 1 | 39 | 10 |
| Oxoid No. 3 | 61 | 69 |
| Ionagar | 17 | 10 |
| Davis | Toxic | Toxic |

All larvae cultured on Medium C of Sang (1956) J. Exp. Biol. 33.

There is as yet no explanation as to why some agars are toxic or why the two genotypes above respond differently to the various agars. Attention is directed to the problem since it seems to have affected many studies in the U.K., and because we take agar so much for granted that we tend to ignore its importance in Drosophila work.

Seiger, M. B. An inexpensive microbalance with high sensitivity for rapid serial weighings.

Accurate body weights of individual desiccated D. melanogaster males were required for a series of experiments. The weight of each fly was determined by the deflection of the tip of a delicate quartz thread housed in a plastic body (see Figure next page). Quartz has a constant Young's modulus. Consequently, for each increase of a standard increment of weight, the quartz thread is deflected an exact amount. The apparatus was calibrated by using standard microweights of 0.1 mg, 0.25 mg and 0.5 mg.

The balance was built of lucite sheeting; 3/8" for the top, back and base, 1/8" for the other sides. The mounts for the gnurled adjusting nobs and the fibreboard chuck were made from scrap lucite stock. Several quartz threads, attached to the quartz rods from which they had been drawn, were provided by a glassblower. These threads were tested until one was found whose length and deflection were suitable. This was put into the plastic mounted chuck and fastened in the case with a metal pivot rod through the case walls and the mount. A pan was attached near the tip of the thread with ethyl acetate cement thinned with acetone. The pan was composed of a short length of a human hair glued to a small circle cut from the thinnest available cigarette paper.

A photograph was taken of a reticle available in the laboratory. The negative was projected by an enlarger so that the size of ten divisions on the reticle was equal to the deflection of the tip of the quartz thread for a 0.1 mg weight increment. The photograph of the reticle was made on a 3" x 4" glass slide and cut to fit the plastic housing. A mirrored photographic cover glass, cut to the same size, was attached to the back of the reticle as an aid for correcting parallax.

The balance was leveled by the use of a small spirit level. The tip of the quartz thread was zeroed with the adjusting screws. To weigh a fly, the door of the housing is opened and a fly taken from the desiccator with a pair of jeweler's forceps and placed on the pan. The door is closed and a reading taken with a magnifying glass as an aid to accuracy. The door is then opened and the fly removed. The process for a single weighing takes 10-15 seconds.

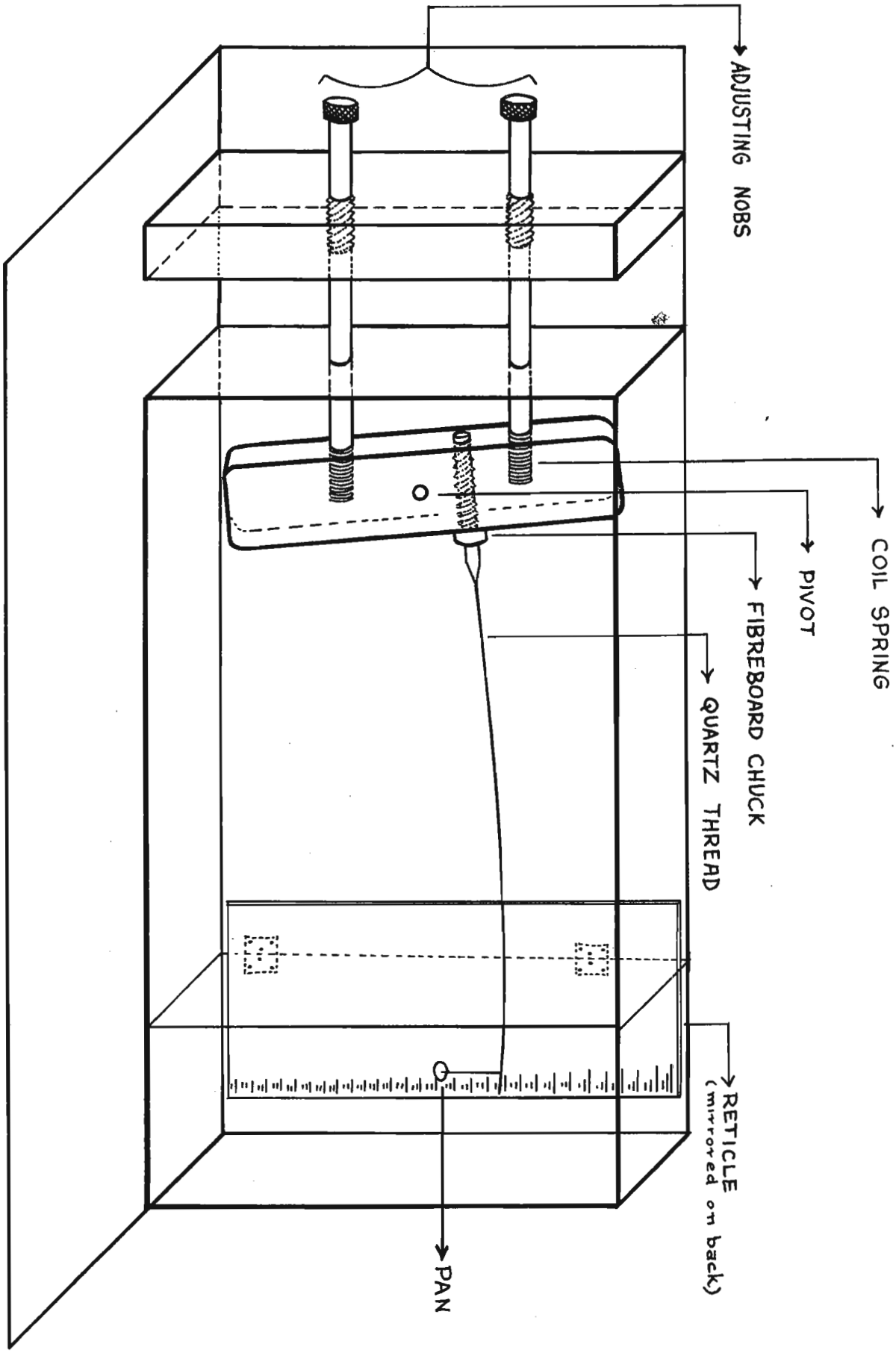
During the course of 1100 weighings the scale was tested periodically by reweighing the standard microweights and by comparing the weights of samples obtained on this device and a sensitive Sartorius microbalance. Discrepancies were never more than 2%.

Seiger, M. B. An improved method of staining minute tissues.

Often the most time consuming and painstaking part of staining small bits of tissue is changing the tissue from one solution to the next. This is most often accomplished by pipetting off or decanting the liquid from the tissue and then adding the next liquid, or by using a pipette to transfer the tissue from one solution to the next. These techniques sometimes lead to the loss of tissue while decanting or to the damage of tissue due to excessive handling while transferring from one solution to another.

While staining ovaries and ring glands for histological study it was noted that the number of tissues from different strains which could be stained in a given time and the precision of the staining technique was limited by our handling methods. An apparatus was devised to effectively eliminate loss and damage of the tissue and to facilitate the staining procedure. A hole was bored through a rubber stopper to contain a Gooch type Pyrex low form crucible with fritted glass disc. A Neoprene filter adapter can be used in place of a rubber stopper. The stopper with crucible was inserted into the neck of a filter flask which, in turn, was connected to a filter pump attached to a water faucet. The tissue was put into the crucible with an appropriate solution for a given time. At the end of that time, the filter pump was turned on until the fluid was aspirated from the crucible. The next fluid was added and the process repeated to the end of the staining regimen. When the filter pump was off, the fluid in the crucible barely filtered through the fritted disc. Changing fluids took a matter of seconds and there was neither loss of nor damage to the tissue. Several crucibles were used concomitantly to stain a series of different tissues.

The crucible (e.g. Pyrex #32960 at $3.67 each) had a capacity of 8 ml and the coarse (C) porosity of fritted disc was used. The coarse fritted disc has a nominal maximum pore size of 40-60 microns. The disc has remained white after the staining program and the stained tissue, easily seen against the white background, was transferred with a micropipette to a slide for mounting.


ADJUSTING NOBS
COIL SPRING
PIVOT
FIBREBOARD CHUCK
QUARTZ THREAD
RETICLE
(mirrored on back)
PAN

Irwin H. Herskowitz, Editor

D. = Drosophila D.m. = Drosophila melanogaster

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Late Additions:

Rasmuson, B., and Rasmuson, M. 1961. Diversity of interaction properties in eight back mutations from the mutant $w^a$ in D.m. Hereditas, 47:619-630.

Rasmuson, M. 1961. Unequal crossing over in the Bar region of D.m. II. Influence of temperature, X-rays and EDTA. Hereditas, 47: 357-378.

1962. Analysis of quantitative characters in D. (Abstr.) Hereditas, 48:540.

Daniel Marien is spending a sabbatical year in Genetical Institute, Haren (Gr.), Netherlands on an N.S.F. fellowship.

D. R. Wolstenholme is at the Genetical Institute, Haren, Netherlands for a year as a visiting lecturer.

W. D. Kaplan, Ph. D., Department of Biology, City of Hope Medical Center, Duarte, California is spending a sabbatical year in the laboratory of Per Oftedal, Norsk Hydro's Institute for Cancer Research, in Oslo, Norway.

J. A. Thomson of the Department of Zoology, University of Melbourne, is spending nine months from September, 1963, at the Department of Biology, Yale University.

R. Peter Kernaghan, formerly of the Department of Genetics, The University of Connecticut, ha: moved to the Department of Anatomy, College of Physicians and Surgeons, Columbia University, New York, New York.

Dr. L. (Nikki) Erlenmeyer-Kimling has moved from the Department of Medical Genetics, the New York State Psychiatric Institute, Columbia University to the University of Illinois, Urbana, where she is Assistant Professor of Psychology and Research Associate in the Institute for Research on Exceptional Children. She is studying Behavior Genetics in both Drosophila and man.

A. M. Clark, formerly of the Zoology Department, University of Tasmania, has joined the University of Adelaide as Professor of Biology and Chairman of the School of Biological Sciences Bedford Park.

Kathryn E. Fuscaldo, formerly of the Department of Biology at St. John's University has joine the faculty of the Department of Anatomy at The Hahnemann Medical College and Hospital of Philadelphia, Pennsylvania.

DuWayne C. Englert, formerly of the Population Genetics Institute at Purdue University, has been appointed Assistant Professor in the Department of Zoology at Southern Illinois Universi

Dr. T. Kanehisa, University of Kobe (Japan), has been at the Istituto di Genetica, Università di Milano, as a Research Fellow, working on the extrachromosomal factor of tumorous stock Freckled.

A. Prevosti, formerly research worker of the Centro de Genética Animal y Humana del C.S.I.C., Barcelona, has been appointed Professor of Genetics at the University of Barcelona (Spain). His new address will be: Laboratory of Genetics, Faculty of Sciences, University, Barcelona (Spain).

Yasuhiro Miyoshi, postdoctral fellow of the Department of Zoology of the Kyoto University, ha: suddenly deceased by the atrophy of kidney on January 28, 1963 in the midst of his hopefull research life. His manuscripts left, "On the resistibility of Drosophila to sodium chloride II-VII", will be printed at some time.

Richard Stevenson, former chairman of the department of biology at East Tennessee State University, Johnson City, has been appointed dean of the graduate school of that institution. H will continue to direct the genetics program there.

Eileen Gersh has moved from Chicago to Philadelphia and is working at the School of Veterinar Medicine, University of Pennsylvania.

Rosalyne Spurway, Research Assistant, Genetics Laboratory, Zoology Dept. University of Queens land, Australia, will be spending a year in the U.K. from October 1964 and is anxious to obtain employment.

Dr. William W. Johnson has accepted a position as Assistant Professor in the Department of Biology at the University of New Mexico in Albuquerque where he is establishing a Drosophila laboratory.

Dr. Samuel E. Moyer has gone as a Postdoctoral Fellow to the Genetics Department of the University of North Carolina in Raleigh where he will work on quantitative genetics in Drosophila pseudoobscura.

Antonio R. Cordeiro has been appointed to organize the new Departamento de Genética of the new Universidade de Brasília to begin January 1964. The Drosophila genetic staff that remains in Pôrto Alegre will be directed by Edmundo Kanan Marques.

## MATERIALS REQUESTED OR AVAILABLE

J. Krivshenko would appreciate receiving a yellow mutation which appeared spontaneously in the CS-120 (Canton-120) stock. He is also interested in D. busckii from natural populations.

Richard Stevenson, announces that the genetics program at East Tennessee State University, Johnson City, is being expanded with its recent removal to a new laboratory. He would like to receive reprints, particularly in population genetics and ecology, speciation, aging, and crossing over.

F. Mainx, Institut f. Allgemeine Biologie, University of Vienna, Wien IX. Schwarzspanierstr. 17, would appreciate obtaining strains of Megaselia scalaris (=Aphiochaeta xanthina) from different places as well as strains of other species of Phoridae easily bred in the laboratory.

Jack Bennett, Biology Department, NIU, DeKalb, Illinois, would appreciate receipt of wild-caught samples of D. immigrans and D. tripunctata. Attention is also called to the listing of DDT resistant and other melanogaster stocks (in this issue) which will be discarded soon; investigators desiring samples should request them immediately.

Raphael Falk Translations from Russian: The following Russian papers in genetics of Drosophila have been translated into English and were published as separate reprints:

Agol, I.I., 1929. Step-allelomorphism in Drosophila melanogaster. Zhur. eksper. biol.-medits. 5:86-101. PST Cat. No. 518.

Alikhanyan, S.I., 1937. A study of the lethal mutations in the left end of the sex-chromosome in Drosophila melanogaster. Zool. Zhur. 16 (2):247-278. PST Cat. NO.522.

Bel'govskii, M.L., 1939. Dependence of the frequency of minute chromosome rearrangements in Drosophila melanogaster upon X-ray dosage. Izv.Akad.Nauk SSSR 159-170. PST Cat. No.525

Bel'govskii, M.L., 1944. The causes of mosaicism associated with heterochromatic chromosome regions. Zhur.obsh.biol. 5(6):325-356. PST Cat. No. 529

Dubovskii, N.V., 1935. The interaction of genes, affecting the wing in Drosophila melanogaster. Izv.Akad.Nauk SSSR 2:1163-1168. PST Cat. No. 520

Medvedeva, G.B., Nechaev, I.A., 1950. Hereditary changes of physiological characters in Drosophila melanogaster. Akad.Nauk SSSR 18:247-259 PST Cat. No.535

Neuhaus, M.E., 1938. A cytogenetic study of the Y-chromosome in Drosophila melanogaster. Biol. Zhur. 7(2):335-358. PST Cat. No.523

Panshin, I.B., 1938. The cytogenetic nature of the position effect of the genes white (mottled) and cubitus interruptus. Biol.Zhur. 7(4):837-865. PST Cat. No.524

Panshin, I.B., 1935. The analysis of a bilateral mosaic mutation in Drosophila melanogaster. Akad. Nauk SSSR (Tr. Instit. Genet.) 10:227-232. PST Cat. No. 521

Prokof'eva-Bel'govskaya, A.A., 1939. A cytological study of the breaks at the white locus of the X-chromosome in Drosophila melanogaster. Izv. Akad. Nauk SSSR 2:215-227. PST Cat. No. 526

Prokof'eva-Bel'govskaya, A.A., 1939. A cytological study of the "simple breaks" in the inert region of the scute$^{8}$ chromosome of Drosophila melanogaster. Izv. Akad. Nauk SSSR - (Otd.biol nauk) 349-361. PST Cat. No. 527

Prokof'eva-Bel'govskaya, A.A., 1939. Inert regions in the inner part of the X-chromosome of Drosophila melanogaster. Izv.Akad. Nauk SSSR (Otd. biol. nauk). 362-370. PST Cat. No.528

Rapoport, I.A., 1943. The oxidation and the mechanism of action of mutagenous factors. Zhur. obsh. biol. 4(2):65-72. PST Cat. No. 530

Rapoport, I.A., 1947. Derivatives of carbamic acid and mutations. Byull. eksper. biol. medits. 23:198-201. PST Cat. No. 531

Rapoport, I.A., 1948. The alkylation of the gene molecule. Doklady Akad. Nauk SSSR 59 (6):1183-1186. PST Cat. No. 532

Rapoport, I.A., 1948. Effect of ethylene oxide, glycidol, and glycols on gene mutations Doklady Akad. Nauk SSSR 60(3):469-472. PST Cat. No. 533

Rapoport, I.A., 1948. Mutations under the influence of unsaturated aldehydes. Doklady Akad. Nauk SSSR 61(4):713-715. PST Cat. No.534

Tsubina, M.G., 1935. Hypomorphic and antimorphic genes. Biol Zhur. 4(6):997-1004. PST Cat. No. 519

They may be obtained from the "Israel Program for Scientific Translations", P.O.B. 7145, Jerusalem, Israel. Please refer to PST Catalogue Number.

## ANNOUNCEMENTS

Lindsley, D. L. and E. H. Grell: "The Mutants of Drosophila Melanogaster":

Virtually everyone agrees that a new edition of "The Mutants of Drosophila melanogaster" is a sine qua non of future work in Drosophila genetics. On the other hand many people have failed to respond to the numerous requests for contributions of material that we have sent ou Since it is evident that the utility of the new edition will be determined by the extent of the individual contributions, we implore all Drosophila geneticists who have yet to submit th material, to do so without further ado. We would like all workers

1) to extract from their own published work, including contributions to the "Notes and News" section of Drosophila Information Service, all information appropriate for incl sion in the revised edition (see below for types of information desired).
2) to extract from their unpublished material all information appropriate for inclusion in the revised edition.
3) to write this material up in such a way that it can be added to the revised edition with little alteration.
4) to send to us with the above information any suggestions for improving the revised edition.

We hope to include in the revised edition

1) most new mutations and chromosome rearrangements studied or described since the first edition.
2) new linkage information including maps of pseudoallelic loci.
3) new information on mutants that appeared in the first edition; included information will be genetic, cytological, morphological, embryological, biochemical, physiologica behavioral, and immunological.
4) breakpoints of as many rearrangements, old and new, as can be obtained.
5) all possible correlations between salivary bands and genetic loci.
6) information on strictly cytological entities such as puffs, heterochromatic blocks, nucleolus organizers, etc.
7) special purpose chromosomes (probably not special purpose stocks).
8) cross indexing in as many useful ways as seem practical. Suggestions would be welcom here.

Sokoloff, A. Availability of Tribolium Information Bulletin.

Tribolium Information Bulletin (TIB), published yearly by the Department of Genetics, University of California, Berkeley, California, is in its sixth year of publication. A limited supply of TIB 5 and 6 is still available. One copy of each will be supplied, free of charge, on a first-come first-served basis, to geneticists who may be interested in using Tribolium in their courses or to their institutional libraries. A re-issue of TIB 2 and 3 which includes information on the type of equipment needed for culturing flour beetles and some information on the biology of these organisms will be available about March, 1964.

Sokoloff, A. Establishment of a Tribolium Stock Center.

A grant from NSF will make it possible to operate a center for the preservation of mutant in Tribolium and related forms at the Department of Genetics, University of California, Berkeley, California. The mutants, as well as information on their culture, will be distributed free of charge to those interested in using them in laboratory exercises in genetics. Investigators finding useful mutants in tenebrionids (or in beetles of other families easily main-

tained in the laboratory) are urged to forward descriptions of their mutants to the Editor, Tribolium Information Bulletin (see accompanying announcement), and to send samples to the writer for inclusion in the stock center as soon as the mutants are ready to be released. Address inquiries regarding available Tribolium stocks to the writer.

The Social Science Research Council committee on Behavior Genetics (see DIS 36:137) is sponsoring an NIH supported summer institute in Behavior Genetics at the University of California, Berkeley for biologists and behavioral scientists for the early summer of 1964.

It is sponsoring a symposium on "Behavioral Consequences of Genetic Differences in Man" at Burg Wartenstein, Austria for the late summer of 1964.

It is planning two conferences and a volume on "Genetics and the Behavioral Sciences." The first conference will be held at the Center for Advanced Study in the Behavioral Sciences, Stanford, California in the fall of 1964.

Amos Hawley of the Department of Sociology, University of Michigan and Ben Willerman of the Social Science Research Council have joined the committee.

Yanders, A. F. and J.P. Perras. We have been informed that the stock originally furnished to us as D. hydei is actually D. funebris; so the sperm length measurement of 6.640 mm which we reported for D. hydei (DIS 34:112) is incorrectly listed, and should be changed to funebris.

SIXTH DROSOPHILA RESEARCH CONFERENCE: April 1, 2, and 3, 1964, Madison, Wisconson

In accordance with the decision reached at the St. Louis Conference in November, 1962, the next Drosophila Research Conference will be held April 1-3, 1964 instead of during the forthcoming November. It was felt that a meeting in November would come too soon after the International Congress in late summer.

The meeting will be held in Madison rather than in East Lansing for the following reasons:

1) A. H. Sturtevant will be a visitor in Madison during the Spring of 1964, and this will provide an opportunity for a kind of testimonial from the community of Drosophila workers.

2) Since I volunteered to be host for the next meeting, and have moved to Madison, the Ad Hoc, Permanent, Temporary Committee (with the agreement of the people in East Lansing) has voted to stick me with the job. I hereby announce the appointment of Seymour Abrahamson and James Crow as Co-Hosts.

Please write me, according to the form below, so that we can get a preliminary idea of the number of people who will attend and the number who will want to present informal reports.

Allen S. Fox
Department of Genetics
University of Wisconsin
Madison 6, Wisconsin

Name_____
Address_____
_____

I probably will / will not attend the Sixth Drosophila Research Conference in Madison, April 1-3, 1964.

I probably will be accompanied by the following persons:

Informal reports will probably be presented by:

## Geographical

(Alphabetically arranged according to country, city, laboratory.)

### ARGENTINA

Buenos Aires
Atomic Energy Commission, Biology Institute — Telephone No. 70-7711

Kirschbaum, Werner F. Research Assistant (on leave of absence to study at the University of Florida, U. S. A.).
Leon, Williams N. Technical Assistant.
de Marinic, Susana Ercolini (Mrs.). Research Assistant.
Mazar-Barnett, Beatriz (Mrs.). Doctora en Ciencias Naturales. Chemical induction of mutations.
Muñoz, Enzo Ruben. Research Assistant.
Paz, Carmen (Miss). Research Assistant, curator of stocks.
Pereyra, Edith (Miss). Technical Assistant.
Valencia, Ruby Marie (Mrs.), Ph.D. Chief of Laboratory. Radiation genetics.

Buenos Aires
Universidad de Buenos Aires, Facultad de Ciencias Exactas y Naturales, Biología Exp. Genética I
(See DIS:37).

### AUSTRALIA

Adelaide, South Australia
University of Adelaide, Department of Genetics

Hayman, D.L., Ph.D. Lecturer. Cytogenetics, melanogaster.
Levy, Anne, M.Sc. Demonstrator. Population studies, melanogaster.
Mayo, M. Jean, Ph.D. Lecturer.

Adelaide, South Australia
University of Adelaide, School of Biological Sciences, Bedford Park

Clark, A. M. Professor.
Clark, E. G. (Mrs.) Research assistant.

Brisbane, Queensland
University of Queensland, Department of Zoology.

Angus, D., B.Sc. (Hons) Graduate student. Population genetics.
Khan, F. M., M.Sc. Graduate student (Pakistan). Population genetics.
Mather, Wharton B., Ph.D. Senior Lecturer. Population genetics, cytogenetics, chromosomal polymorphism.
Spurway, Rosalyne. Research Assistant.

Melbourne, Victoria
University of Melbourne, Department of Zoology — Telephone No. 34.0484, Ext. 832

Connor, Mary A. (Miss), B.A. Fulbright Student. Migration.
Gunson, Mary M. (Miss), M.Sc. Lecturer. Salivary gland chromosomes.
Hosgood, Sally M. (Miss), B.Sc. Research student. Bristle variability in D. melanogaster.
Kaul, Dipika (Miss), M.Sc. Research student. Population genetics of D. pseudo-obscura.
Mader, Merryl (Miss), B.Sc. Research student. Population and human genetics.
Martin, J., M.Sc. Senior Demonstrator. Non-random association of inversions.
Parsons, P.A., Ph.D. Reader. Population and human genetics.
Strangio, V.A., M. Sc. Senior Demonstrator. Radiation genetics.

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## QUOTABILITY OF NOTES

There is still some misunderstanding about the restrictions on "quotability". There are several aspects to this problem that may be more obvious to your editor than to the individual contributors. In the first place, it has always been assumed that notes appearing in DIS are casual and informal reports of research in progress, or other fragmentary bits of information, important enough to be called to the attention of others, but not meriting publication in a major journal as a full-fledged scientific paper. For this reason it is to be expected that a sizeable fraction of notes will be incomplete or even, to some extent, inaccurate, and that subsequent work will round them out. In the second place, the informal nature of these contributions has gone along with an informal method of presentation to the editor. The rules which are commonly observed for clarity and accuracy in regular manuscripts do not apply here. While it is true that many of these notes are submitted in elegant form, it is also true, alas, that a goodly fraction of them are illegible or incomprehensible and at times neither I nor the typist can make complete sense out of them, although they do appear finally in DIS in the best form that we can manage. I might say parenthetically that the majority of errors that appear in DIS are errors that are copied from the original notes as sent in by the author and do not originate with us. Nevertheless, we must admit that on occasion we have failed to reproduce a note accurately; in the last issue two notes had sizeable sections accidentally omitted and these omissions changed the sense of the presentation.

In view of the above points your editor is still reluctant to include permission for quotation of notes which the author has not yet had an opportunity to examine (i.e., those in the current issue). He also feels unhappy about the blanket statement made by many workers to the effect that "all of my notes are quotable". This latter statement, unfortunately, suggests that the worker has not, in fact, looked closely at his DIS notes to see if they are accurate. Furthermore, it precludes the possibility of the author's suggesting in his list of quotable notes where more recent and accurate information may be found with respect to any DIS note that is in some way deficient.

For this situation to change, I think one of the essential conditions (pointed out by A. H. Sturtevant many years ago) would be that we provide each contributor with a proof of his article prior to its appearance in DIS. An operation of this magnitude could be considered only in conjunction with the conversion of DIS into a regular journal - a problem which will have to be faced squarely before many more issues come out.

In view of these points, then, we shall continue to include in our list of quotable notes only those which are given specifically by the author by issue and page number. We shall interpret this as indicating that the author has read the note, as it finally appeared in DIS, and wishes to make no corrections or additions of a typographical or substantive nature.

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